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THE DEVIANT DYNAMICS OF DEATH IN HETEROGENEOUS POPULATIONS

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The simplest kind of life-cycle process involves one transition that leads to exit. Examples abound. Animals and plants die, the healthy fall ill, the unemployed find jobs, the childless reproduce, and the married divorce. Residents move out, machines wear out, natural resources get used up, and buildings are torn down. Infidels convert, ex-convicts recidivate, abstainers become addicted, and holdouts adopt new technologies.

In many such collections or populations, some units are more likely to make the transition than others. Standard analytic methods largely ignore this heterogeneity; the methods assume that all members of a population (or subpopulation, such as black American males) at a given age face the same probability of change. This chapter presents methods for studying what difference heterogeneity within a population makes in the behavior of the changing population.

The analytic methods are illustrated by examples drawn from the study of human mortality, and, henceforth, the word *death* will be used instead of the more general terms *change* and *transition.* Readers interested in applications other than human mortality should associate death with an analogous notion like failure, separation, occurrence, or movement.

ROOTS OF THE RESEARCH

A small but growing body of research is relevant to the analysis of differences in behavior over time between heterogeneous and homogeneous populations. Some strands of this research can be traced to Cournot's study of judicial decisions (1838) and Weinberg's investigation of the frequency of multiple births (1902). Darwin's "population thinking" and emphasis on diversity and selection were crucial contributions (Darwin, 1859; Mayr, 1976). Gini (1924) considered heterogeneity in female fecundity; Potter and Parker (1964) and Sheps and Menken (1973) developed this approach. In their influential study of the industrial mobility of labor, Blumen, Kogan, and McCarthy (1955) distinguished "movers" from "stayers" and then considered an arbitrary number of groups with different proneness to movement; Silcock

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(1954) used a continuous distribution over individuals to describe the "rate of wastage" in labor turnover. This research on the mobility of labor was extended by McFarland (1970), Spilerman (1972), Ginsberg (1973), Singer and Spilerman (1974), and Heckman and Singer (1982); it was generalized to event-history analysis by Tuma (1983) and Tuma and Hannon (1984), among others. Contributions to the analysis of human mortality and morbidity were made by Beard (1963), Shepard and Zeckhauser (1975, 1980), Keyfitz and Littman (1980), and Vaupel, Manton, and Stallard (1979).

This rich body of research indicates that there is a core of mathematical methods that can be usefully applied to the analysis of heterogeneity in such diverse phenomena as accidents, illness, death, fecundity, labor turnover, migration, and equipment failure. These sundry applications and the varied disciplinary backgrounds of the researchers make it hardly surprising that key elements of this common core of mathematics were independently discovered by several researchers. Further progress, however, surely would be accelerated if the wide applicability of the underlying mathematics of heterogeneity were recognized.

A UNIFYING QUESTION

Building on this body of research and, most directly, on Vaupel, Manton, and Stallard (1979), this chapter addresses a basic question: How does the observed rate of death, over time, for a cohort of individuals born at the same time relate to the probability of death, over time, for each of the individuals of the cohort?[1] This question provides a unifying focus for developing the mathematical theory of the dynamics of heterogeneous populations. It is also a useful question in applied

[1] The word *rate* means different things to different specialists. In this chapter, rate of death is a measure of the likelihood of death at some instant. The phrase *rate of death* as used here has numerous aliases, including hazard rate and force of mortality. The rate of death for an individual, or individual death rate, is defined by Equation (1a); the cohort death rate is defined by Equation (1b). Note that rate of death, as used here, is neither a probability nor an average over some time period. Furthermore, note that the rate of death for an individual is a function of that individual's probability of death at some instantaneous age conditional on the individual's surviving to that age. Some readers may find it helpful to read "force of mortality" whenever the phrase *rate of death* appears.

work because researchers usually observe population death rates but often are interested in individual death rates. The effect of a policy or intervention may depend on individual responses and behavior. Furthermore, individual rates may follow simpler patterns than the composite population rates. And explanation of past rates and prediction of future rates may be improved by considering changes on the individual level.

It turns out that the deviation of individual death rates from population rates implies some surprising and intriguing results. Death rates for individuals increase more rapidly than the observed death rate for cohorts. Eliminating a cause of death can *decrease* subsequent observed life expectancy. A population can suffer a higher death rate at older ages than another population even though its members have lower death rates at all ages. A population's death rate can be increasing even though its members' death rates are decreasing.

The theory leads to some methods that may be of use to policy analysts in evaluating the effects of various interventions — for example, a medical care program that reduces mortality rates at certain ages. The theory also yields predictions that may be of considerable interest to policy analysts. In the developed countries of the world, for example, death rates after age 70 and especially after age 80 may decline faster — and at an accelerating rate — than now predicted by various census and actuarial projections. As a result, pressures on social security and pension systems may be substantially greater than expected.

MATHEMATICAL PRELIMINARIES

Let Ω be some set of parameters ω. Assume that each parameter value characterizes a homogeneous class of individuals and that the population is a mix of these homogeneous classes in proportions given by some probability distribution on Ω.

Denote by $p_\omega(a)$ the probability that an individual from homogeneous class ω will be alive at age a, and let $\mu_\omega(a)$ be the instantaneous age-specific death rate at age a for an individual in class ω. By definition,

$$\mu_\omega(a) = -[dp_\omega(a)/da]/p_\omega(a) \tag{1a}$$

Similarly, let $\bar{p}(a)$ be the probability that an arbitrary individual from the population will be alive at age a. That is, let $\bar{p}(a)$ be the expected value of the probability of surviving to age a for a randomly chosen individual at birth. Alternatively, $\bar{p}(a)$ can be interpreted as the expected value of the proportion of the birth cohort that will be alive at age a. The cohort death rate $\bar{\mu}(a)$ is then defined by

$$\bar{\mu}(a) = -[d\bar{p}(a)/da]/\bar{p}(a) \tag{1b}$$

Throughout this chapter, superscript bars will be used to denote variables pertaining to expected values either for a randomly chosen individual at birth or, equivalently, for the entire cohort.

Suppose that all individuals in a population are identical and their chances of survival are described by $p(a)$. Then $\bar{p}(a)$ is the same as $p(a)$. Thus a cohort described by $\bar{p}(a)$ could be interpreted as being a homogeneous population composed of identical individuals each of whom had life chances given by $p(a)$ equaling $\bar{p}(a)$. This remarkable fact means that researchers interested in population rates can simplify their analysis by ignoring heterogeneity; this simplification has permitted the development of demography, actuarial statistics, reliability engineering, and epidemiology.

For some purposes, however, the simplification is inadequate, counterproductive, or misleading. Sometimes researchers are interested in individual rather than population behavior, sometimes patterns on the individual level are simpler than patterns on the population level, and sometimes the impact of a policy intervention can be correctly predicted only if the varying responses of different individuals are taken into account. That is, sometimes individual differences make enough difference that it pays to pay attention to them; a variety of specific examples are given later in the chapter. Furthermore, the complexities introduced by heterogeneity are not intractable; indeed, the mathematical methods presented in this chapter are fairly simple.

