

The Impact of Heterogeneity in Individual Frailty on the Dynamics of Mortality

James W. Vaupel; Kenneth G. Manton; Eric Stallard

Demography, Vol. 16, No. 3. (Aug., 1979), pp. 439-454.

Stable URL:

http://links.jstor.org/sici?sici=0070-3370%28197908%2916%3A3%3C439%3ATIOHII%3E2.0.CO%3B2-9

Demography is currently published by Population Association of America.

Your use of the JSTOR archive indicates your acceptance of JSTOR's Terms and Conditions of Use, available at http://www.jstor.org/about/terms.html. JSTOR's Terms and Conditions of Use provides, in part, that unless you have obtained prior permission, you may not download an entire issue of a journal or multiple copies of articles, and you may use content in the JSTOR archive only for your personal, non-commercial use.

Please contact the publisher regarding any further use of this work. Publisher contact information may be obtained at http://www.jstor.org/journals/paa.html.

Each copy of any part of a JSTOR transmission must contain the same copyright notice that appears on the screen or printed page of such transmission.

JSTOR is an independent not-for-profit organization dedicated to and preserving a digital archive of scholarly journals. For more information regarding JSTOR, please contact support@jstor.org.

THE IMPACT OF HETEROGENEITY IN INDIVIDUAL FRAILTY ON THE DYNAMICS OF MORTALITY

James W. Vaupel

Institute of Policy Sciences and Public Affairs, Duke University, Durham, North Carolina 27706

Kenneth G. Manton

Center for Demographic Studies, Duke University, Durham, North Carolina 27706

Eric Stallard

Center for Demographic Studies, Duke University, Durham, North Carolina 27706

Abstract—Life table methods are developed for populations whose members differ in their endowment for longevity. Unlike standard methods, which ignore such heterogeneity, these methods use different calculations to construct cohort, period, and individual life tables. The results imply that standard methods overestimate current life expectancy and potential gains in life expectancy from health and safety interventions, while underestimating rates of individual aging, past progress in reducing mortality, and mortality differentials between pairs of populations. Calculations based on Swedish mortality data suggest that these errors may be important, especially in old age.

INTRODUCTION

That individuals differ substantially in their endowment for longevity is well known (e.g., Strehler, 1977; Keyfitz, 1978), yet currently used life table methods ignore this heterogeneity. A handful of recent papers (e.g., Shepard and Zeckhauser, 1975, 1977; Tolley et al., 1978; Manton and Stallard, 1979) have focused on the effects on human survival of differences in individual susceptibility to specific causes of death. In this paper, life table methods are developed for populations whose members differ in their general susceptibility to all causes of death. The methods are then used to explore the impact of such heterogeneity in frailty on the dynamics of total mortality.

The model yields insights with intriguing and potentially important implications:

- (1) Mortality rates for individuals may increase faster with age than observed mortality rates for cohorts.
 - (2) The life expectancy of those whose

lives might be saved by some health or safety intervention may be less than currently estimated.

- (3) Past progress against mortality may be underestimated, and as a consequence, predictions of future progress against mortality may be too low.
- (4) Current methods of computing life tables may confound past mortality experiences with current ones. Indeed, if current mortality levels remained unchanged mortality rates presented in life tables, as currently calculated, might increase in the future.
- (5) Heterogeneity in frailty may be a factor in observed declines and reversals with age of mortality differentials between pairs of populations.

This paper is organized into four sections. The first contains a model of individual differences in frailty. Frailty is defined and then assumptions are made concerning the distribution of frailty in populations.

The second section of the paper ex-

plains how mortality rates for individuals at any specified level of frailty can be estimated on the basis of the mortality rates calculated for the cohort to which the individuals belong. Next, as an illustration. individual and cohort mortality rates are compared using data for Swedish females born in 1875. It is argued that it is more informative to measure historical progress in reducing mortality on the basis of changes in the force of mortality for individuals at specific levels of frailty rather than changes in cohort mortality rates. Swedish mortality data are again used to illustrate the empirical differences between these two kinds of measures.

The third section of the paper focuses on period life tables. If heterogeneity in frailty is substantial, standard life table methods will not correctly represent the current pattern of mortality. We propose a method for calculating adjusted period life tables that do reflect current mortality, given population heterogeneity. The standard and adjusted period life tables are compared for Swedish females in 1975.

In the final section of the paper, we extend the methodology to analyze mortality differentials between two populations. To illustrate the effect of population heterogeneity on such differentials we contrast population versus individual mortality rates for Swedish females and males in 1975.

A MODEL OF INDIVIDUAL DIFFERENCES IN FRAILTY

The Definition of Frailty

Let $\mu_i(x, y, z)$ be the force of mortality for an individual—in population group i, at exact age x, at some instant in time y, and with a "frailty" of z. Demographers have traditionally studied how mortality rates vary across populations, by age, and over time; what is unusual about this formulation is the inclusion of a fourth variable z to allow for individual differences in mortality rates. This "frailty" variable could be defined in numerous ways; we have chosen to define it in terms of the following relationship:

$$\mu_l(x, y, z)/\mu_l(x, y, z') = z/z',$$
 (1a)

or, alternatively,

$$\mu_i(x, y, z) = z \cdot \mu_i(x, y, 1)$$
. (1b)

An individual with a frailty of 1 might be called a "standard" individual. Then, an individual with a frailty of 2 is twice as likely to die, at any particular age and time, as the standard individual; an individual with a frailty of 1/2, on the other hand, is only one-half as likely to die.

We chose to define frailty in terms of the force of mortality, μ , rather than the age-specific probability of death, q_x , because of two major difficulties in defining frailty in terms of q_x . First, since q_x is bounded above by one, the range of z would necessarily also be bounded above. Second, q_x is known to be a nonlinear function of the size of the age interval used. Consequently, we will develop the model in terms of μ , and later give the necessary equations to calculate the q_x 's for life table construction.

