

## Inherited Frailty and Longevity

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The life spans of parents and children appear only weakly related, even though parents affect their children's longevity through both genetic and environmental influences. These influences can be summarized as a correlation between parents' and children's frailty. It is shown that even if children perfectly inherit their frailty from their parents, parents' life spans explain little of the variance in children's life spans, because the variance in life expectancies among people with different frailties is small compared with the variance in life spans among people at the same level of frailty. By interpreting frailty as a relative risk in a proportional-hazard model, longevity as a duration or waiting time, and inheritance as an invariance in relative risk over time, one can extend this result to repeatable events involving fertility, migration, marriage, unemployment, and so forth.

Do long-lived parents tend to have long-lived children? Cohen (1964) meticulously examined the extensive research on this question up to the early 1960s, including work by Beeton and Pearson (1899, 1901–1902), Bell (1918), and Pearl (1922, 1931; Pearl and Pearl, 1934). More recent studies include those by Abbott et al. (1978), Welter (1978), Philippe (1980), Crawford and Rogers (1982), Jacquard (1982), Vandenbroucke et al. (1984), and Wolf (in press). Despite folklore typified by Oliver Wendell Holmes's advice that those wishing long lives should "advertise for a couple of parents both belonging to long-lived families" (quoted by Cohen, 1964:131), the relationship between the longevity of parents and their children appears to be weak. For instance, the results of a synoptic synthesis by Welter (1978; reported by Jacquard, 1982:303) suggest that "knowing the parents' ages at death decreases the variance of the son's age at death by . . . 2.6 percent" (i.e.,  $r^2 = 0.026$ ).

This conclusion seems "surprising and paradoxical" (Jacquard, 1982:303) because numerous studies link genes with susceptibility or resistance to various diseases and because parents also influence their children's health through nongenetic transmission of attitudes, behavior, education, wealth, and so on.

One possible explanation is that what children inherit from their parents is not their longevity per se but rather their frailty, that is, a set of susceptibilities and risk factors that alters their chances of death at different ages (Vaupel, Manton, and Stallard, 1979). In the simple case of fixed frailty, frailty is equivalent to relative risk in a proportional-hazard model. Individuals with frailty  $z$  face a force of mortality,  $\mu(x, z)$ , that, at all ages  $x$ , is  $z$  times the force of mortality,  $\mu(x)$ , faced by "standard" individuals with frailty of 1:

$$\mu(x, z) = z\mu(x, 1) = z\mu(x). \quad (1)$$

Thus a person born with a frailty of 2 would suffer twice the chances of death at every age as a standard individual with a frailty of 1. (As discussed subsequently, the distribution of frailty among individuals can be modeled in various ways.)

The central finding of this article is that even in the extreme case in which sons (or daughters) perfectly inherit their fixed frailty from their fathers (or mothers), so that the  $r^2$  between fathers' and sons' frailties is 1.0 at all ages, the  $r^2$  between fathers' and sons' life spans will be close to 0.

Some understanding of this puzzling result can be developed by using Bayes' theorem, the theory of causal modeling, and a decomposition of variance. The discussion section treats this and the fact that  $r^2$  may, for some purposes, be a misleading statistic to report in answering whether long-lived parents tend to have long-lived children.

Finally, in a concluding section on further research, three implications of the analysis are highlighted. First, if frailty rather than longevity per se is inherited, empirical studies, including those that control for all nonfamilial influences, will tend to find low values of  $r^2$  between the life spans, and high values of  $r^2$  between the frailties, of parents and children, twins, and other pairs of relatives. Second, appropriately designed empirical research may shed light on whether both frailty and longevity are inherited and hence on the question of whether life expectancy is limited by some genetically determined maximum life span. Third, by interpreting frailty as a relative risk in a proportional-hazard model, longevity as a duration, and inheritance as an invariance in relative risk over time, one can extend the analysis in this article to a wide variety of processes with successive waiting times in heterogeneous populations, including fertility, migration, marriage, and unemployment processes with repeatable events within lifetimes.

## Methods

Let  $X$  denote the life spans of one of the two parents and  $Y$  the life spans of children. For convenience, the terms "fathers" and "sons" will henceforth be used, but "mothers" or "daughters" could equally well be used. Gender is irrelevant. What is important is that the model assumes that a child inherits his or her frailty from one of his or her parents. Although this assumption is biologically incorrect, it is a useful extreme case to consider in explaining the low correlation between parents' and children's life spans. A more realistic two-sex model is developed at the end of this section.