BASIC MATHEMATICAL FORMULATION

In mortality analysis, the adjective *heterogeneous* usually implies that individuals of the same age differ in their chances of death. As in many other problems involving relative measurement, it is useful to have some standard or baseline to which the death rates of various individuals can be compared. Let $\mu(a)$ be this baseline death rate; how

values of $\mu(a)$ might be chosen will be discussed later. The relative risk for individuals in homogeneous class ω at time a will be defined as

$$z(a,\omega) = \mu_\omega(a)/\mu(a) \tag{2}$$

It is convenient to use $\mu(a,z)$ to denote the death rate at time a of individuals at relative risk z. Clearly

$$\mu(a,z) = z\mu(a) \tag{3}$$

Thus

$$\mu(a) = \mu(a,1) \tag{4}$$

The standard death rate $\mu(a)$ can therefore be interpreted as the death rate for the class of individuals who face a relative risk of 1.

This formulation is simple and broadly applicable. More important, it yields a powerful result that is central to the mathematics of heterogeneity. Let $f_a(z)$ denote the conditional density of relative risk among survivors at time a. As shown in Vaupel, Manton, and Stallard (1979) the expected death rate in the population $\bar{\mu}(a)$ is the weighted average of the death rates of the individuals who comprise the population:

$$\bar{\mu}(a) = \int_0^\infty \mu(a,z) f_a(z) dz \tag{5}$$

Since $\bar{z}(a)$, the mean of the relative-risk values of time a, is given by

$$\bar{z}(a) = \int_0^\infty z f_a(z) dz \tag{6}$$

it follows from (3) that

$$\bar{\mu}(z) = \mu(a)\bar{z}(a) \tag{7}$$

This simple result is the fundamental theorem of the mathematics of heterogeneity, since it relates the death rate for the population to the death rates for individuals. The value of $\mu(a)$ gives the death rate for the hypothetical "standard" individual facing a relative risk of 1; multiplying $\mu(a)$ by z gives the death rate for an individual facing a relative risk of z. The value of $\bar{z}(a)$ gives the average relative risk of the surviving population at time a. In interpreting $\bar{z}(a)$ it may be useful, following Vaupel and colleagues, to view z as a measure of "frailty" or

"susceptibility." Thus $\bar{z}(a)$ measures the average frailty of the surviving cohort.

UNCHANGING FRAILTY

The relationship over time of $\bar{\mu}(a)$ versus $\mu(a)$ is determined by the trajectory of $\bar{z}(a)$. The simplest case to study is the case where individuals are born at some level of relative risk (or frailty) and remain at this level all their lives. In this case, the only factor operating to change $\bar{z}(a)$ is the higher mortality of individuals at higher levels of relative risk; thus this pure case most clearly reveals the effects of differential selection and the survival of the fittest.

Imagine a population cohort that is born at some point in time. Let $f_0(z)$ describe the proportion of individuals in the population born at various levels of relative risk z; $f_0(z)$ can be interpreted as a probability density function. Assume that each individual remains at the same level of z for life. For convenience, the mean value of $f_0(z)$ might as well be taken as 1, so that the standard individual at relative risk 1 is also the mean individual at birth and hence $\mu(0)$ equals $\bar{\mu}(0)$. As before, let $\mu(a,z)$ and $\mu(a)$ be the death rates of individuals at relative risk z and of the standard individual. Let $H(a,z)$ be the cumulative hazard experienced from birth to time a:

$$H(a,z) = \int_0^\infty \mu(a,z)da \tag{8}$$

Clearly

$$H(a,z) = zH(a) \tag{9}$$

The probability that an individual at relative risk z will survive to age a is given by

$$p(a,z) = p(a)^z = \exp[-zH(a)] \tag{10}$$

Consequently

$$f_a(z) = f_0(z)\exp[-zH(a)] \Big/ \int_0^\infty f_0(z)\exp[-zH(a)]dz \tag{11}$$

where the denominator is a scaling factor equal to $\bar{p}(a)$, the proportion

of the population cohort that has survived to age a. Thus

$$\bar{z}(a) = \int_0^\infty z f_0(z) \exp[-zH(a)]dz \Big/ \int_0^\infty f_0(z) \exp[-zH(a)]dz \qquad (12)$$

Differentiating Equation (12) with respect to a yields

$$d\bar{z}(a)/da = -\mu(a)\sigma_z^2(a) \qquad (13)$$

where $\sigma_z^2(a)$ is the conditional variance of z among the population that is alive at time a. Since $\mu(a) > 0$ and $\sigma_z^2(a) > 0$, the value of $d\bar{z}(a)/da$ must be negative. Therefore, as might be expected, the mean relative risk declines in time as death selectively removes the frailest members of the population. This means that $\mu(a)$ increases more rapidly than $\bar{\mu}(a)$.

Mean relative risk declines monotonically not only with age (or time) a but also with the proportion surviving p. This can be shown as follows. If $\mu(a)$ is greater than zero for all a, then

$$\bar{z}(a) > \bar{z}(b) \qquad \text{if and only if } a < b \qquad (14)$$

and

$$\bar{p}(a) > \bar{p}(b) \qquad \text{if and only if } a < b \qquad (15)$$

Consequently

$$\bar{z}[\bar{p}^{-1}(\bar{p})] < \bar{z}[\bar{p}^{-1}(\bar{p}^*)] \qquad \text{if and only if } \bar{p} < \bar{p}^* \qquad (16)$$

where $\bar{p}^{-1}(\bar{p})$ is the inverse function of $\bar{p}(a)$ and where $\bar{p}$ and $\bar{p}^*$ are two specific values of the survival function.

HOW μ DIVERGES FROM $\bar{\mu}$

The magnitude of the divergence $\mu(a)$ from $\bar{\mu}(a)$ depends on the distribution of relative risk. Several researchers in different fields, including Silcock (1954), Spilerman (1972), Mann, Schafer, and Singpurwalla (1974), and Vaupel, Manton, and Stallard (1979), have discovered that the gamma distribution is especially convenient to work with, since it is one of the best-known nonnegative distributions, it is analytically tractable, and it takes on a variety of shapes depending on parameter values. If the mean relative risk at birth is 1, then the gamma probability density function at birth is given by

$$f_0(z) = k^k z^{k-1} \exp(-kz)/\Gamma(k) \qquad (17)$$

where k, the so-called shape parameter, equals (when the mean is 1) the inverse of the variance σ^2. When $k = 1$, the distribution is identical to the exponential distribution; when k is large, the distribution assumes a bell-shaped form reminiscent of a normal distribution.

If relative risk at birth is gamma-distributed with mean 1, it can be shown (see Vaupel, Manton, and Stallard, 1979) that

$$\bar{z}(a) = \bar{p}(a)^{\sigma^2} = \exp[-\sigma^2 \bar{H}(a)] = \exp\left[-\sigma^2 \int_0^\infty \bar{\mu}(a)da\right] \tag{18}$$

and that

$$\bar{z}(a) = 1/[1 + \sigma^2 H(a)] \tag{19}$$

Thus the relationship of $\mu(a)$ to $\bar{\mu}(a)$, as determined by $\bar{z}(a)$, can be determined by the cumulative hazard for either the population or the standard individual. In the special case where $\sigma^2 = 1$, the value of $\bar{z}(a)$ falls off with $\bar{p}(a)$, the proportion of the cohort that is surviving. It also can be shown (Vaupel, Manton, and Stallard, 1979) that $f_a(z)$ is gamma-distributed with a mean of $\bar{z}(a)$ and a shape parameter equal to the same value of k at birth.

