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Foreword

One of the important directions of research in IIASA's Population Program is focused on understanding the effects of heterogeneity in human mortality. Various models of constant "frailty" developed in the authors' earlier papers capture well the selection effects. They, however, can not explain the debilitation phenomena which are often observed in the analysis of statistical mortality data.

The paper is devoted to the analysis of stochastic process models of mortality which can explain both selection and debilitation processes in the evolution of cohort mortality. The relative importance of each process is analyzed. The examples of various regimes of mortality evolution are demonstrated.

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James W. Vaupel, Anatoli I. Yashin**, Kenneth G. Manton****

INTRODUCTION

Wars, famines, epidemics, and depressions debilitate as well as decimate and the lingering morbidity consequences of a calamity may elevate mortality levels for years afterwards (Kermack et al. 1934a and b; Livi-Bacci 1962; Forsdahl 1977; Preston and van de Walle 1978; Okuba 1982; Horiuchi 1983; Marmot et al. 1984; Waaler 1984; Lawrence et al. 1985; Fogel 1986; Caselli et al. 1985 and 1986; Hearst et al. 1986). Healing and recuperation, fostered perhaps by social and public health programs, may restore the debilitated to normal health. Furthermore, death may prune the population of the most debilitated; this is the well-known process of selection in a heterogeneous population modeled by Beard (1963), Vaupel et al. (1979), and others reviewed in Vaupel and Yashin (1985). As a result, death rates among the recovered or selected survivors may decline to normal or even below-normal levels.

The dynamic interaction of debilitation, recuperation, and selection is complicated by aging. Disasters may have a stronger debilitating effect at some ages than others; we will refer to this phenomenon as vulnerability. The evidence in the various articles cited above suggests that the childhood and adolescent years are particularly vulnerable ones. Death rates tend to rise exponentially with age, so at older ages there may not be time for full recuperation before death strikes. Selection accelerates with age because the rate of selection is proportional to the level of mortality.

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In this paper we make use of a stochastic differential equation model, proposed by Woodbury and Manton (1977) and developed by Yashin et al. (1985), to disentangle and clarify the evolving interplay among debilitation, recuperation, selection, vulnerability, and aging. We motivate the model by beginning with Gompertz's differential equation model and then adding complications step by step. The completed model leads to a formula that decomposes the mortality rate at any age into two additive components which we call the baseline mortality rate and excess mortality rate. The relative change with age of the excess mortality rate can, in turn, be decomposed into four additive components which we call the forces of vulnerability, debilitation, recuperation, and selection. To gain some insights about when one of these forces predominates and about the interactions among the forces, we present the results of some computer simulations.

MODELS OF MORTALITY

Two disparate sets of mortality models have been developed, for different purposes and reasons. The first set of models, which might be called descriptive or graduation models, were developed to describe empirical mortality patterns without attention to underlying physiological or environmental processes. As discussed by Keyfitz (1982), such models are useful:

- "To smooth the data",
- "To make the result more precise",
- "To construct life tables",
- "To aid inferences from incomplete data",
- "To facilitate comparisons of mortality", and
- "To aid forecasting".

The multi-parameter curves of Thiele (1872) and Heligman and Pollard (1980), the graduation methods of Reed and Merrell (1939) and Greville (1943) and more recent spline approaches, the model life tables of Coale and Demeny (1966), Ledermann (1969), and Petrioli and Berti (1979), and the relational transformations of Brass (1975), Zaba (1979), and Ewbank et al. (1983) all fit into this category.

Mortality models of the second kind start with some biologically plausible process that is hypothesized to determine the age trajectory of mortality. Then the mortality curve is calculated from the process, either by derivation of a formula or by numerical approximation. It is sometimes forgotten that Gompertz (1825)

pioneered this approach. As discussed in the next section, Gompertz started with a differential equation that described the process of "indisposition" over age and then derived his familiar mortality curve from this differential equation. The subsequent mortality models of Makeham (1867), Armitage and Doll (1954), Strehler and Mildvan (1960), Sacher and Truco (1962), Beard (1963), Woodbury and Manton (1977), Vaupel et al. (1979), Economos (1982), and Moolgavkar (1986), all were based on biologically justified processes on the individual level (e.g., involving loss of vitality or accumulation of environmental insults) or on the population level (e.g., selection resulting from heterogeneity among individuals in their frailty).