Note that the definition of frailty assumes that each individual is born at a certain level of relative frailty and stays at this level all his or her life. The definition does not imply, however, that individuals at the same level of frailty are identical even if they are contemporaries from the same population. The variable μ merely measures the likelihood of death; the exact moment of death will be determined by various individual differences beyond population group, age, date of birth, and frailty level. Frailty, as used here, is just one component—and a very special ageinvariant one—of an individual's complex makeup.

The assumption that frailty is constant for individuals is a first approach to acknowledging, in life table computations, the known heterogeneity in populations. A more complete model would recognize (a) that frailty is probably a result of a large number of factors, (b) that frailty is probably not constant for life, (c) that frailty should probably include differential susceptibility to cause-specific mortality, and (d) that mortality due to chronic

diseases (in particular) is the end result of a process which may involve different components of frailty at different stages. Such a model has been proposed by Woodbury and Manton (1977) for the analysis of longitudinal data on coronary heart disease. The mathematical complexity and the lack of appropriate data (e.g., individual medical records) preclude the use of such a model in demographic analysis of population mortality. To the extent that the biological basis of many chronic diseases is associated with intrinsic age-related changes and to the extent that most mortality occurs at advanced ages, it seems plausible that even given the inadequacies of the assumption that frailty is constant, its use is a reasonable first step in modifying standard life table techniques.

For simplicity, subscripts and arguments will be dropped throughout this paper whenever they are not essential to convey meaning. For example, $\mu_l(x, y, z)$ will generally be written as $\mu(z)$ and $\mu_l(x, y, 1)$ as $\mu(1)$ or just μ . Following this convention, equation (1b) reduces to:

$$\mu(z) = z \cdot \mu \ . \tag{1c}$$

Let $H_i(x, y, z)$ be the cumulative hazard of mortality an individual in some population group i of frailty z who is born at time y - x will face up through age x. That is, let

$$H_{l}(x, y, z) = \int_{0}^{x} \mu_{l}(t, y - x + t, z)dt.$$
 (2a)

Clearly, using the convention of simplified notation,

$$H(z) = z \cdot H . \tag{2b}$$

Define $s_i(x, y, z)$ as the probability that an individual will survive to age x. It is well known that

$$s = e^{-H}. (3)$$

Consequently, it follows from equation (2b) that

$$s(z) = s^z, (4)$$

(where s = s(x, y, 1) for some x and y).

Thus if a standard individual has a 50 percent chance of surviving to some age, an individual with a frailty of 2 will only have a 25 percent chance of surviving to this age and an individual with a frailty of 3 only a 12.5 percent chance.

The Distribution of Frailty

Let $\bar{\mu}_i(x, y)$ be the force of mortality for a cohort of individuals from population group i at age x at time y. That is, let

$$\bar{\mu}_l(x, y) = \int_0^\infty \mu_l(x, y, z) \cdot f_x(x) dz. \quad (5)$$

where $f_x(z)$ is the p.d.f. (probability density function) of frailty at age x among the surviving individuals in the cohort. (That the force of mortality for the cohort is indeed the same as the average force of mortality for the surviving individuals in the cohort is proven in the fourth section of the Appendix.) Average frailty in the cohort, \bar{z} , is defined by

$$\bar{z}_t(x, y) = \int_0^\infty z \cdot f_x(z) dz.$$

Consequently, it follows from the definition of frailty in equation (1b) that

$$\bar{\mu}_i(x, y) = \mu_i(x, y, 1) \cdot \bar{z}_i(x, y),$$
 (6a)

or, in simpler notation,

$$\bar{\mu} = \mu \cdot \bar{z}. \tag{6b}$$

Frail individuals with high values of z will tend to die first. Thus, \bar{z} , the average frailty of the surviving cohort, will decline with age. Consequently, equation (6b) implies that the force of mortality for individuals increases more rapidly than for the cohort the individuals belong to: in this sense, individuals "age faster" than cohorts. An intriguing implication is that studies of human aging based on cohort mortality data may be systematically biased or based on erroneous functional forms.

The precise nature of the relationship between individual and cohort aging depends on the distribution of frailty among individuals. This paper assumes that frailty at birth is gamma distributed, with p.d.f.:

$$f_0(z) = \lambda^k \cdot z^{k-1} \cdot e^{-\lambda z}/\Gamma(k),$$
 (7)

where λ and k are the parameters of the distribution. The mean and variance of a gamma variate are given by:

$$\bar{z} = k/\lambda$$
 (8a)

and

$$\sigma^2 = k/\lambda^2. \tag{8b}$$

Figure 1 plots the shape of gamma p.d.f.'s for three values of k that will be used in the empirical sections of this study: k = 1, 4, and 8. These three values were selected because they represent a broad range of distributions of frailty. A value of k of 1 may at first seem extreme, but some empirical research on mortality crossovers (Manton et al., 1979) suggests values of k around 1 and some empirical work with certain diseases, e.g., lung cancer (Manton and Stallard, 1979) suggests values of k much less than 1. In each case, the mean \bar{z} is set equal to 1, so that $\lambda = k$ and $\sigma^2 = 1/k$.

The gamma distribution was chosen because it is analytically tractable and readily computable. It is a flexible distribution that takes on a variety of shapes as k varies: when k = 1, it is identical to the well-known exponential distribution; when k is large, it assumes a bell-shaped form reminiscent of a normal distribution. Frailty cannot be negative and the gamma distribution is, along with the log-normal and Weibull distribution, one of the most commonly used distributions to model variables that are necessarily positive. At least one other study of heterogeneity (Shepard and Zeckhauser, 1977) also uses the gamma distribution for these various reasons.

It turns out, as shown in the first section of the Appendix, that the assumption that frailty at birth is gamma distributed yields some useful mathematical results, including:

(1) Frailty among the survivors at any age x is gamma distributed with the same value of the shape parameter k as at birth. The value of second parameter, however, is now given by

$$\lambda(x) = \lambda + H(x). \tag{9a}$$

The mean frailty of the survivors is therefore given by

$$\bar{z}(x) = \bar{z} \cdot k/(k + \bar{z} \cdot H(x)), \quad (9b)$$

where \bar{z} is the mean frailty of the cohort at birth (as given in equation 8a). When k = 1 and \bar{z} , which is essentially an arbitrary scaling value, is set equal to 1, equation (9b) reduces to

$$\bar{z}(x) = 1/(1 + H(x)).$$
 (9c)

This equation clearly illustrates how the average frailty of a cohort decreases as the cumulative hazard suffered by the cohort increases.