These results for the gamma distribution with mean 1 at birth are easily generalized to the case of any mean $\bar{z}(0)$ at birth. Equation (18) then becomes

$$\bar{z}(a) = \bar{z}(0)\bar{p}(a)^{\sigma^2} \tag{20}$$

and Equation (19) becomes

$$\bar{z}(a) = \bar{z}(0)/[1 + \sigma^2 \bar{z}(0)H(a)] \tag{21}$$

There is, however, little reason to use this generalized formulation. Let

$$\bar{z}^*(a) = \bar{z}(a)/\bar{z}(0) \tag{22a}$$

and

$$\mu^*(a) = \mu(a)/z(0) \tag{22b}$$

This simple transformation converts Equations (20) and (21) back to (18) and (19). Furthermore, as indicated earlier, the standard death rate $\mu(a)$ might as well be associated with the mean individual at birth.

Instead of working with a gamma distribution, it might seem more natural to assume that there is some normally distributed risk

factor w that determines relative risk z:

$$z = w^2 \tag{23}$$

It turns out that if w is normally distributed with mean zero and any variance σ^2, then z will be gamma-distributed with a shape parameter of $\frac{1}{2}$. Thus nothing is to be gained by working with the normal distribution with mean zero rather than with a gamma distribution.

In the mover/stayer model developed by Blumen, Kogan, and McCarthy (1955), individuals fall into two groups with relative risk z_1 and z_2. The value of z_1 can be assumed equal to zero, but more generally z_1 can simply be taken as less than z_2. Using (12), it is not difficult to confirm that when mean relative risk at birth is 1, then

$$\bar{z}(a) = \frac{[z_1/(1-z_1)]\exp[-z_1H(a)] + [z_2/(z_2-1)]\exp[-z_2H(a)]}{[1/(1-z_1)]\exp[-z_1H(a)] + [1/z_2-1)]\exp[-z_2H(a)]} \tag{24}$$

Consequently $\bar{z}(a)$ will start at a value of 1 when a is zero and will fall off to a value of z_1 as the individuals at relative risk z_2 die off at a relatively rapid rate.

Another distribution of interest may be the uniform distribution, stretching from $1-\alpha$ to $1+\alpha$, with $\alpha < 1$. In this case, it is possible to show that

$$\bar{z}(a) = 1 - \alpha\left\{\frac{\exp[\alpha H(a)] + \exp[-\alpha H(a)]}{\exp[\alpha H(a)] - \exp[-\alpha H(a)]}\right\} + \frac{1}{H(a)} \tag{25}$$

In deriving this result, it is helpful to realize that $\bar{z}(a)$ can be considered to be a function of H and that the equation for $\bar{z}(H)$ can be expressed as

$$\bar{z}(H) = [df^*(H)/dH]/f^*(H) \tag{26}$$

where $f^*(H)$ is the Laplace transform of $f_0(z)$. Equation (25) implies that $\bar{z}(a)$ approaches $1-\alpha$ as a increases.

Although formulas for $\bar{z}(a)$ have not been derived for other distributions, the value of $\bar{z}(a)$ can generally be readily computed, to a close approximation, by applying numerical methods to Equation (12). The values in Table 1 for the Weibull and log-normal distributions were calculated in this way.

Table 1 is designed to show how $\mu(a)$ diverges from $\bar{\mu}(a)$ given different initial distributions of relative risk with different variances. The table presents values of $\mu(a)$ divided by $\bar{\mu}(a)$, which equals the inverse of $\bar{z}(a)$. The results are presented for different values of $\bar{p}(a)$,

TABLE 1
Divergence of μ from $\bar{\mu}$

Variance and Forms of Initial Distribution of Relative Risk	Values of $\mu/\bar{\mu}$ When $\bar{p}$ Is					
	1.00	0.75	0.50	0.25	0.10	0.05
$\sigma^2 = 0.1$						
Gamma	1.00	1.03	1.07	1.15	1.26	1.35
Weibull	1.00	1.03	1.08	1.17	1.34	1.49
Log normal	1.00	1.03	1.07	1.14	1.23	1.30
$\sigma^2 = 1$						
Exponential[a]	1.00	1.33	2.00	4.00	10.00	20.00
Log normal	1.00	1.27	1.64	2.30	3.33	4.24
$\sigma^2 = 2$						
Gamma	1.00	1.78	4.00	16.00	100.00	400.00
Weibull	1.00	1.70	3.32	9.56	36.10	99.00
Log normal	1.00	1.49	2.23	3.46	5.61	7.65

[a] When $\sigma^2 = 1$, the gamma and Weibull distributions are identical to the exponential distribution.

the proportion of the initial population that is surviving; presenting the results for values of $\bar{p}(a)$ rather than for values of a is convenient since assumptions about the rate of aging (that is, about how $\mu(a)$ changes with a) do not have to be made. Table 1 indicates that $\mu(a)$ can be substantially greater than $\bar{\mu}(a)$ when only a fraction of the population is alive. Even when the variance in relative risk is only 0.1 (compared with a mean level of birth of 1), $\mu(a)$ is 30 to 50 percent higher than $\bar{\mu}(a)$ when 5 percent of the population is surviving. As the table demonstrates, the degree of divergence of $\mu(a)$ from $\bar{\mu}(a)$ depends on both the form of the initial distribution of relative risk and the variance of this distribution.

THE SHAPE OF THE AGING TRAJECTORY

Although Table 1 and Equations (18), (19), (24), and (25) provide information about the divergence between the death rate for the standard individual, $\mu(a)$, and the observed cohort death rate, $\bar{\mu}(a)$, analysis of the shape of $\mu(a)$ and $\bar{\mu}(a)$ requires some assumptions about how one of these two curves increases with a. If relative risk at birth is gamma-distributed with mean 1 and variance σ^2, the correspondence between six different formulas for $\mu(a)$ and $\bar{\mu}(a)$ is as given in Table 2. Figure 1 depicts how the curves for $\mu(a)$ and $\bar{\mu}(a)$ diverge in four cases.

TABLE 2
Algebraic Expressions for μ and $\bar{\mu}$

When z is Gamma-Distributed with Variance σ^2 at Birth	
If the value of $\mu(a)$ is given by:	. . . then the value of $\bar{\mu}(a)$ is given by:
c	$c/(1 + \sigma^2 ca)$
ca	$ca/(1 + \sigma^2 ca^2/2)$
$c \exp(ba)$	$c \exp(ba)/\{1 + \sigma^2 c[\exp(ba) - 1]/b\}$
$c(\exp - ba)$	$c \exp(-ba)/\{1 + \sigma^2 c[1 - \exp(-ba)]/b\}$
$c \exp(ba) \exp\{\sigma^2 a[\exp(ba) - 1]/b\}$	$c \exp(ba)$
$c \exp(-ba) \exp\{\sigma^2 a[1 - \exp(-ba)]/b\}$	$c \exp(-ba)$

Note: If $\mu(a) = ca$, then $\bar{\mu}(a)$ reaches a maximum of $(c/2\sigma^2)^{1/2}$ when $a = (2/c\sigma^2)^{1/2}$. If $\mu(a) = c \exp(ba)$, then as $a \to \infty$, $\bar{\mu}(a) \to b/\sigma^2$. If $\bar{\mu}(a) = c \exp(ba)$ (that is, follows a Gompertz curve), then the ratio of $\mu(a)$ to $\bar{\mu}(a)$ can be expressed as a double-exponential equation: $\mu(a)/\bar{\mu}(a) = \alpha\beta^{\gamma^a}$.