To avoid complications arising from the interaction of mortality and fertility, assume that each father in the study has one son in the study, that both father and son have life spans longer than some minimum length  $\alpha$ , and that all fathers had their sons by age  $\alpha$ . The value of  $r^2$  is given by the standard formula

$$r^2 = \frac{[E(XY) - E(X)E(Y)]^2}{[E(X^2) - E(X)^2][E(Y^2) - E(Y)^2]}, \quad (2)$$

where  $E$  is the expectation operator. In the special case we consider, in which fathers and sons suffer the same forces of mortality, this reduces to

$$r^2 = \left[ \frac{E(XY) - E(X)^2}{E(X^2) - E(X)^2} \right]^2. \quad (3)$$

In this formula,  $E(X)$  is simply life expectancy at birth (among those surviving at least  $\alpha$  years) and the expression  $E(X^2) - E(X)^2$  gives the variance in life spans at birth (again among those surviving at least  $\alpha$  years).

If frailty  $z$  is continuously distributed over nonnegative values of  $z$ , with density function  $f(z)$  at birth, and  $s(x, z)$  is the survival function

$$s(x, z) = \exp\left[-z \int_0^x \mu(t) dt\right], \tag{4}$$

then

$$E(X) = \int_0^\omega \int_0^\omega x\mu(x, z)s(x, z)f(z) dx dz = \int_0^\omega e_o(z)f(z) dz, \tag{5}$$

where  $e_o(z)$  represents life expectancy at birth among those with frailty  $z$ :

$$e_o(z) = \int_0^\omega x\mu(x, z)s(x, z) dx = \int_0^\omega s(x, z) dx. \tag{6}$$

Similarly,

$$E(X^2) = \int_0^\omega \int_0^\omega x^2\mu(x, z)s(x, z)f(z) dx dz = \int_0^\omega v(z)f(z) dz, \tag{7}$$

where  $v(z)$  is the second moment of life spans among those with frailty  $z$ :

$$v(z) = \int_0^\omega x^2\mu(x, z)s(x, z) dx. \tag{8}$$

Finally,

$$\begin{aligned} E(XY) &= \int_0^\omega \int_0^\omega \int_0^\omega \int_0^\omega xy\mu(x, z_1)\mu(x, z_2)s(x, z_1)s(x, z_2)g(z_2|z_1)f(z_1) dx dy dz_2 dz_1, \\ &= \int_0^\omega \int_0^\omega e_o(z_1)e_o(z_2)g(z_2|z_1)f(z_1) dz_2 dz_1. \end{aligned} \tag{9}$$

In these formulas  $\omega$  is an age beyond which no one lives and  $g(z_2|z_1)$  is the conditional density of a son's frailty  $z_2$  given his father's frailty  $z_1$ . If sons perfectly inherit their frailty from their fathers, so that  $z_2$  and  $z_1$  are identical, then equation (9) reduces to

$$E(XY) = \int_0^\omega e_o(z)^2f(z) dz. \tag{10}$$

When the distribution of frailty is discrete, a summation over the possible values of  $z$  should be substituted in the formulas instead of integration over  $z$ .

For computational convenience assume that the force of mortality follows a Gompertz trajectory after age  $\alpha$ ,

$$\mu(x) = ae^{bx}, \quad x \geq \alpha. \tag{11}$$

Since fathers and sons are assumed to live at least  $\alpha$  years,

$$\mu(x) = 0, \quad x < \alpha. \tag{12}$$

The use of a minimum life span of  $\alpha$  was justified earlier as a means of avoiding interactions between fertility and mortality. Many empirical studies also excluded fathers and sons who did not live longer than some minimum life span; Wilson and Doering (1926), Yuan (1931, 1932), and Abbott et al. (1978), for instance, used a minimum life span of 20. One justification for this is the asymmetry in fathers' and sons' life spans. Under identical mortality regimes, the life expectancy of sons is less than the life expectancy of fathers. Boys must survive to a certain age before they can have sons, but their sons can die in infancy. Use of a minimum life span is sometimes also justified because of poor data on mortality at younger ages or because deaths at younger ages are hypothesized to be attributable to accidental or special causes unrelated to those factors influencing longevity.