(2) Frailty among those who die at any age x is also gamma distributed, with the same parameter $\lambda(x)$ as among those surviving to age x but with shape parameter k+1. This implies that the mean frailty of those who die, \bar{z}' , is somewhat greater than the mean frailty of the survivors:

$$\bar{z}'(x) = \bar{z}(x) \cdot (k+1)/k.$$
 (10)

This result may prove useful in refining calculations of the benefits of programs to "save" lives (or, more precisely, to delay deaths).

Computing Individual Life Tables from Cohort Life Tables

If frailty is gamma distributed, a simple formula (derived in the second section of the Appendix) relates the force of mortality for an individual at any age x and any level of frailty z to the cohort force of mortality:

$$\mu(x, z) = \bar{\mu}(x) \cdot (z/\bar{z}(0)) \cdot (\bar{s}(x))^{-1/k}$$
 (11)

where $\bar{z}(0)$ is the mean frailty of the cohort at birth and $\bar{s}(x)$ is the proportion of the cohort surviving at age x. For theoretical purposes equation (11) defines the relationship between individual and cohort mortality, but it is inconvenient for empirical calculations since mortality rates are published in terms of cohort age-specific probability of death, \bar{q}_x , rather than cohort force of mortality $\bar{\mu}(x)$. Fortunately, it is possible (as shown in the Appendix) to compute $q_x(z)$, the age-specific probability of death for an individual of frailty z, from data on cohort survival, \bar{s}_x :

$$_{n}q_{x}(z) = 1 - \exp\{-k \cdot (z/\bar{z}(0)) \cdot (\bar{s}(x+n)^{-1/k} - \bar{s}(x)^{-1/k})\}.$$
 (12)

And $\bar{s}(x)$ can be calculated, at any exact age x, from cohort age-specific mortality rates:

$$\bar{s}(0) = 1$$

and

$$\bar{s}(x) = \prod_{t=0}^{x-1} (1_{t} - q_{t}) \qquad x \ge 1. \quad (13)$$

The parameter $\bar{z}(0)$, which measures the mean frailty of the cohort at birth, is essentially just a scaling factor that, for most purposes, can simply be set equal to one. That leaves a single parameter k, which measures the degree of heterogeneity in frailty: the greater k, the less heterogeneity. Given an estimate of k, equations (12) and (13) allow for the translation from published cohort life tables to life tables for individuals at any specific level of frailty.

Cohort vs. Individual Mortality for Swedish Females

To illustrate the nature of the difference between cohort and individual mortality, it is useful to look at some empirical results. We decided to base the empirical calculations in this paper on data for Swedish females and males because high quality mortality data are available for nearly two centuries for these populations. Since the results presented here are intended to be illustrative rather than definitive, we have relegated discussion of the sources of these data and various interpolations and calculations performed on them to a separately available working paper (Vaupel et al., 1979).

Figure 2 compares cohort mortality rates with mortality rates for individuals of standard frailty (i.e., z = 1), at three values of k: k = 1, 4, and 8. The mortality

rates pertain to Swedish females born in 1875.

Figure 1 plotted the gamma p.d.f. for these three values of k. When k is infinite, there is no variability in frailty and cohort and individual mortality rates are identical.

Note in Figure 2 that as k increases (i.e., as variability in frailty decreases), mortality rates for standard individuals become more like the observed cohort rates. Also, note that the effects of selection increase with age. The most striking feature of these plots is the rapid increase in mortality rates when k = 1, 4, or even 8. This implies that if heterogeneity in frailty is substantial, the maximum life span of an individual of a given frailty is well determined within a few years.

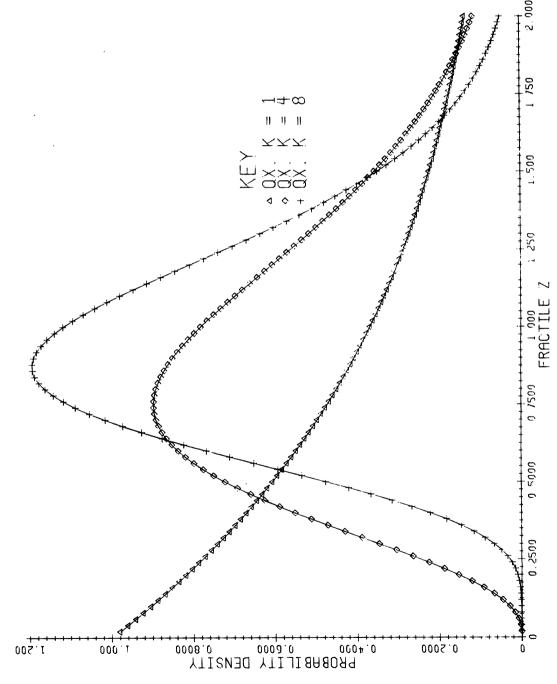
Figure 3 compares cohort mortality rates, \bar{q} , with individual mortality rates, q, for individuals at four levels of relative frailty: z = 1/4, 1/2, 1, and 2. In calculating each of the four individual mortality curves, k was assumed to equal 1. The curves, as before, are based on estimates of the mortality experience of Swedish females born in 1875.

The four individual mortality curves plotted in Figure 3 clearly illustrate the effect of relative frailty on individual mortality. The manner in which the cohort mortality curve cuts through the individual curves at lower and lower values of frailty demonstrates the fact that as death selectively removes the relatively frail, the average frailty of a cohort decreases. Cohort mortality rates thus increase less rapidly than mortality rates for *any* individual in the cohort.

How Much Progress Has Been Made in Reducing Mortality?