In this set of process models the model proposed by Woodbury and Manton (1977) is useful for our purposes. It includes the key elements of debilitation, recuperation, and selection. In addition, it can be specified to include vulnerability (i.e., different mortality effects of disaster at different ages) and the lasting impact of temporary external conditions, like wars and famines, on physiological indisposition. In the following sections we elaborate the Gompertz model of human mortality to derive a univariate form of the general Woodbury-Manton model.

GOMPERTZ AND MAKEHAM

Following Gompertz (1825), suppose that "the average exhaustions of a man's power to avoid death were such that at the end of equal infinitely small intervals of time, he lost equal portions of his remaining power to oppose destruction", so

$$\frac{d\mu(x)}{dx} = \alpha\mu(x) \tag{1}$$

where $\mu(x)$ is the force of mortality at age x and α is some scaling parameter. Given an initial value

$$\mu(0) = \mu_0 \quad ,$$

the solution follows that

$$\mu(x) = \mu_0 e^{\alpha x} \quad . \tag{2}$$

A natural generalization of this approach is to let

$$\frac{d\mu(x)}{dx} = \alpha_0 + \alpha_1\mu(x) \quad . \tag{3}$$

The parameter α_0 represents the constant change in the force of mortality,

whereas a_1 is the proportional change. The solution is

$$\mu(x) = (\mu_0 + \frac{a_0}{a_1})e^{a_1 x} - \frac{a_0}{a_1} \quad . \tag{4}$$

If $a_0 < 0$, but

$$c_1 = \mu_0 + \frac{a_0}{a_1} > 0$$

and letting

$$c_2 = \frac{a_0}{a_1}$$

this is equivalent to Makeham's model:

$$\mu(x) = c_1 e^{a_1 x} + c_2 \quad . \tag{5}$$

These familiar models of Gompertz and Makeham are often adequate for the analysis of mortality data. However, to separate the age process of deterioration from the age process of mortality a more complicated model is needed.

EXHAUSTION AND AGING

To distinguish Gompertz's exhaustion process from the changes in mortality due to aging, let $Y(x)$ represent what Gompertz referred to as "exhaustion", "indisposition", and "inability to withstand destruction". If the force of mortality $\mu(x)$ is inversely proportional to $Y(x)$, as Gompertz assumes, then there is little point in distinguishing indisposition from the force of mortality. A more complicated relationship might, however, make sense.

Suppose, for instance, that there is some optimal state where the force of mortality is minimal; the force of mortality increases as an individual's condition deviates from this optimum, either in a positive or negative direction; the force of mortality is hardly affected if the deviation is small but a large deviation results in a disproportionately large increase in the force of mortality. Under these biologically plausible suppositions, it may be reasonable to let

$$\mu(x) = \mu_0(x) + \lambda(x)Y^2(x) \quad , \tag{6}$$

where $\mu_0(x)$ might be interpreted as the baseline force of mortality under optimal

conditions, where the indisposition $Y(x)$ measures the deviation in conditions from the optimal, and where the vulnerability $\lambda(x)$ determines the level of excess mortality resulting from this indisposition.

Two special cases are of interest. The value of $\lambda(x)$ might be constant. This simplifying assumption implies that the greatest *relative* increase in mortality levels produced by a given level of indisposition occurs at the ages where the absolute level of baseline mortality is lowest, an implication that may be plausible given the evidence on the disproportionate impact of disasters on children and adolescents.

Alternatively, $\lambda(x)$ might be equal to $\mu_0(x)$. Then

$$\mu(x) = \mu_0(x)(1 + Y^2(x)) \quad . \tag{7}$$

In this case, $Y^2(x)$ measures excess risk in the usual proportional hazards formulation.