Various distributions of frailty have been used (Vaupel and Yashin, 1985a). Two distributions are employed in this article to produce the results presented later: the gamma distribution and a two-point distribution in which half of the population is at one frailty level and the other half at another level. The gamma distribution is often used in studies of mortality in heterogeneous populations (e.g., Beard, 1963; Horiuchi and Coale, 1983; Manton, Stallard, and Vaupel, 1981, 1986; Vaupel, Manton, and Stallard, 1979); the two-point distribution is employed to check whether the choice of a distribution other than the gamma distribution might strongly influence the value of the  $r^2$  between fathers' and sons' life spans.

Both the gamma and the two-point distributions can be characterized by their means and variances. As noted in Vaupel, Manton, and Stallard (1979), the mean of frailty at birth might as well be set equal to 1. The empirical studies of Manton, Stallard, and Vaupel (1981, 1986) and Horiuchi and Coale (1983) suggest that a  $\sigma^2$  value of about 0.25 fits human mortality data. A  $\sigma^2$  value of 0.5 is also used here to explore the effect of the variance in frailty on the correlation between fathers' and sons' life spans. This value of 0.5 is probably unrealistic, at least in the context of a model with gamma-distributed frailty and exponentially increasing mortality rates for individuals. A model with a  $\sigma^2$  of 0.5 yields a prominent and early leveling off in the force of mortality that is not observed in actual human mortality curves.

In the case considered here, in which the force of mortality is assumed to increase exponentially, numerical approximation methods must be employed to estimate equations (5), (7), and (10) and hence equation (3). (This was done on an IBM PC by replacing the integrals with summations.)

Use of the two-point distribution permits easy extension of the model to the case in which both fathers and mothers influence their son's frailty. In the 50 percent of cases in which a father and mother have the same frailty, the son can be assumed to inherit it; in the remaining cases in which a father and mother have different frailties, the son can be assumed to have an equal chance of inheriting his father's or his mother's frailty. Thus altogether there is a 75 percent chance that a son will have the same frailty as his father.

## Results

**Table 1** presents the values of  $r^2$  between fathers' and sons' life spans for various model specifications and for the two  $\sigma^2$  values of 0.25 and 0.5. In addition, life expectancy at birth and the standard deviation in life spans are given for reference, for the population that survived at least  $\alpha$  years.

In the standard model, the force of mortality is set at a moderate level, frailty is gamma distributed, and the minimum life span is 20 years. Variations on this standard model raise or lower the force of mortality, change the minimum life span to 12 or 40, substitute the two-point distribution for the gamma distribution, and generalize the model such that a son's

Table 1. Mean and Standard Deviation (S.D.) of Life Spans and of the  $r^2$  Between Fathers' and Sons' Life Spans, for Various Models and for Two Values of  $\sigma^2$ , the Variance of Frailty

Model	$\sigma^2$			
	0.25		0.5	
	$e_0 \pm \text{S.D.}$	$r^2$	$e_0 \pm \text{S.D.}$	$r^2$
Standard	71.8 ± 13.4	0.02	73.1 ± 14.6	0.08
Standard except:				
Lower $\mu$	80.8 ± 13.6	0.02	82.1 ± 14.7	0.06
Higher $\mu$	49.9 ± 11.8	0.05	51.4 ± 13.1	0.16
Life spans >12	71.7 ± 13.6	0.02	73.0 ± 14.8	0.08
Life spans >40	72.7 ± 12.1	0.05	74.1 ± 13.4	0.16
Two-point distribution	71.9 ± 13.4	0.03	73.9 ± 15.1	0.11
Two-point distribution with two sexes	71.9 ± 13.4	0.007	73.9 ± 15.1	0.03

Notes: In the standard model,  $\mu(x) = 0.00005 \exp(0.1x)$ , frailty at birth is gamma distributed with mean 1 and variance  $\sigma^2$  and persons are considered only if they live 20 years or more. The lower  $\mu(x) = 0.00002 \exp(0.1x)$  and the higher  $\mu(x) = 0.0005 \exp(0.1x)$ . The two-point distribution consists of two equally likely values with mean 1 and variance  $\sigma^2$ . When  $\sigma^2 = 0.25$ , the values are 0.5 and 1.5; when  $\sigma^2 = 0.50$ , the values are 0.293 and 1.707. When there are two sexes, a son has a 75 percent chance of having the same frailty as his father and a 25 percent chance of having the other frailty value. The life expectancy values give the life expectancy of those surviving beyond age 20 (or 12 or 40) plus or minus the standard deviation of the distribution of life spans. See the text for further explanation of the table.

frailty is influenced by both his mother's and his father's frailty. This generalization is, as discussed previously, based on the two-point distribution of frailty.