Figure 1. Patterns of divergence.

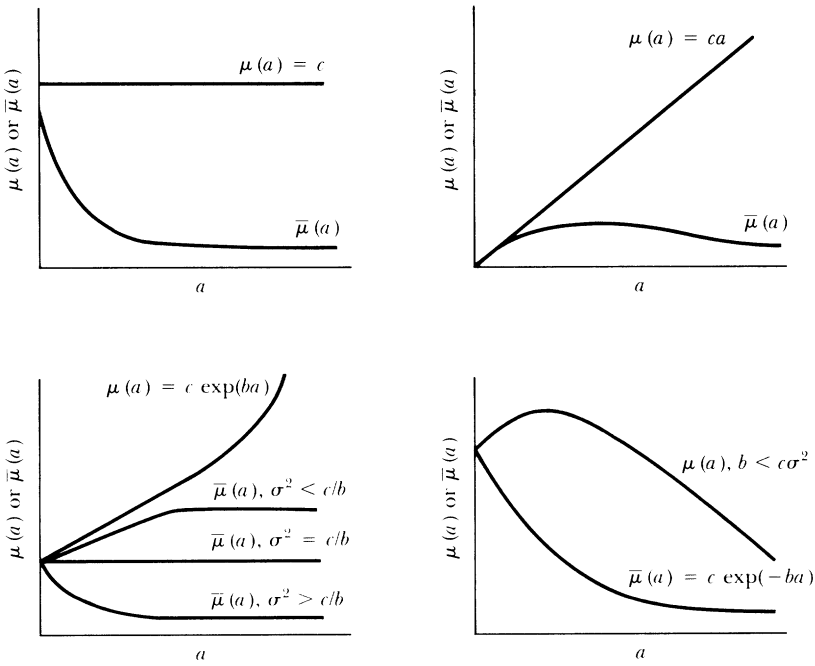


Table 2 and Figure 1 clearly demonstrate that the pattern of individual aging can differ radically from the observed pattern of aging in the surviving cohort. When $\mu(a)$ is constant, for instance, $\bar{\mu}(a)$ declines with age; heterogeneity introduces spurious age dependence on the population level (McFarland, 1970; also see Beard, 1963).

THE DISTRIBUTION OF LIFE SPANS

Although the discussion so far has focused on the divergence of μ and $\bar{\mu}$ over time, comparisons of individual versus cohort behavior in heterogeneous populations could also be expressed in terms of other statistics. Consider, for example, the fractiles of the distribution of life spans or, equivalently, the distribution of age of death. Table 3 presents some of these fractiles for a population and for individuals. Fractiles for the standard individual are given for three levels of heterogeneity as measured by σ^2; fractiles are also presented for individuals at three levels of relative risk z. The calculations assume that relative risk is gamma-distributed with mean 1 at birth and that the observed death rate for the population is given by a Gompertz function, $c \exp(ba)$, where c equals 0.00012 and b equals 0.085. Table 3 indicates that the distribution of life spans in a population is more spread out than the distribution of possible life spans for an individual. In particular, the right-hand tail of the distribution is shorter for individuals, especially for robust individuals where variance in heterogeneity is high.

MORTALITY CONVERGENCE AND CROSSOVER

For many pairs of populations, reported mortality rates converge and even cross over with age. In the United States, for example, blacks have lower mortality rates than whites after age 75 or so (Manton and Stallard, 1981). In most developed countries, male and female death rates converge in old age. Nam, Weatherby, and Ockay (1978) present statistics on this and a variety of other convergences and crossovers.

These reported convergences and crossovers of population death rates may be the result of age misreporting or actual individual differences in rates of aging. To some extent, they may also be artifacts of heterogeneity in individual death rates. Let $r(a)$ denote the ratio of

TABLE 3
Distribution of Life Spans

Category	Age at Which Probability of Being Alive Equals 0.75	0.50	0.25	0.10	0.01	0.001	Length of Right-Hand Tail $a_{0.001} - a_{0.50}$
Entire cohort	62.6	72.9	81.1	87.0	95.2	100.0	27.1
Individuals							
$z = 1$							
$\sigma^2 = 0.1$	62.4	72.5	80.3	85.8	92.9	96.7	24.2
$\sigma^2 = 1$	61.1	69.7	75.6	79.3	83.6	85.8	16.1
$\sigma^2 = 10$	53.8	58.8	61.9	63.8	66.0	67.2	8.4
$\sigma^2 = 1$							
$z = 0.1$	80.8	85.8	88.9	90.8	93.1	94.2	8.4
$z = 1$	61.1	69.7	75.6	79.3	83.6	85.8	16.1
$z = 10$	35.9	45.7	53.3	58.8	65.8	69.7	24.0

Note: See text for discussion of the underlying assumptions.

death rates for the standard individual in population 2 versus 1:

$$r(a) = \mu_2(a)/\mu_1(a) \tag{27a}$$

Similarly, let $\bar{r}(a)$ denote the ratio of the population death rates:

$$\bar{r}(a) = \bar{\mu}_2(a)/\bar{\mu}_1(a) \tag{27b}$$

For simplicity, assume that the ratio is constant over time on the individual level, so that individuals at any level of relative risk in the second population are always r times more likely to die than corresponding individuals in the first population:

$$r(a) = r > 1 \qquad \text{for all } a \tag{28}$$

Further assume that relative risk is gamma-distributed in the two populations with mean 1 and variances σ_1^2 and σ_2^2 at birth. Let

$$\rho = \sigma_2^2/\sigma_1^2 \tag{29}$$

Then it follows from Equations (7) and (19) that at birth

$$\bar{r}(0) = r(0) = r \tag{30a}$$

but as a increases,

$$\bar{r}(a) \to 1/\rho \tag{30b}$$

Depending on the value of ρ—that is, on the ratio of the variances in relative risk—$\bar{r}(a)$ can either increase or decrease. If ρ is greater than 1, then $\bar{r}(a)$ will fall to a value less than 1. This means that although, on the individual level, $\mu_2(a)$ is always r times higher than $\mu_1(a)$, the cohort death rate $\bar{\mu}_2(a)$ will start out higher than $\bar{\mu}(a)$ and will end up below $\bar{\mu}_1(a)$. The crossover point will occur when

$$\bar{p}_1(a) = (\rho - 1)/(\rho - 1/r) \tag{31}$$

where $\bar{p}_1(a)$ is the proportion of population 1 still surviving at age a. If r equals 2 and ρ equals 1.5, for example, the crossover will occur when $p_1(a)$ equals 0.5. Figure 2 compares the trajectories of r and $\bar{r}$; Table 4 presents specific numerical results.