If indisposition changes with age such that

$$\frac{dY(x)}{dx} = \alpha_0 + \alpha_1 Y(x) \quad , \quad Y(0) = Y_0 \quad , \tag{8}$$

then $Y(x)$ is given by a formula analogous to (4) and

$$\mu(x) = \mu_0(x) + \lambda(x)[(Y_0 + \frac{\alpha_0}{\alpha_1})e^{\alpha_1 x} - \frac{\alpha_0}{\alpha_1}]^2 \quad . \tag{9}$$

Depending on the signs of the parameters, this trajectory for the force of mortality can take on a variety of shapes, even when $\mu_0(x)$ and $\lambda(x)$ are constant and equal to μ_0 and λ. An interesting case involves negative Y_0 and α_1 with positive α_0, μ_0, and λ. Given (8), the positive value of α_0 might be interpreted as representing debilitation, whereas the negative value of α_1 might be interpreted as representing recuperation or homeostasis. These parameter values produce a trajectory that is reminiscent of human mortality curves, with declining mortality in infancy, rapidly rising mortality in middle age, and some leveling off at advanced ages.

This model, however, implicitly assumes that there is no heterogeneity in frailty among individuals and hence no selection. To capture the effects of selection, some additional features have to be added to the model.

FIXED FRAILTY: PURE SELECTION MODEL

The familiar heterogeneity model with constant, proportional hazards,

$$\mu(x,z) = z\,\mu(x) \quad , \tag{10}$$

where $\mu(x,z)$ is the force of mortality of individuals age x with frailty z and $\mu(x)$ is the force of mortality for standard individuals with frailty 1, is readily extended to:

$$\mu(x,z) = \mu_0(x) + z\,\mu(x) \quad , \tag{11}$$

where frailty z could be identified with the square of indisposition Y and where $\mu_0(x)$ is some baseline force of mortality. Then $\bar{\mu}(x)$, the observed force of mortality among surviving individuals, which is given by

$$\bar{\mu}(x) = E(\mu(x,z) \mid X > x) \tag{12}$$

where X denotes age at death, can be expressed as

$$\bar{\mu}(x) = \mu_0(x) + \mu(x)\bar{z}(x) \tag{13}$$

where $\bar{z}(x)$ is the average frailty of surviving individuals, defined by

$$\bar{z}(x) = E(z \mid X > x) \quad . \tag{14}$$

As shown by Vaupel and Yashin (1984a), the process of selection resulting from the higher death rates of frailer individuals implies that

$$\frac{d\bar{z}(x)}{dx} < 0 \quad . \tag{15}$$

Thus, $\bar{\mu}(x)$ increasingly deviates downward from $\mu(x)$ with age.

This model of fixed frailty incorporates both aging and selection but it fails to explicitly capture debilitation and recuperation. Since fixed frailty is assumed in many empirical studies (including Manton et al. 1981 and 1986, Heckman and Singer 1984, and Trussel and Richards 1985), it would seem useful to investigate what effect debilitation and recuperation might have and when these effects could be ignored. Furthermore, interest in catastrophes requires attention to debilitation and recovery. Thus it is appropriate to combine models such that frailty both changes over age (and time) and varies across individuals.

WHEN EVERYONE'S FRAILTY CHANGES

Consider, then, the model

$$\mu(x,Y) = \mu_0(x) + \lambda(x)Y^2(x) \quad , \quad Y(0) = Y_0 \quad , \tag{16}$$

but now assume that Y_0 is a random variable that differs from individual to individual. Suppose that the change over age in indisposition is described by

$$\frac{dY(x)}{dx} = \alpha_0(x) + \alpha_1(x)Y(x) \quad , \tag{17}$$

noting that now the parameters α_0 and α_1 may vary with age (and time). To develop appropriate methods of analysis for this model, it is useful to step back and consider the arbitrary, perhaps random, process $Y(x)$ and not just the particular process in (17). Then

$$\bar{\mu}(x) = \mu_0(x) + \lambda(x)(m^2(x) + \gamma(x)) \quad , \tag{18}$$

where, as before,

$$\bar{\mu}(x) = E(\mu(x,Y) | X > x) \quad ,$$

where X denotes age of death and where $m(x)$ and $\gamma(x)$ are the conditional mean and variance of $Y(x)$ among surviving individuals; we use the notation $\gamma(x)$ rather than $\sigma^2(x)$ to emphasize that $\gamma(x)$ is not a usual unconditional variance but a conditional variance. To derive (18), note that

$$E(Y^2(x) | X > x) = E((Y(x) - m(x) + m(x))^2 | X > x)$$

$$= E((Y(x) - m(x))^2 | X > x) + Em^2(x) + E(2m(x)(Y(x) - m(x)) | X > x) \quad .$$

The first term in this expression is, by definition, $\gamma(x)$, the second term is just $m^2(x)$, and the third term has a value of zero.