The central message of Table 1 is that models in which frailty rather than longevity is inherited produce values of the  $r^2$  between fathers' and sons' life spans that are close to 0. The values are higher when the variance in frailty, the mortality rates, and the minimum life span all are high, but the values are low. Use of the two-point distribution instead of the gamma distribution makes little difference. Use of the two-sex model cuts the value of  $r^2$  by roughly a factor of four, to 0.007 when  $\sigma^2$  is 0.25.

The values of the correlation coefficient  $r$  corresponding to the  $r^2$  statistics in the table are, in every instance, the positive square root of the  $r^2$  values. Consequently, there is always some direct relationship between fathers' and sons' longevity. This relationship, however, is weak, even though in all of the models except the last, the correlation between fathers' and sons' frailties is 1.0.

### Discussion

The results demonstrate that the following two contentions can both be true:

1. There is a weak correlation between the life spans of parents and their children.
2. (a) There is substantial heterogeneity in frailty such that some individuals face, at all ages, several times the chance of death as other individuals; and (b) there is a strong correlation between the frailty of parents and their children.

A simple example is helpful in understanding why these two contentions are not inconsistent. Suppose that there is a two-point distribution of frailty with mean 1 and variance 0.25. In this case, half of the father-son pairs have a frailty of 0.5 and the other half a frailty of 1.5, so the high-risk group suffers, at all ages, fully three times the force of mortality as the low-risk group. As summarized in the first two rows of Table 2, the life expectancies of the two groups, under the same standard assumptions as before, are 77.3 and

Table 2. Risk Group, Mean, and Standard Deviation of Life Span and Probability of Death Before 65 and After 80 for Sons of Fathers in Low- or High-Risk Groups and With Various Life Spans

Knowledge about father	Inference about son				
	Probability in		$e_0 \pm$ S.D.	Probability of death	
	Low-risk group	High-risk group		Before 65	After 80
In low-risk group	1	0	77.3 $\pm$ 12.5	0.15	0.47
In high-risk group	0	1	66.5 $\pm$ 12.1	0.39	0.11
Father died at:					
20	0.25	0.75	69.2 $\pm$ 12.2	0.33	0.20
50	0.26	0.74	69.3 $\pm$ 12.2	0.33	0.20
60	0.29	0.71	69.6 $\pm$ 12.2	0.32	0.21
65	0.32	0.68	69.9 $\pm$ 12.2	0.32	0.22
70	0.37	0.63	70.4 $\pm$ 12.2	0.30	0.24
80	0.60	0.40	72.9 $\pm$ 12.3	0.25	0.33
90	0.95	0.05	76.7 $\pm$ 12.5	0.16	0.46
95	0.996	0.004	77.3 $\pm$ 12.5	0.15	0.47
100	1.000	0.000	77.3 $\pm$ 12.5	0.15	0.47
Father's risk group and life span unknown	0.50	0.50	71.9 $\pm$ 12.3	0.27	0.29

Notes: There is a two-point distribution of frailty with half of the father-son pairs at frailty 0.5 and half at 1.5. The standard assumptions of Table 1 hold, with moderate mortality and a minimum life span of 20 years.

66.5 years. The more robust individuals face a 15 percent chance of death before age 65 and a 47 percent chance of surviving past 80; the frailer individuals, in contrast, suffer a 39 percent chance of early death and have only an 11 percent chance of becoming octogenarians.

Note, however, that some robust people die early and some frail people live long. The 11-year gap between the two groups' life expectancies is somewhat less than the standard deviation in life spans. If a father dies, say, at age 50, it cannot be concluded that he was necessarily in the high-risk group.