Over the last century or two, mortality rates at most ages have declined at the same time that the proportion of cohorts that reach any particular age has increased. Customary measures of progress consider only changes in cohort mortality—these measures ignore increases in survivorship. Consequently, to the extent that heterogeneity in frailty is significant,







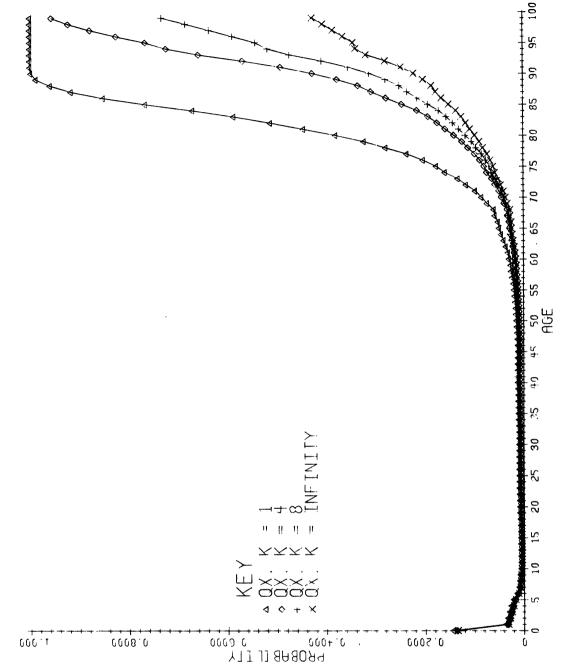
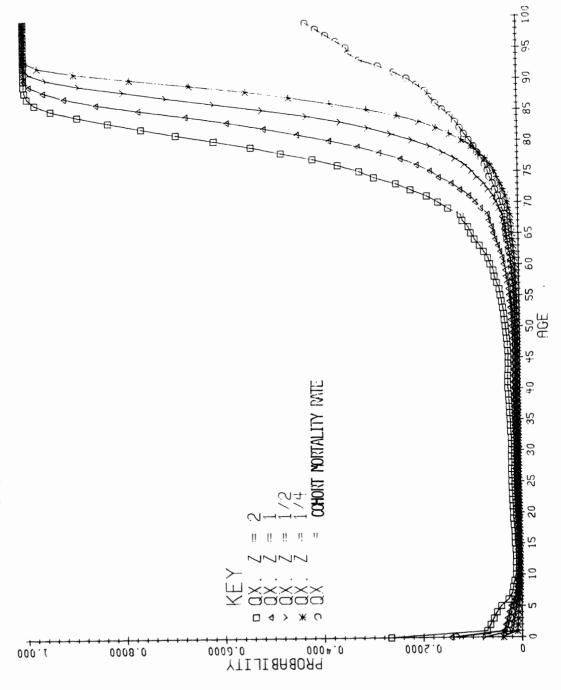


FIGURE 3: MORTALITY RATES FOR INDIVIDUALS OF DIFFERENT FRAILITY (Z = 2, 1, 1/2, 1/4) FROM FRAILITY DISTRIBUTION WITH K=1.



progress against mortality is being underestimated.

Instead of measuring progress in terms of cohort mortality rates, it may be more appropriate to measure such progress in terms of the force of mortality for standard individuals. Table 1 compares these two approaches by measuring Swedish mortality progress from 1875 to 1975 (for females) in terms of the likelihood of death at various ages, under different assumptions about heterogeneity, namely, k = 1, 4, 8, and ∞ .

In the table, mortality rates in 1875 and 1975 are measured, as is customary, in terms of q, but progress against mortality is measured not by the ratio q(1875)/q(1975) but rather by the ratio $\mu(1875)/\mu(1975)$, where the values of μ are estimated values for mid-year, (i.e., μ is estimated by H(x+1)-H(x)). The ratio used seemed more appropriate for two reasons:

(1) The definition of μ , as given in equation (1a), implies that

$$\mu(x, 1875, z)/\mu(x, 1975, z) =$$

$$\mu(x,1875,z')/\mu(x,1975,z'),$$

at any age x, for any pair of frailty values z and z'. Thus, the ratio of the μ 's measures progress against mortality at any level of frailty: this is not true for the ratio of the q's.

(2) In youth and middle age, when μ and q are close to zero, μ approximately equals q; in old age, however, μ , which is not bounded by 1, can greatly exceed q. As a result, progress that substantially reduces μ may have much less effect on q. For example, consider a reduction in μ from 2 to 1: if these values of μ stayed constant over the course of a year, q would only be reduced from 0.86 to 0.63. We felt that a substantial reduction in the likelihood of death at any point in time (as measured by μ) did indeed represent substantial progress against mortality.

Note in Table 1 that at any age and value of k progress for individuals is greater than the synthetic measure of progress based on the cohort force of mortality. The relative difference is strik-

ing in old age with substantial heterogeneity in frailty. For example, with k=1, the force of mortality for individuals aged 84 in 1875 was 8.1 times greater than in 1975, although the force of mortality for the cohort was only 60 percent greater. Even for k=8 the force of mortality for individuals aged 84 in 1875 was twice as great as in 1975.

Also note that for cohorts the ratio of the force of mortality in 1875 to the force of mortality in 1975 declines dramatically with age, but for individuals the decline is less. Indeed, for the extreme case of k=1 the amount of progress made in reducing mortality for individuals *increases* after age 60. It may be, contrary to assertions made on the basis of cohort mortality data, that considerable progress has been achieved in reducing the force of mortality in old age.

Such progress has not resulted in large declines in q_x or gains in life expectancy \mathcal{E}_x because the values of μ in old age remain quite high. For example, the eightfold reduction, when k=1, in the force of mortality at age 84 represents a decline for an individual of standard frailty from a force of mortality of 4.2 to the still substantial level of 0.52. As indicated in the table the corresponding values of q fell only by roughly a factor of 2 from about 0.98 in 1875 to about 0.41 in 1975. If, over the course of the next century, the force of mortality for individuals at age 84 could again be reduced by a factor of eight, the force of mortality would fall from 0.52 to 0.065. The corresponding q's would then show a striking decline—from 0.41 to only 0.063.