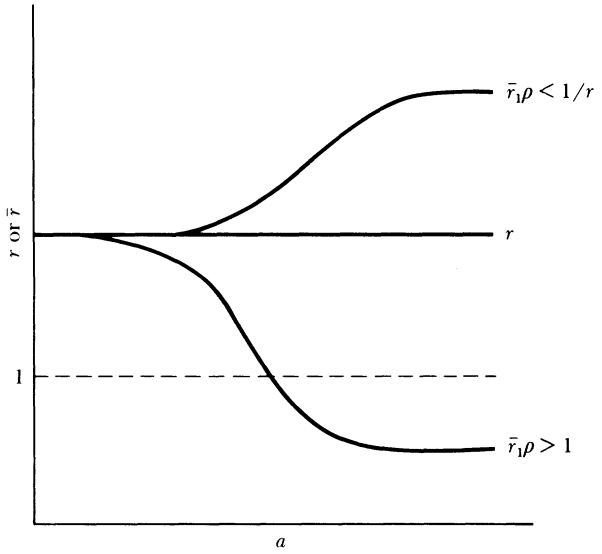
Empirical data on convergences and crossovers in mortality rates can be used to estimate the degree of heterogeneity in relative risk in a population. If some assumption is made about the distribution of relative risk—for example, that it is gamma-distributed—and about the relationship of $\mu_1(a)$ to $\mu_2(a)$—for example, that one is a constant multi-

TABLE 4
A Mortality Crossover

a	$\bar{\mu}_1$	$\bar{\mu}_2$	$\bar{r} = \bar{\mu}_2/\bar{\mu}_1$	μ_1	μ_2	$r = \mu_2/\mu_1$
0	0.00010	0.00020	2.00	0.00010	0.00020	2.0
20	0.00073	0.00144	1.96	0.00074	0.00148	2.0
40	0.00518	0.00899	1.74	0.00546	0.01092	2.0
60	0.02877	0.03092	1.07	0.04034	0.08069	2.0
70	0.05233	0.04075	0.78	0.10966	0.21933	2.0
90	0.08902	0.04851	0.54	0.81031	1.62062	2.0
∞	—	—	—	—	—	2.0

Assumptions: In calculating this table, which illustrates how an observed crossover in death rates in two populations may be an artifact of heterogeneity, it was assumed that relative risk is gamma-distributed with mean, $\sigma_2^1 = 1$, and $\sigma_2^2 = 2$. Furthermore $\mu_2(a) = 2\mu_1(a)$ for all a, where $\mu_1(a) = 0.0001 \exp(0.1a)$.

Figure 2. Patterns of mortality convergence and divergence.



ple of the other—then estimates of the variance in heterogeneity can be calculated. Manton, Stallard, and Vaupel (1981) applied this method to various cohorts of the four populations of male and female Swedes and American whites. The results suggest that for these populations the variance in heterogeneity may be between 0.1 and 1. More research, however, is needed here, especially concerning the robustness of the estimates to assumptions about the distribution of relative risk. As shown by Heckman and Singer (1982), different assumptions about the distribution of relative risk may lead to radically different empirical estimates.

GERONTOLOGICAL FAILURES OF PEDIATRIC SUCCESS

Heterogeneity slows observed rates of progress in reducing population death rates at older ages. Essentially, reductions in death rates at younger ages permit frailer individuals to survive to older ages. This influx of frailer individuals serves as a brake on reductions in mortality rates at the older ages.

As a simple illustration, divide life into two parts — youth and old age, say — at age a_0. Suppose that a proportion $\bar{p}(a_0)$ of every birth cohort used to survive to age a_0, but that because of some pediatric advance a proportion $\bar{p}^*(a_0)$, greater than $\bar{p}(a_0)$, now survives. Because $\bar{z}$ increases with $\bar{p}$ monotonically, $\bar{z}(a_0)$ will increase. Consequently, if the values $\mu(a)$, where a is greater than a_0, remain the same, the values of $\bar{\mu}(a)$, where a is greater than a_0, will increase. If observed death rates at younger ages are reduced to low levels, however, further progress will add fewer and fewer *additional* persons to the ranks of the elderly. Thus progress in reducing population mortality rates will not be slowed to the extent it previously was.

Until now this chapter has focused on a single cohort aging through time; thus a represents both age and time. Generalization to the case of multiple cohorts is straightforward: Let $\mu(a,t)$, $\bar{\mu}(a,t)$, and $\bar{z}(a,t)$ be the values of μ, $\bar{\mu}$, and $\bar{z}$ for a cohort of age a in year t. Then the fundamental theorem (7) can be rewritten as

$$\bar{\mu}(a,t) = \mu(a,t)\bar{z}(a,t) \tag{32}$$

and it follows that

$$\frac{\partial\bar{\mu}(a,t)/\partial t}{\bar{\mu}(a,t)} = \frac{\partial\mu(a,t)/\partial t}{\mu(a,t)} + \frac{\partial\bar{z}(a,t)/\partial t}{\bar{z}(a,t)} \tag{33}$$

Let

$$\pi_a(t) = -\frac{\partial\mu(a,t)/\partial t}{\mu(a,t)} \tag{34a}$$

and

$$\bar{\pi}_a(t) = -\frac{\partial\bar{\mu}(a,t)/\partial t}{\bar{\mu}(a,t)} \tag{34b}$$

Thus π and $\bar{\pi}$ are measures of the rate of progress in reducing individual and population death rates. Equation (33) can be rewritten as

$$\bar{\pi}_a(t) = \pi_a(t) - \frac{\partial\bar{z}(a,t)/\partial t}{\bar{z}(a,t)} \tag{35}$$

When individuals remain at the same level of relative risk for life, progress in reducing individual death rates will reduce the value of the negative term in this formula; at any age a the value of $\bar{z}(a,t)$ will approach 1 as t increases, and the value of $\partial(\bar{z})(a,t)/\partial t$ will approach zero.

This is easy to see in the special case where relative risk is gamma-distributed at birth with a mean and variance of 1. Then $\bar{z}(a)$ equals $\bar{p}(a)$ so that

$$\frac{\partial \bar{z}(a,t)/\partial y}{\bar{z}(a,t)} = \frac{\partial \bar{p}(a,t)/\partial t}{\bar{p}(a,t)} \tag{36}$$

The proportion surviving at any age a will clearly approach 1 as progress in reducing death rates continues. Furthermore, the change over time in the proportion surviving will approach zero.

Equation (35) consequently indicates that as progress in reducing individual death rates continues,

$$\bar{\pi}_a(t) \rightarrow \pi_a(t) \qquad \text{for any } a \tag{37}$$

Since progress in reducing death rates permits frailer individuals to survive to older ages,

$$\partial \bar{z}(a,t)/\partial a < 0 \tag{38}$$

But, of course, $\bar{z}(a,t)$ is greater than zero. Therefore

$$\bar{\pi}_a(t) < \pi_a(t) \qquad \text{for any } a \tag{39}$$

In short, the observed rate of progress in reducing the population death rate at any age a will be less than, but will eventually approach, the rate of progress in reducing individual death rates at age a. Table 5 presents numerical results concerning $\bar{\pi}_a(t)$ when $\pi_a(t)$ is constant for all a and t; Figure 3 depicts the pattern of these results.