By employing Bayes' theorem, it is possible to calculate the probability that a son (and his father) is at high (or low) risk given the life span of his father:

$$\Pr(z|X) = \frac{h(X|z)\Pr(z)}{h(X|z)\Pr(z) + h(X|z')\Pr(z')}, \tag{13}$$

where the density function of life spans is given by

$$h(X|z) = \mu(X, z)s(X, z). \tag{14}$$

The first two columns in Table 2 present the results of such calculations for various life spans of fathers. Note that even if a father died at age 20, there is a 25 percent chance that he (and his son) are in the robust group and that even if a father died at age 80, there is a 40 percent chance that he (and his son) are in the frail group.

The probability distribution of the life span of a son is the weighted average of the distributions for the two groups, the weights corresponding to the probability that the son is in one group or the other. Hence life expectancy and survivorship values are easily

calculated. Table 2 presents the results. Whether a father dies at 20, 50, or even 65 makes little difference in his son's life expectancy or chances of early or late death. Furthermore, even if a father lives to 80, his son has only one chance in three of such longevity, compared with one chance in five for a son whose father died at 20.

Thus knowledge of a person's life span provides fairly weak information about that person's frailty—and knowledge of a person's frailty provides fairly weak information about that person's life span. The strength of this relationship can be quantified by calculating the correlation coefficient between life span and frailty. The value of this  $r$  is  $-0.39$  and the corresponding  $r^2$  is  $0.15$ .

In the frailty model, a father influences his son's longevity only via the intervening frailty variable. In this case, the  $r^2$  between fathers' and sons' life spans is the product of the  $r^2$  between the life span and frailty of fathers, the frailties of fathers and sons, and the frailty and life span of sons, that is,  $(0.15)(1.0)(0.15)$ , which yields the value of  $0.02$  previously reported in Table 1. [See Simon's (1957) or Blalock's (1961) work on causal modeling for discussion of this kind of relationship between correlation coefficients.]

Some further understanding of the weak correlation between the longevity of fathers and sons can be gained by considering the meaning of the terms in equation (3). The formula for  $E(XY)$  in equation (10) implies that the expression  $E(XY) - E(X)^2$  in the numerator of equation (3) can be interpreted as the variance in the life expectancies of people with different frailties, whereas, as noted earlier, the expression in the denominator of equation (3) represents the variance in life spans for the population as a whole. In other words, the correlation coefficient  $r$  equals the variance in mean life spans divided by the variance in life spans.

The well-known conditional variance formula,

$$\text{var}(A) = E \text{ var}(A|B) + \text{var } E(A|B), \quad (15)$$

implies that the variance in life spans can be decomposed into two additive components, the mean variance in life spans and the variance in mean life spans. The first component is the within-group variance and the second component the between-group variance. Thus the correlation coefficient  $r$  equals the between-group variance divided by the sum of the within- and between-group variances.

The standard deviations in life spans given in Table 2 for the frail and robust groups are 12.1 and 12.5 years; the average of the corresponding variances is more than 150. On the other hand, the variance in the two life expectancies of 66.5 and 77.3 is less than 30. Thus the within-group variance is fully five times greater than the between-group variance. This implies a value for  $r$  of about one in six; more exact calculations confirm that  $r$  is about  $0.15$  and that  $r^2$  is about  $0.02$ , as reported earlier.

These insights about why the  $r^2$  between fathers' and sons' life spans is so low raise the interesting issue of how best to report the relationship between two variables (Blalock, 1961; see Tversky and Kahneman, 1981; Vaupel, 1982, for discussion of statistical insinuation). It is apparent from Table 2 that a person's frailty affects his or her longevity: a robust person in this model has 11 more years of life expectancy than a frail person. Furthermore, viewed from the perspective of life expectancy or probability of early or late death, the life span of a father is of considerable importance to his son. Consider, for instance, a son whose father died at 65 compared with one whose father died at 90. The son of the longer-lived father faces half of the chance of death before age 65 and twice the chances of surviving past 80 and has a life expectancy nearly 7 years longer. For some purposes, this summary of the relationship between fathers' and sons' life spans may be more informative than reporting an  $r^2$  of  $0.02$ ; perhaps the most judicious course is to report both kinds of summary statistics.