The belief is widely held that progress against mortality in old age has been and will continue to be slow. These calculations suggest the possibility that this belief is overly pessimistic.

PERIOD LIFE TABLES

Determining Current Mortality Rates

What if progress in reducing mortality ceased? That is, suppose that

Table 1.—Progress Against Mortality for Swedish Females from 1875 to 1975

	1 k = 8	18.0 30.2 14.5 11.6 8.1 4.0 3.4 2.9 2.3
for:	$\frac{\text{Individual}}{k=1} $	18.1 31.3 15.2 12.1 8.5 4.2 3.7 2.7
1(1875)/u(1975) for:	k = 1	19.1 38.7 19.5 11.5 5.9 5.8 6.2 7.1
ц (1875)	차 8	17.8 29.2 13.9 11.0 7.7 3.8 3.1 2.6 2.0
	* 8	0.00802 0.00020 0.00040 0.00123 0.00317 0.00743 0.0763
	$\frac{\text{Individual}}{k=1} k=4$	0.00803 0.00020 0.00040 0.00071 0.00124 0.00323 0.00766 0.02766
975 for:	$\frac{1}{k} = 1$	0.00805 0.00020 0.00041 0.00073 0.00132 0.00357 0.03352 0.18537
1 ⁴ x in 1975 for:	** = 8	0.00802 0.00020 0.00040 0.00070 0.00122 0.00720 0.00720 0.02108
	* *	0.13491 0.00603 0.00580 0.00810 0.01263 0.02498 0.06291 0.16912
19x in 1875 for:	dividual k = 4	0.13605 0.00625 0.00607 0.01054 0.01358 0.02774 0.077442
	In k = 1	0.14312 0.00771 0.00795 0.01157 0.01505 0.02102 0.05187 0.19059 0.76864
	치 8	0.13379 0.00582 0.00555 0.00770 0.0036 0.01174 0.05248 0.05341 0.12883
	Age	0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0

$$\mu(x,y,z) = \mu(x,y^0,z) \text{ , all } x,$$
 all z, all $v > v^0$.

where y^0 is the point in time when progress ceases. In such a steady-state situation, the future values of cohort mortality rates (as measured by $\bar{\mu}$ or \bar{q} will, surprisingly enough, increase to levels greater than their current levels.

To see this, let $\tilde{\mu}$ and \tilde{s} represent the values of $\tilde{\mu}$ and \tilde{s} in the long run. It follows from equation (11) that

$$\tilde{\mu} = \mu \cdot \bar{z}(0) \cdot \tilde{s}^{1/k}. \tag{14}$$

Solving equations (11) and (14) for μ yields, respectively,

$$\mu = \bar{\mu}/(\bar{z}(0) \cdot \bar{s}^{1/k})$$

and

$$\mu = \tilde{\mu}/(\tilde{z}(0) \cdot \tilde{s}^{1/k}).$$
 (15)

Equating the right-hand side of these equations and solving for $\tilde{\mu}$ then yields:

$$\tilde{\mu} = \tilde{\mu} \cdot (\tilde{s}/\tilde{s})^{1/k}.$$

This equation makes apparent the relationship between $\bar{\mu}$, the currently observed cohort force of mortality, and $\tilde{\mu}$, the cohort force of mortality at current mortality rates, i.e., the cohort force of mortality that would be eventually observed if current levels of mortality remained unchanged. In particular, to the extent progress has been achieved in the past in reducing mortality rates, a greater proportion of individuals will survive to any particular age than before, i.e., § will be greater than s. As a result, future populations at any age will tend to be frailer on average than current populations. The equation indicates that unless future progress in reducing mortality rates is sufficient to counterbalance this effect, future mortality rates will rise-even if some progress is actually being made. As in Lewis Carroll's Through the Looking Glass, it may take "all the running you can do to keep in the same place."

Adjusted Period Life Tables

Period life tables are designed to represent current patterns of population mor-

tality. Standard life table methods construct period life tables according to the same basic set of equations as cohort life tables. In particular, period (and cohort) life tables are based on the cohort agespecific probabilities of death, the \bar{q}_x 's. As indicated above, however, the frailty hypothesis suggests that the values of $\bar{\mu}(x)$ and hence the values of q_x as well—are really a mixture of current and past mortality experiences. Thus the values of \tilde{q} , not \bar{q} , are the correct mortality rates for a period life table, i.e., the mortality rates that a newborn cohort would experience as it aged if current patterns of mortality remained unchanged.

As shown in the Appendix, it is possible to compute \tilde{q}_x on the basis of the following formula:

$$n\tilde{q}_x = 1 - \{1 + (\tilde{s}(x)/\tilde{s}(x))^{1/k} \cdot [1/(1 - n\tilde{q}_x)^{1/k} - 1]\}^{-k}.$$
 (16)

The values of \bar{q}_x can be obtained from standard period or cohort life tables and values of cohort survivorship, $\bar{s}(x)$, at any exact age x, can be calculated from cohort life tables as indicated in equation (13). The values of $\tilde{s}(x)$, which represent period survivorship and thus are analogous to the values often designated by ℓ_x or $\ell(x)$, can be iteratively calculated as follows:

$$\tilde{s}(0) = 1$$
,

and

$$\tilde{s}(x) = \tilde{s}(x-1) \cdot (1-\tilde{q}_{x-1}), x \ge 1.$$
 (17)

Table 2 illustrates the difference it would make to base period life tables on \tilde{q} rather than \bar{q} . The table presents four alternative estimates of the life expectancy of Swedish females in 1975, as currently calculated (under the implicit assumption that $k = \infty$) and as adjusted for k = 1, 4, and 8. Note that the statistics in this table pertain not to the standard individual but to the entire population. The statistics indicate that life expectancy at current mortality rates may be overestimated by customary life table calculations and that this overestimation becomes more significant as heterogeneity increases.