TABLE 5
Acceleration in Observed Rates of Progress in Reducing Mortality Rates

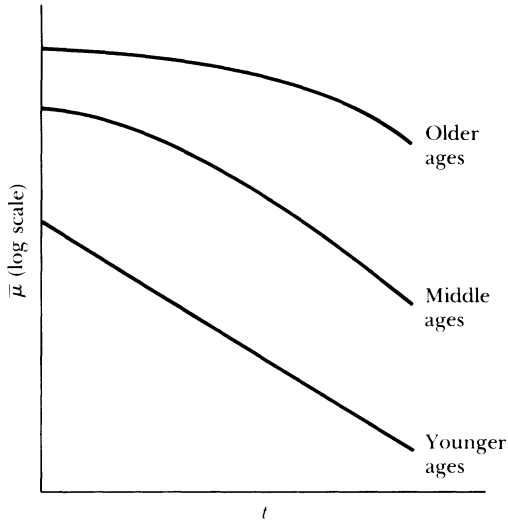
Year t	Observed Rate of Progress $\bar{\pi}(a,t)$ When Age a Equals 20	40	60	80
0	0.00986	0.00894	0.00528	0.00131
40	0.00991	0.00927	0.00626	0.00184
80	0.00994	0.00950	0.00714	0.00252
120	0.00996	0.00966	0.00788	0.00334
∞	0.01000	0.01000	0.01000	0.01000

Note: It is assumed that the rate of progress on the individual level is 0.01:

$$[\partial \mu(a,t)/\partial t]/\mu(a,t) = \pi = 0.01 \qquad \text{for all } a,\ t$$

Furthermore, z is assumed to be gamma-distributed with mean 1 and variance 1 at birth and $\mu(a,0) = 0.0002 \exp(0.1a)$.

Figure 3. Trajectories of progress in reducing mortality rates.



The pattern shown in Figure 3 is roughly the pattern actually observed in the United States, Sweden, and other countries over the course of this century. Thus the observed acceleration of progress in reducing mortality at older ages may be, at least in part, an artifact of heterogeneity. To the extent that this is true, death rates after age 70 and especially after age 80 may decline faster in the future than now predicted—and at an accelerating rate. The various implications of an increase in the size of the elderly populations, including the pressures it would place on pension systems, are discussed by Myers (1981).

WHEN PROGRESS STOPS

Suppose progress has been made over a number of years in reducing individual mortality rates and then, suddenly, the progress stops so that the mortality rates henceforth remain constant. In the succeeding years (that is, as t increases), the value of $\bar{p}(a,t)$, the proportion of the original birth cohort surviving to age a in year t, will increase and then level off. The increase in $\bar{p}(a)$ results from the aging of the

younger cohorts that have experienced lower death rates because of the previous progress. Since, as noted earlier, $\bar{z}$ is a monotonically increasing function of $\bar{p}$, it follows that $\bar{z}$ will increase as well. The value of $\mu(a,t)$, any a and t, will be constant—that is what no progress means. But then it follows from Equation (32) that $\bar{\mu}(a,t)$ at any age a will increase in time.

In short, current mortality rates for populations are lower than the mortality rates that would prevail if current mortality rates for individuals persisted. If progress in health conditions stops, death rates will rise. This implies that estimates of current life expectancy are too high. These estimates are based on current population death rates, but they are supposed to represent what life expectancy would be if health conditions remained unchanged. Vaupel, Manton, and Stallard (1979) indicate how the correct value of current life expectancy, adjusted for the effects of heterogeneity and past health progress, might be calculated. Table 6 and Figure 4 compare the patterns of $\mu(a,t)$ and $\bar{\mu}(a,t)$ when health progress stops.

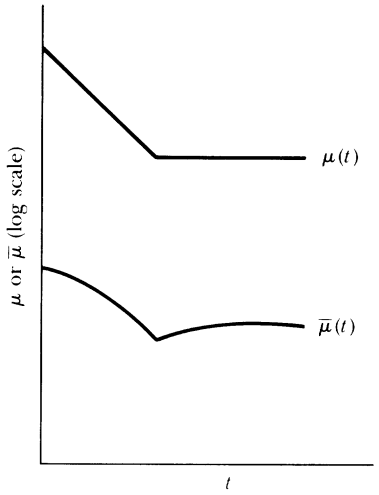
If progress in reducing μ accelerates and decelerates with time, the observed trajectory of $\bar{\mu}$ will be bumpy and might show periods of apparent negative progress; this phenomenon might underlie the increase in death rates observed in the United States in the middle and late 1960s, following a relatively rapid decrease in the 1950s.

TABLE 6
When Progress in Reducing Mortality Rates Stops

Year t	$\mu(60,t)$	$\bar{\mu}(60,t)$
0	0.08069	0.04264
20	0.06606	0.03817
40	0.05409	0.03384
60	0.04428	0.02972
80	0.03625	0.02588
81	0.03625	0.02595
90	0.03625	0.02635
∞	0.03625	0.02662

Assumptions: $\mu(a,0) = 0.0002 \exp(0.1a)$
$\mu(a,t) = \mu(a,0) \exp(-0.01t)$, $t < 80$
$\mu(a,t) = \mu(a,80)$, $t > 80$

Figure 4. When progress in reducing mortality rates stops.



INDEPENDENT COMPETING RISKS

Suppose there are several causes of death and that an individual can be at different relative risks for the different causes. Let z_j denote the level of relative risk for cause of death j and let $\mu_j(a,z_j)$ be the death rate from cause j at time (or age) a for individuals at relative risk z_j. As before, define z_j such that

$$\mu_j(a,z_j) = z_j\mu_j(a,1) = z_j\mu_j(a) \tag{40}$$

Assume that an individual's relative risk for any cause of death is independent of his or her relative risk for any other cause of death. Then, as shown in the appendix, a straightforward generalization of the fundamental theorem (7) yields

$$\bar{\mu}_j(a) = \mu_j(a)\bar{z}_j(a) \tag{41a}$$

and

$$\bar{\mu}(a) = \sum_{i=1}^{n} \bar{\mu}_j(a) \tag{41b}$$

where $\bar{\mu}_j$ represents the population death rate from cause j and where $\bar{z}_j(a)$ is the mean relative risk from cause j among the individuals surviv-

ing to time a. The value of $\bar{z}_j(a)$ for any cause of death j can be calculated on the basis of $f_0(z_j)$, the distribution of z_j at birth, and $\mu_j(a)$, the death rate from cause j:

$$\bar{z}_j(a) = \frac{\int_0^\infty z_j f_0(z_j) \exp\left[-\int_0^a z_j \mu_j(s)ds\right] dz_j}{\int_0^\infty f_0(z_j) \exp\left[-\int_0^a z_j \mu_j(s)ds\right] dz_j} \tag{42}$$

Thus the dynamics of mortality from any specific cause of death can be studied without knowing the death rates and distributions of relative risks for other causes of death.

Suppose that the z_j are gamma-distributed with mean 1 and variances σ_j^2. (As before, the means might as well be set equal to 1, as in that case the standard individual at relative risk 1 will be the mean individual at birth.) Then Equation (19) generalizes to

$$\bar{z}_j(a) = 1/[1 + \sigma_j^2 H_j(a)] \tag{43}$$

where

$$H_j(a) = \int_0^a \mu_j(s)ds \tag{44}$$

Furthermore, Equation (18) generalizes to

$$\bar{z}_j(a) = \bar{p}_j(a)^{\sigma_j^2} \tag{45}$$

where $\bar{p}_j(a)$ is the proportion that would survive to age a if j were the only cause of death:

$$\bar{p}_j(a) = \exp\left[-\int_0^a \bar{\mu}_j(s)ds\right] \tag{46}$$

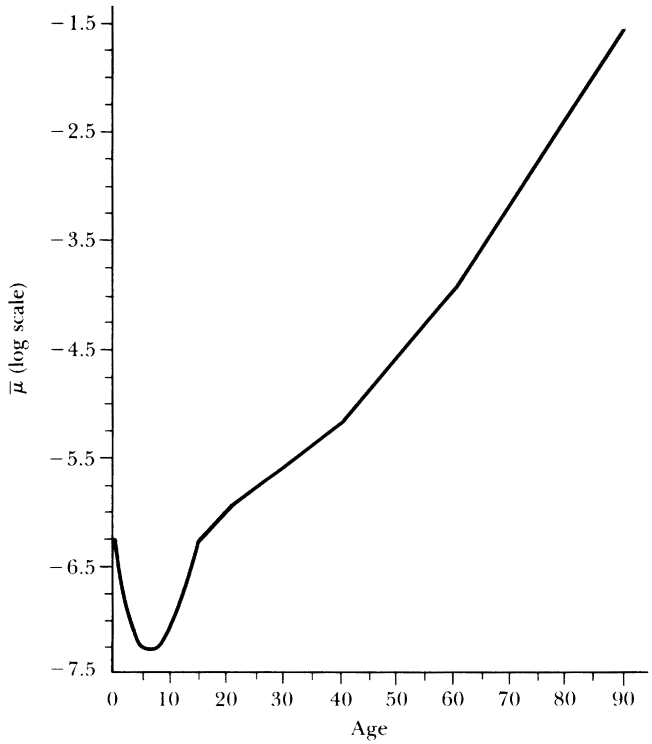
The formulas for the uniform distribution (25) and the two-point distribution (24) similarly generalize.

Thus the case of independent, competing risks is almost as easy to analyze as the simpler case of a single cause of death. In a sense the competing risk case adds another dimension of heterogeneity, as now individuals not only differ from each other but also differ within themselves in susceptibility to various causes of death.

Patterns of aging for individuals can be compared with observed patterns of aging for the surviving cohort in much the same way when

there are several causes of death as when there is only a single cause of death. Figure 5 presents an example. The mortality curve shown in Figure 5, which is plotted on a log scale, is intriguing because it resembles the observed mortality curves of most developed countries: Mortality falls off after infancy, begins increasing again after age 7 or so, rises through a hump roughly between ages 15 and 30, and then at older ages increases more or less exponentially. Figure 5 was created by assuming there were three causes of death. For individuals, the incidence of the first cause is constant, the incidence of the second cause increases exponentially, and the incidence of the third cause increases according to the double-exponential form that produces, on the population level, an observed exponential increase. The three independent causes of death act, on the individual level, as follows: $\mu_1(a) = 0.02$ and z_1 is gamma-dis-

Figure 5. A population mortality curve produced by three causes of death.



tributed with $\sigma_1^2 = 500$; $\mu_2(a) = 0.00001 \exp(0.04a)$ and z_2 is gamma-distributed with $\sigma_2^2 = 200$; $\mu_3(a) = a \exp(ba) \exp\{a[\exp(ba) - 1]/b\sigma_3^2\}$, $a = 0.00015$, $b = 0.08$, and z_3 is gamma-distributed with $\sigma_3^2 = 1$.

Just as mortality convergences and crossovers for two populations may be artifacts of heterogeneity, convergences and crossovers for two causes of death may also be artifacts of heterogeneity. In the earlier discussion of population crossovers, the subscript j denoted population 1 or 2—for example, $\bar{\mu}_j$ was the death rate for population j. The mathematics is equally valid if the subscript j denotes cause of death 1 or 2. So, for example, cause of death 2 might be twice as likely as cause of death 1, at all ages, for all individuals. If the variance in z_2, however, is greater than twice the variance in z_1, the observed rate of death from cause 2 in the surviving cohort will approach and eventually fall below the observed rate for cause 1.

How will progress in reducing individual death rates affect observed progress in reducing deaths in surviving cohorts? For any specific cause of death, the mathematics is the same as outlined in the preceding section on progress. Furthermore, in the case being considered here of independent causes of death, progress in reducing one cause of death will have no effect on $\mu_j(a)$ or $\bar{\mu}_j(a)$ for any other cause of death j. Since everyone has to die of something, the *number* of people eventually dying from other causes will increase, but the death *rates* μ_j and $\bar{\mu}_j$ will not change.

CORRELATED CAUSES OF DEATH

When causes of death are not independent but are correlated with each other, the mathematics becomes more complicated. The fundamental equations (41a) and (41b) are still valid, but now the value of $\bar{z}_j(a)$ depends on the death rates and distributions of relative risks for correlated causes of death:

$$\bar{z}_j(a) = \frac{\int_0^\infty \cdots \int_0^\infty z_j f_0(z_1 \ldots z_n) \exp[-z_1 H_1(a) \cdots z_n H_n(a)] dz_1 \ldots dz_n}{\int_0^\infty \cdots \int_0^\infty f_0(z_1 \ldots z_n) \exp[-z_1 H_1(a) \cdots z_n H_n(a)] dz_1 \ldots dz_n} \tag{47}$$

where, as before,

$$H_j(a) = \int_0^a \mu_j(s)ds$$

As a simple example, consider the following special case. Suppose that there are two causes of death and that, as in the mover/stayer model, there are two kinds of people. Let $\mu_1(a)$ and $\mu_2(a)$ be the death rates from cause 1 and 2 for the standard individual in the first group, and let $\mu_1^*(a)$ and $\mu_2^*(a)$ be the rates for the second group. Finally, suppose the rates are interrelated as follows:

$$0 < \mu_2^*(a) < \mu_1(a) \qquad \text{for all } a \tag{48a}$$

and

$$\mu_2^*(a) = 0 \qquad \text{for all } a \tag{48b}$$

Thus the second, "robust," group does not die from cause 2 and faces a lower death rate than the first group does from cause 1.

Let $\pi(a)$ denote the proportion of the total population that is in the first group at time a. The observed death rate for the first cause of death will be

$$\bar{\mu}_1(a) = \pi(a)\mu_1(a) + [1 - \pi(a)]\mu_1^*(a) \tag{49a}$$

and the observed death rate for the second cause of death will simply be

$$\bar{\mu}_2(a) = \pi(a)\mu_2(a) \tag{49b}$$

Suppose some progress is made in reducing the incidence of the second cause of death. Then the observed death rate from the first cause will increase. This observed death rate is the weighted average of the death rates for the first and second groups. If death rates for the first group are reduced (as a result of progress against the second cause of death), more of this group will survive. The value of $\pi(a)$ will increase and since $\mu_1(a)$ exceeds $\mu_1^*(a)$, the value of $\bar{\mu}_1(a)$ will also increase. The value of $\pi(a)$, by the way, is given by

$$\pi(a) = \frac{\pi(0)\exp\left\{-\int_0^a [\mu_1(s) + \mu_2(s)]ds\right\}}{\pi(0)\exp\left\{-\int_0^a [\mu_1(s) + \mu_2(s)]ds\right\} + [1 - \pi(0)]\exp\left[-\int_0^a \mu_1^*(s)ds\right]} \tag{50}$$

A general situation in which causes of death are correlated can be described as follows. Let $z_0, \ldots, z_n$ be independent relative risks with mean 1. Let the death rate for an individual be given by

$$\mu_j(a,z) = [w_j z_0 + (1 - w_j)z_j]\mu_j(a) \tag{51}$$

where z is the vector of relative risks for the individual and w_j is a weight such that

$$0 < w_j < 1 \qquad j = 1, \ldots, n \tag{52}$$

The basic idea is that an individual's risk from any specific cause of death j depends on a general relative-risk (or frailty) factor z_0 and a specific relative-risk factor z_j.