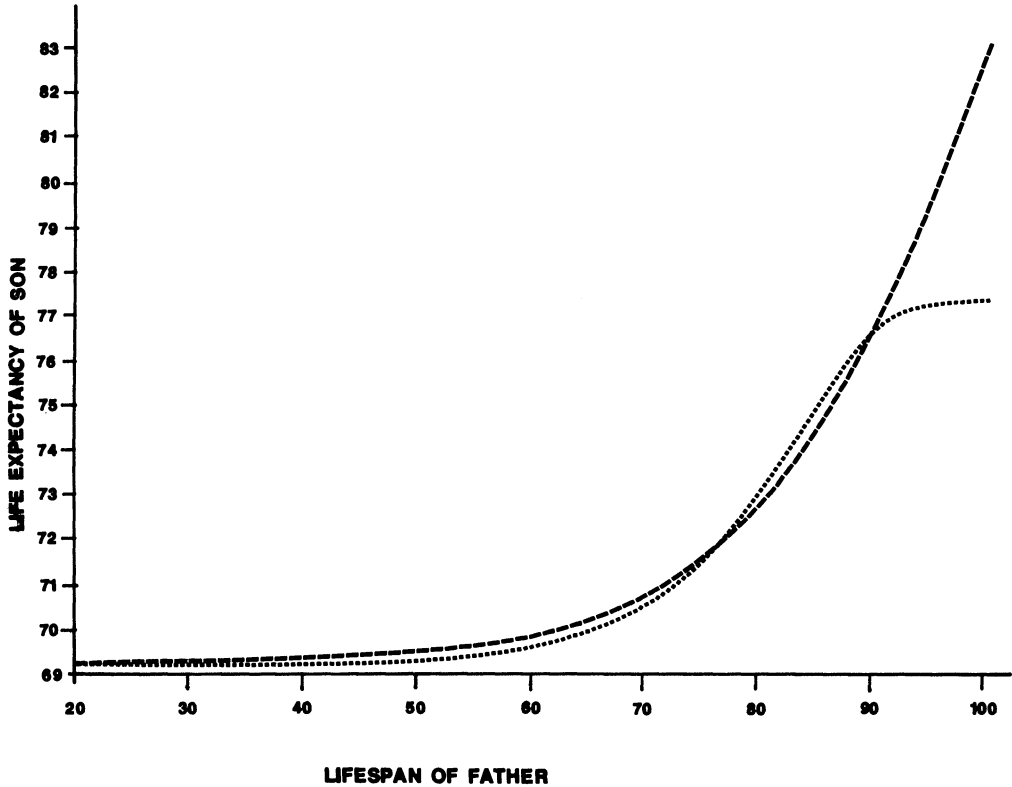


Figure 1. The Effect of Father's Life Span on Son's Life Expectancy (The dotted curve is based on a two-point distribution of frailty and the dashed curve on a gamma distribution.)

Figure 1 provides an informative graphical display of the impact of a father's age at death on his son's life expectancy. The dotted curve is based on the discrete model discussed previously; the values are consistent with those in Table 2. Note that the curve is an ogive rather than a line, with most of the change in a son's life expectancy occurring for lengths of the father's life span between 70 and 90. Each extra year the father lives between 20 and 60 adds only 3 or 4 days to his son's life expectancy.

The dashed curve in the figure is based on the model with gamma-distributed frailty, using the standard values of the various parameters. Bayes' theorem in this case is

$$g(z|X) = \frac{h(X|z)f(z)}{\int_0^\infty h(X|z)f(z) dz}, \tag{16}$$

where  $h(X|z)$  is given, as before, by equation (14). It is not difficult to show that  $g(z|X)$  is a gamma density function. The son's life expectancy conditional on his father's life span is given by

$$E(Y|X) = \int_0^\infty e_o(z)g(z|X) dz. \tag{17}$$



The curve in the figure is similar at younger ages to the curve based on the two-point distribution. Note, however, that each extra year a father lives between 90 and 100 adds nearly 8 months to his son's life expectancy.

### Further Research

Wolf (in press) proposed an empirical study of the relationship between the life spans of parents and children, using a model like the one discussed herein, in which frailty, rather than longevity per se, is transmitted between generations. The results presented here suggest that such a study may find that the small values of  $r^2$  observed between parents' and children's life spans are consistent with a high  $r^2$  between parents' and children's frailty. There may also be some insights for empirical studies, like the study of twins done by Hrubek et al. (1984), that control for observed risk factors in studying the transmission of longevity. Because the results presented in the previous section rest on the assumption that frailty is perfectly inherited, they are, in effect, based on a model that controls for all nonfamilial influences. The inference is that even if all such influences are controlled, the  $r^2$  between parents' and children's life spans—and the life spans of twins, siblings, or other relatives—may be low.