Table 2.—Population	Life	Expectancy	for	Swedish	Females	at	1975	Mortality	Rates,	Under	Various
Assumptions About Heterogeneity											

	Currently				
	Calculated:	Adjusted:	Adjusted:	Adjusted:	
Age 	k = ∞	k = 1	k = 4	k = 8	
0	78.15	76.36	77.52	77.81	
5	73.90	72.09	73.26	73.55	
10	68.98	67.16	68.33	68.63	
15	64.07	62.25	63.42	63.72	
20	59.20	57.37	58.55	58.84	
25	54.30	52.48	53.65	53.95	
30	49.43	47.60	48.78	49.07	
35	44.59	42.76	43.93	44.23	
40	39.80	37.97	39.15	39.45	
45	35.09	33.26	34.44	34.74	
50	30.49	28.67	29.84	30.14	
55	26.03	24.22	25.38	25.68	
60	21.70	19.92	21.05	21.34	
65	17.55	15.83	16.91	17.20	
70	13.73	12.10	13.09	13.38	
75	10.34	8.86	9.73	10.00	
80	7.57	6.33	7.00	7.24	
85	5.38	4.41	4.85	5.06	
90	3.81	3.10	3.34	3.52	
95	2.58	2.01	2.16	2.30	
100	1.93	1.40	1.50	1.62	

COMPARISON OF TWO HETEROGENEOUS POPULATIONS

In addition to being useful in understanding patterns of mortality within a single population, the hypothesis of heterogeneity in frailty may explain some puzzling anomalies in period mortality differentials between populations. Variation in the force of mortality in youth or middle age is much more substantial across countries and various population groups than variation among the elderly. For most pairs of populations—e.g., for U.S. whites vs. blacks, for U.S. males vs. females, or for Americans vs. Swedes-mortality differentials tend to decline and even reverse with age (see Thornton and Nam, 1972; Nam and Okay, 1977; Manton et al., 1979; Strehler, 1977). One striking and surprising reversal concerns Puerto Rico which, among countries for which good mortality statistics are available, is the world's leader in life expectancy at age 65 (Vaupel, 1976). Such convergence and

cross-over of mortality differentials might be at least partially caused by decreases in the average frailty of a population cohort at later ages as frailer members are removed by mortality.

To see this, consider two cohorts for which the cohort force of mortality is described by a series of values $\bar{\mu}_1$ and $\bar{\mu}_2$ and the force of mortality for individuals in the cohorts by a series of values μ_1 and μ_2 . Equation (11) implies that at any age and point in time:

$$\bar{\mu}_2/\bar{\mu}_1 = (\mu_2/\mu_1) \cdot (\bar{z}_2(0)/\bar{z}_1(0)) \cdot (\bar{s}_2^{1/k_2}/\bar{s}_1^{1/k_1}). \quad (18a)$$

In the special case where $\bar{z}_1(0) = \bar{z}_2(0)$ and where $k_1 = k_2$, equation (18a) reduces to:

$$\bar{\mu}_2/\bar{\mu}_1 = (\mu_2/\mu_1) \cdot (\bar{s}_2/\bar{s}_1)^{1/k}$$
. (18b)

As shown in the second section of the Appendix,

$$\bar{s} = \{(k/\bar{z}(0))/(k/\bar{z}(0) + H)\}^k$$
. (19)

Consequently, assuming $\bar{z}(0)=1$, equation (18b) can be rewritten as:

$$\bar{\mu}_2/\bar{\mu}_1 = (\mu_2/\mu_1) \cdot (k+H_1)/(k+H_2).$$
 (20)

The second cohort might be called "disadvantaged" relative to the first if μ_2 exceeds μ_1 and if, as a result of previous excess mortality, H_2 exceeds H_1 . Consider the special case where μ_2/μ_1 equals H_2/H_1 : that is, suppose the current level of disadvantage is the same as the overall historical level. In this case, equation (20) implies that as H_1 and H_2 increase with age, $\bar{\mu}_2$ will approach $\bar{\mu}_1$; i.e., the cohort mortality rates will converge. There will not, however, be any cross-over: $\bar{\mu_2}$ will never fall below $\bar{\mu}_1$. For a cross-over to occur the current mortality differential for individuals must be somewhat less than the overall differential in the past; the precise condition is

$$\mu_2/\mu_1 < (k+H_2)/(k+H_1)$$
.

A simple example illustrates these dynamics. Suppose that H_2/H_1 equals 2, so that historically the disadvantaged cohort has suffered twice the force of mortality of the advantaged cohort. Suppose that H_1 equals 1. And suppose that k=1. In this case, equation (19) indicates that one-half of the advantaged cohort and one-third of the disadvantaged cohort are alive. Thus, as can be seen from either equation (18b) or equation (20), the cohort mortality differential, $\bar{\mu}_2/\bar{\mu}_1$, will be two-thirds of the individual mortality differential, μ_2/μ_1 . Consequently, if μ_2/μ_1 like H_2/H_1 equals 2, the cohort differential $\bar{\mu}_2/\bar{\mu}_1$ will be 1.33. If, however, μ_2/μ_1 equals 1.2, $\bar{\mu_2}/\bar{\mu_1}$ will equal 0.8: although individuals at any specific level of frailty in the disadvantaged cohort suffer a 20 percent higher force of mortality than individuals in the advantaged cohort, the disadvantaged cohort will appear to be doing 20 percent better than the advantaged cohort.

Table 3 displays population and individual mortality rates for Swedish males and females in 1975. In calculating the individual rates, it was assumed that frailty at birth for males and females was

identically distributed, with a mean of 1 and a k of 1. A value of 1.0 was selected for k because it produced a relatively constant male/female ratio after age 30, i.e., it eliminated the convergence in the unadjusted cohort data. The value of 1.0 was the largest value of k that eliminated the convergence; smaller values yield divergence. As in Table 1 and for essentially the same reasons, the male and female cohort and individual mortality rates are measured in terms of q, but the comparisons of the rates are in terms of the ratio of μ_m to μ_f .