It can be readily shown that

$$\bar{\mu}_j(a) = [w_j \bar{z}_0(a) + (1 - w_j)\bar{z}_j(a)]\mu_j(a) \tag{53}$$

If the z_j are gamma-distributed with mean 1 and variances σ_j^2, then

$$\bar{z}_0(a) = \frac{1}{1 + \sigma_0^2 \sum_{j=1}^{n} w_j H_j(a)} \tag{54a}$$

and

$$\bar{z}_j(a) = \frac{1}{1 + \sigma_j^2(1 - w_j)H_j(a)} \qquad j = 1, \ldots, n \tag{54b}$$

If w_k is greater than zero, then reducing the incidence of cause of death k will increase $\bar{z}_0(a)$. This increase in $\bar{z}_0(a)$ will, if w_j is greater than zero, result in an increase in the observed incidence of cause of death j. Indeed, if $H_k(a)$ is reduced by δ_k, then $\bar{\mu}_j(a)$ will increase by

$$\frac{\delta_k w_k w_j \mu_j(a)}{\left[1 + \sigma_0^2 \sum_{j=1}^{n} H_j(a)w\right]\left[1 + \sigma_0^2 \sum_{j=1}^{n} H_j(a)w_j - \delta_k w_k\right]} \tag{55}$$

In short, when relative risks from different causes of death are positively correlated, progress against one cause of death may lead to observed increases in the rates of other causes of death.

WHEN THE RELATIVE RISKS OF INDIVIDUALS CHANGE

So far it has been assumed that an individual is born at some level of relative risk and remains at that level for life. Clearly, however,

individuals' relative-risk levels may in some situations change significantly in time. Sometimes this change is caused by factors, such as improvements in living conditions or progress in medical technology, that may affect individuals proportionately to their current relative-risk levels. That is, for all individuals,

$$dz(a)/da = -\varphi(a)z(a) \qquad z(0) = z_0 \tag{56a}$$

where $z(a)$ is an individual's relative risk at time a and where $\varphi(a)$ measures the intensity of the change. Alternatively, the value of $z(a)$ could be given by

$$z(a) = g(a)z_0 \tag{56b}$$

where z_0 is an individual's relative risk at birth and $g(a)$ measures the cumulative change. The values of $\varphi(a)$ and $g(a)$ are related by

$$g(a) = \exp\left[-\int_0^a \varphi(s)ds\right] \tag{57}$$

Because $\mu(a,z)$ equals $z\mu(a)$, it follows that

$$\mu[a,z(a)] = z_0 g(a)\mu(a) \tag{58}$$

Let

$$\mu^*(a) = g(a)\mu(a) \tag{59}$$

The function $\mu^*(a)$ can be interpreted as describing the trajectory of death rates for the standard individual under the changing conditions described by $g(a)$. Then the fundamental equation (7) becomes

$$\bar{\mu}(a) = \mu^*(a)\bar{z}^*(a) \tag{60}$$

where, analogously to previous formulas,

$$\bar{z}^*(a) = \frac{\displaystyle\int_0^\infty z f_0(z) \exp\left[-\int_0^a \mu^*(t)dt\right]dz}{\displaystyle\int_0^\infty z f_0(z) \exp\left[-\int_0^a \mu^*(t)dt\right]} \tag{61}$$

In short, by combining the function $g(a)$ with $\mu(a)$, all the mathematical apparatus derived earlier can still be applied.

CONCLUSION

Individuals, whether people, plants, animals, or machines, differ from one another. Sometimes the differences affect the probability of some major transition, such as dying, moving, marrying, or converting. In this case the observed dynamics of the behavior of the surviving population — the population that has not yet made the transition — will systematically deviate from the dynamics of the behavior of any individual in the population. Most of the examples and terminology of this chapter were drawn from the study of human mortality, but the mathematics can be applied to various heterogeneous populations for such purposes as explaining population patterns, making inferences about individual behavior, and predicting or evaluating the impact of alternative control mechanisms, policies, and interventions.

Among the interesting results discussed in this chapter are:

- Death rates for individuals increase more rapidly than the observed death rates for cohorts.
- Observed mortality convergences and crossovers, both between populations and between causes of death, may be artifacts of heterogeneity.
- Progress in reducing mortality at younger ages or from some causes of death may increase observed mortality at older ages or from other causes of death.
- Slow but accelerating rates of mortality progress in old age may be an artifact of heterogeneity with a significant consequence: The elderly population may be substantially larger in the future than currently predicted.

APPENDIX: THE COMPETING RISK CASE

Let frailty be the vector $\mathbf{z} = (z_1, z_2, \ldots, z_n)$. Denote by T_j the random death times caused by frailty $\mathbf{z}_j$, where $j = 1, 2, \ldots, n$, and let $T = \min T_j$, $i = 1, 2, \ldots, n$. Let the density function of T when frailty $\mathbf{z}$ is given be

$$\varphi(t|z) = \left[\sum_{i=1}^{n} z_i \mu_i(t)\right] \exp\left[-\sum_{i=1}^{n} z_i \int_0^t \mu_i(a)da\right]$$

Note that from this formula it follows that

$$P(T > a|z) = \exp\left[-\sum_{i=1}^{n} z_i \int_0^a \mu_i(t)dt\right]$$

As in the scalar case note that

$$\bar{\mu}(a) = \frac{dP(T \leq a)/da}{P(T > a)}$$

Denoting by $f(a)$ the density probability function of vector $\mathbf{z} = (z_1, \ldots, z_n)$, we have

$$\bar{\mu}(a) = \frac{\int_0^\infty [dP(T \leq x|z)/da]f(z)dz}{P(T > a)}$$

or using the formula for $\varphi(tz)$,

$$\bar{\mu}(a) = \frac{\int_0^\infty \left[\sum_i z_i\mu_i(a)\right] \exp\left[-\sum_{i=1}^{n} z_i \int_0^a \mu_i(t)dt\right] f(z)dz}{P(T > a)}$$

Noting that

$$\frac{\exp\left[-\sum_{i=1}^{n} z_i \int_0^a \mu_i(t)dt\right] f(z)}{P(T > a)} = f_a(z)$$

where $f_a(z)$ is the conditional probability density function of vector frailty $\mathbf{z} = (z_1, \ldots, z_n)$ when the event $\{T > x\}$ is given, we get the following for $\bar{\mu}(a)$:

$$\bar{\mu}(a) = \sum \mu_i(a)\hat{z}_i(a)$$

where

$$\hat{z}_j(a) = E\{\mathbf{z}_j|T > a\}$$

It is essential to know when $\hat{z}_j(a)$ coincides with $\bar{z}_j(a)$, where $\bar{z}_j = E\{\mathbf{z}_j|T_j > a\}$ is the conditional frailty that was defined before. For this purpose note that the random event $\{T > a\}$ may be represented as

$$\{T > a\} = \bigcap_{i=1}^{n} \{T_i > a\}$$

The equality $\hat{z}_j(a) = \bar{z}_j(a)$ means that

$$E(\mathbf{z}_i | \bigcap_i \{T_i > a\}) = E(\mathbf{z}_i | T_i > a)$$

The last equality may take place only in the case when frailty $\mathbf{z}_j$ for any j does not depend on T_k, where $j \neq k$ and $j, k = 1, 2, \ldots, n$.

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