A crucial and hotly debated issue in demography and gerontology is whether life expectancy will level off at age 85 or continue to rise (e.g., see Fries, 1980, 1983, 1984; Manton, 1982; Myers and Manton, 1984; Vaupel and Gowan, 1986). One way to formulate the underlying question here is to ask whether there are two kinds of death, premature and predestined death, or whether all deaths are premature. Premature deaths are associated with risk factors and frailty levels; predestined deaths are determined by some genetically programmed maximum life span. If some deaths are predestined, then there is some direct genetic transmission of longevity between parents and children in addition to the indirect effects arising from transmission of frailty. Hence appropriately formulated models of inherited frailty and longevity, applied to data on the life spans of parents and children or other related individuals such as twins, may shed light on future trends in life expectancy and the growth in the population above age 85.

Frailty is equivalent to relative risk in a proportional-hazard model, mortality can be used as a metaphor for many kinds of transitions, and longevity is equivalent to duration or waiting time: Vaupel and Yashin (1985a,b) provided a variety of examples. As Samuel Preston (personal communication, Jan. 1987) pointed out, inheritance can be interpreted as an invariance in relative risk over a person's lifetime: a person's relative risk at some age is "inherited," perhaps imperfectly, from the person at a younger age. This insight opens up, as he suggested, a wide range of applications to repeatable events involving births, marriages, moves, unemployment spells, and so forth.

Sheps and Menken's (1973) pathbreaking study of models of conception and birth provides an illuminating example. In a section on "correlation between two conception times," fecundity is assumed to be constant for each woman but to vary across women: fecundity is thus equivalent to frailty  $z$  in the special case in which the hazard function  $\mu(x)$  equals 1 at all ages. Sheps and Menken derived a formula for the correlation coefficient between the number of months a susceptible woman waits before conceiving on two separate occasions. This formula can be interpreted as a special case of the formula discussed earlier, namely that  $r$  is the ratio of variance in mean life spans to the sum of the variance in mean life spans and the mean variance in life spans. If  $\gamma$  denotes the variance in mean time to conception and  $m$  denotes the mean time to conception for the population as a whole, Sheps and Menken's formula may be written as

$$r = \gamma/[2\gamma + m(m - 1)] \quad (18)$$

[because under Sheps and Menken's assumptions, the mean variance in "life spans" equals  $\gamma + m(m - 1)$ ].

As Sheps and Menken noted,  $r$  can be at most 0.5 (so  $r^2$  cannot exceed 0.25). Plausible assumptions yield much lower values of  $r$  and  $r^2$ . Suppose, for instance, that  $m$  is 6 and  $\gamma$  is 9. This implies that women one standard deviation above the mean wait, on average, three times longer than women a standard deviation below the mean (9 months vs. 3 months) and, consequently, are only a third as fecund. This substantial heterogeneity, however, leads to an  $r$  value of 0.19 and an  $r^2$  value of 0.035.

Sheps and Menken suggested that a correlation between two conception times may be evidence for heterogeneity of fecundity. If, however,  $r$  and  $r^2$  are unresponsive measures that fall close to zero even when women differ widely in their fecundity, these measures may be weak and perhaps misleading analytical lenses to use in empirical studies.

### Conclusion

Demographers, statisticians, economists, and others frequently study such waiting times as life spans, times to conception, durations of unemployment spells, and so on. In some cases the correlation between pairs of waiting times is of interest, and the hazard functions faced during the two intervals can be assumed to be the same for individuals experiencing two successive events or for pairs of linked individuals, such as fathers and sons or twins. Although the individuals (or linked pairs) may face the same hazard functions, the level of these functions may differ across individuals; for example, some individuals may be frailer or more fecund than others. If such heterogeneity exists, there will be a positive correlation between waiting times. As the results of this article demonstrate, however, even substantial heterogeneity in relative risk and perfect inheritance or invariance of relative risk may yield correlations that are close to zero.

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