DISCUSSION

The three variables usually considered in studies of mortality—population group, age, and year—are well defined and readily measurable. The variable "frailty," on the other hand, could be defined in any of a number of ways and, however defined, is difficult to measure. As a consequence, demographers have largely ignored heterogeneity in frailty, presumably in the hope that such neglect would result in estimation errors that are small and centered around zero.

The results of this study, however, suggest that ignoring frailty may result in biased estimates. Individual aging rates, past and future progress in reducing mortality, and mortality differentials between populations may be underestimated. On the other hand, current life expectancy and potential gains in life expectancy from averting specific causes of death may be overestimated. Furthermore, illustrative calculations based on Swedish mortality data suggest the possible magnitude of bias. Given the importance of understanding the dynamics of mortality in demographic and biomedical research and in public policy analysis, the results indicate that heterogeneity in frailty is an area of research that may well prove worthy of considerable attention.

APPENDIX

I. In this study we have defined frailty, z, as a continuous random variable. In mor-

Table 3.—Population and Individual Mortality Rates for Swedish Males vs. Females in 1975

	$k = \infty$ $1^{\overline{q}_X}$ for	$1^{\overline{q}_{_{\mathbf{X}}}}$ for		$k = 1.0$ $1^{q_x \text{ for}}$	1 ^q x for	
Age	males	females	μ̄m/μ̄f	males	females	μ _m /μ
0	0.01022	0.00802	1.28	0.01027	0.00805	1.28
5	0.00050	0.00030	1.67	0.00051	0.00030	1.70
10	0.00030	0.00020	1.50	0.00031	0.00020	1.55
15	0.00074	0.00030	2.47	0.00076	0.00031	2.45
20	0.00120	0.00040	3.00	0.00124	0.00041	3.03
25	0.00118	0.00050	2.36	0.00124	0.00052	2.39
30	0.00126	0.00070	1.80	0.00134	0.00073	1.84
35	0.00156	0.00072	2.17	0.00170	0.00076	2.24
40	0.00258	0.00122	2.12	0.00289	0.00132	2.19
45	0.00354	0.00182	1.95	0.00407	0.00201	2.03
50	0.00584	0.00312	1.87	0.00702	0.00357	1.97
55	0.00928	0.00444	2.10	0.01182	0.00532	2.23
60	0.01448	0.00720	2.02	0.01998	0.00921	2.18
65	0.02424	0.01190	2.05	0.03798	0.01670	2.30
70	0.03840	0.02108	1.84	0.07245	0.03352	2.21
75	0.06306	0.03837	1.66	0.15811	0.07536	2.20
80	0.09974	0.06730	1.51	0.36567	0.18537	2.22
85	0.15386	0.12087	1.30	0.77558	0.50974	2.10
90	0.21759	0.18046	1.23	0.99867	0.95221	2.18

tality analysis age of death, a, may also be considered to be a continuous random variable. This section of the Appendix derives the joint probability density function (p.d.f.) of a and z and various marginal and conditional p.d.f.'s involving a and z.

As indicated in the text, we assume in this study that the marginal p.d.f. of z, $f_z(z)$, is a gamma p.d.f.:

$$f_z(z) = \lambda^k \cdot z^{k-1} \cdot e^{-\lambda z} / \Gamma(k)$$
. (A.1)

The force of mortality by definition is given by:

$$\mu(a,z) = f_{a+z}(a|z)/s(a,z),$$
 (A.2)

where $f_{a|z}(a|z)$ is the *p.d.f.* of *a* conditional on *z*. Solving this equation for $f_{a|z}(a|z)$ and substituting equations (1c), (3), and (4) yields:

$$f_{a|z}(a|z) = z \cdot \mu(a) \cdot e^{-z \cdot H(a)}$$
. (A.3)

The joint p.d.f. of a and z, $f_{a,z}(a,z)$ is the product of the two p.d.f.'s:

$$f_{a,z}(a,z) = f_z(z) \cdot f_{a|z}(a|z)$$
. (A.4)

Hence.

$$f_{a,z}(a,z) = \mu(a) \cdot \lambda^k \cdot z^k \cdot e^{-z \cdot \lambda(a)} / \Gamma(k),$$
(A.5)

where

$$\lambda(a) = \lambda + H(a).$$

Integrating (A.5) with respect to z from 0 to ∞ yields the marginal p.d.f. of a:

$$f_a(a) = \mu(a) \cdot k \cdot \lambda^k / (\lambda(a))^{k+1}. \tag{A.6}$$

Since the p.d.f. of z conditional on a (i.e., the p.d.f. of frailty among those who die at age a) is given by:

$$f_{z|a}(z|a) = f_{a,z}(a,z)/f_a(a),$$
 (A.7)

it follows that

$$f_{z|a}(z|a) = (\lambda(a))^{k+1} \cdot z^k \cdot e^{-\lambda(a) \cdot z} / \Gamma(k+1).$$
(A.8)

This p.d.f. is clearly a gamma p.d.f. with parameters $\lambda(a)$ and k+1.

Because survival at age x implies an age of death greater than x, integrating (A.4) with respect to age of death from x to ∞ , remembering that

$$\int_{x}^{\infty} f_{a/z} (a/z) da = e^{-z \cdot H(x)},$$

and then normalizing the result, yields the p.d.f. of z in the surviving population at age x:

$$f_x(z) = (\lambda(x))^k z^{k-1} e^{-\lambda(x)\cdot z} / \Gamma(k). \tag{A.9}$$

This p.d.f. is also a gamma p.d.f., but with parameters $\lambda(x)$ and k.

II. To derive the individual force of mortality $\mu(x,z)$ as given in equation (11), observe that it follows from (6b) and (8a) that

$$\bar{\mu}(x) = \mu(x) \cdot k/\lambda(x)$$
. (A.10)

By definition the cohort force of mortality is given by:

$$\bar{\mu}(x) = f_a(x)/\bar{s}(x). \tag{A.11}$$

Solving (A.11) for $\bar{s}(x)$ and then substituting (A.6) and (A.10) yields:

$$\bar{s}(x) = (\lambda/\lambda(x))^k$$
. (A.12)

The mean frailty of the cohort at age x can be derived from the parameters of $f_x(z)$ in (A.9):

$$\bar{z}(x) = k/\lambda(x). \tag{A.13}$$

Equations (A.12) and (A.13) together imply that:

$$\bar{z}(x) = \bar{z}(0) \cdot (\bar{s}(x))^{1/k}, \quad (A.14)$$

(since $\lambda = \lambda(0)$.) Solving (6b) for $\mu(x)$ and substituting the result and (A.14) in (1c) yields the form of $\mu(x,z)$ given in (11).

Also note that equations (A.12) and (A.13) and the definition of $\lambda(x)$ as

$$\lambda(x) = \lambda + H(x)$$

imply the formula for \bar{s} as used in equation (19).

III. To derive the individual age-specific probability of death $_nq_x(z)$ given in (12), note that by definition:

$$s(x+n,z) = s(x,z) \cdot (1 - nq_x(z)).$$
 (A.15)

Using the formulas for s in (3) and (4) it follows from (A.15) that:

$$_{n}q_{x}(z) = 1 - \exp\{-(H(x+n,z) - H(x,z))\}.$$
(A.16)

As indicated in (2a), integrating (11) with respect to age from 0 to x, remembering that

$$\bar{s}(x) = \exp \left\{-\int_0^x \bar{\mu}(t)dt\right\},$$

yields the cumulative hazard function for individuals:

$$H(x,z) = k \cdot (z/\bar{z}(0)) \cdot (1/\tilde{s}(x))^{1/k}$$
. (A.18)

When (A.17) is substituted in (A.16), equation (12) is the result.

To derive the adjusted period mortality rates \tilde{q}_x given in (16) observe that the cumulative hazard function for individuals derived from the adjusted period life table can be expressed, by analogy to (A.17), as:

$$H(x,z) = k \cdot (z/\bar{z}(0)) \cdot (1/\tilde{s}(x))^{1/k}$$
. (A.18)

Thus, the individual age-specific probability of death $_nq_x(z)$ given in (A.16) may be evaluated using either (A.17) or (A.18). Equating the two forms and using (13) and (17) to introduce the variables \bar{q} and \tilde{q} yields equation (16).

IV. Equation (5) assumes that the force of mortality for a cohort equals the average force of mortality for the surviving individuals in the cohort. Though this is an intuitively plausible assertion, its truth is not immediately apparent. Here we indicate the required steps for a formal proof.

The force of mortality is defined by (A.11):

$$\bar{\mu}(x) = f_a(x)/\bar{s}(x).$$

The p.d.f. $f_a(x)$ is given by:

$$f_a(x) = \int_0^\infty f_{a,z}(x, z) dz.$$
 (A.19)

Equations (A.4) and (A.3) imply that this can be rewritten as:

$$f_a(x) = \int_0^\infty f_z(z) \cdot z \cdot \mu(x) \cdot s(x, z) dz. \quad (A.20)$$

It is apparent that

$$f_x(z) = f_z(z) \cdot s(x,z)/\bar{s}(x)$$
. (A.21)

Reexpressing (A.20) in terms of $f_x(z)$ and then substituting the result in (A.11) yields:

$$\bar{\mu}(x) = \mu(x) \cdot \int_0^\infty z \cdot f_x(z) dz. \quad (A.22)$$

The integral in (A.22) simply equals $\bar{z}(x)$. Thus, we have equation (6), which can be true if and only if equation (5) is true.

ACKNOWLEDGEMENTS

The authors thank Philip W. P. Yen, Philip J. Cook, and Max A. Woodbury for helpful suggestions. Comments made by the referees also proved to be very useful. The research in this paper was supported by NIA Grant AG 01159-02 and is one of a series on multiple cause mortality sponsored by the Center for Demographic Studies at Duke University.

REFERENCES

Keyfitz, N. 1978. Improving Life Expectancy: An Uphill Road Ahead. American Journal of Public Health 10:954-956.

Manton, K. G., and E. Stallard. 1979. Maximum Likelihood Estimation of a Stochastic Compartment Model of Cancer Latency: Lung Cancer Mortality Among White Females in the U.S. In press: Computers and Bio-Medical Research.

E. Stallard, and J. W. Vaupel. 1979. Ex-

plaining the Black-White Mortality Crossover: A Model of Selection on Heterogeneous Populations. (Unpublished manuscript).

Nam, C. B., and K. A. Okay. 1977. Factors Contributing to the Mortality Crossover Pattern. XVII General Conference of the International Union for the Scientific Study of Population, Mexico City.

Shepard, D. S., and R. J. Zeckhauser. 1975. The Assessment of Programs to Prolong Life, Recognizing Their Interaction with Risk Factors. Discussion paper 32-D, Kennedy School of Government, Harvard University, Cambridge, Mass.

. 1977. Heterogeneity Among Patients As a Risk Factor in Surgical Decision-Making. In J. P. Bunker et al. (ed.), Costs, Risks, and Benefits of Surgery. New York: Oxford University Press.

Strehler, B. L. 1977. Time, Cells, and Aging. New York: Academic Press.

Thornton, R. G., and C. B. Nam. 1972. The Lower Mortality Rates of Non-Whites at the Older Ages: An Enigma in Demographic Analysis. Research Reports in Social Science 11:1-8.

Tolley, H. D., D. Burdick, K. G. Manton, and E. Stallard. 1978. A Compartment Model Approach to the Estimation of Tumor Incidence and Growth: Investigation of a Model of Cancer Latency. Biometrics 34: 377-389.

Vaupel, J. W. 1976. Early Death: An American Tragedy. Law and Contemporary Problems 4:73– 121.

——, K. G. Manton, and E. Stallard. 1979. What Demographic Difference Does It Make That the Frail Die First?: A Model of Mortality and Some Suggestive Results Based on Swedish Mortality Since 1778. Working Paper 179, Institute of Policy Sciences and Public Affairs, Duke University, Durham, N.C.

Woodbury, M. A., and Manton, K. G. 1977. A Random Walk Model of Human Mortality and Aging. Theoretical Population Biology 11:37-48.