The problem now becomes a problem of determining $m(x)$ and $\gamma(x)$. For the process in $Y(x)$ described by (17) it follows, as a special case of the results in Yashin et al. (1985), that if Y_0 is normally distributed with mean m_0 and variance γ_0, then

$$\frac{dm(x)}{dx} = \alpha_0(x) + \alpha_1(x)m(x) - 2\lambda(x)m(x)\gamma(x) \tag{19}$$

and

$$\frac{d\gamma(x)}{dx} = 2\alpha_1(x)\gamma(x) - 2\lambda(x)\gamma(x)^2 \quad . \tag{20}$$

The evaluation of this pair of differential equations can be approximated by computer numerical methods. Interestingly, the conditional distribution of $Y(x)$ among the surviving at age x is normal (with mean $m(x)$ and variance $\gamma(x)$).

The model developed above is based on the assumption that individuals' initial indispositions change deterministically over time. This may be an appropriate assumption in studies focusing on evolving external conditions that affect all the individuals in a cohort more or less the same way. However, the model fails to capture the impact of turbulent disturbances that affect different individuals differently.

PURE STOCHASTIC INDISPOSITION

In many situations it may be reasonable to allow the indisposition of one individual to change with age *relative* to the indisposition of another individual. As individuals get sick, get well, stop smoking, start drinking, etc., their relative indispositions may change, and famines, wars, epidemics, and depressions may harm some individuals more than others.

As a simple case of changing relative indispositions, consider the process

$$Y(t) = \int_0^t b(x)dW(x) \quad , \tag{21}$$

where W is a Wiener (or Brownian motion) process and where b is a parameter that may change over age (and time). The Wiener process is a continuous time, continuous path stochastic process with independent, normally distributed increments such that

$$E(W(x_2) - W(x_1)) = 0 \tag{22}$$

and

$$E(W(x_2) - W(x_1))^2 = x_2 - x_1 \quad . \tag{23}$$

Thus, (21) implies that if an individual has some indisposition $Y(x_1)$ at age x_1, then the individual's indisposition at age x_2 will be normally distributed with a mean of $Y(x_1)$ and a variance of $\int_{x_1}^{x_2} b^2(x)dx$.

Given this formulation, it can be shown, as a special case of the results in Yashin et al. (1985), that if, as before, an individual's chance of death at age x and indisposition Y is given by

$$\mu(x,Y) = \mu_0(x) + \lambda(x)Y^2(x) \quad , \tag{24}$$

then the conditional distribution of vulnerability $Y(x)$ among survivors at age x is Normal with mean $m(x)$ and variance $\gamma(x)$ described by

$$\frac{dm(x)}{dx} = -2\lambda(x)m(x)\gamma(x) \tag{25}$$

and

$$\frac{d\gamma(x)}{dx} = b^2(x) - 2\lambda(x)\gamma^2(x) \quad , \tag{26}$$

where, as before,

$$\bar{\mu}(x) = \mu_0(x) + \lambda(x)(m^2(x) + \gamma(x)) \quad . \tag{27}$$

PUTTING IT ALL TOGETHER

A model that includes the various elements discussed so far of changing mortality and vulnerability with age, heterogeneity among individuals in their innate indisposition, and both deterministically and stochastically changing individual indisposition would be

$$\mu(x,Y) = \mu_0(x) + \lambda(x)Y^2(x) \quad , \tag{28}$$

and

$$dY(x) = [a_0(x) + (a_1(x) - a_1'(x))Y(x)]dx + b(x)dW(x) \tag{29}$$

with $Y(0)$ normally distributed with mean m_0 and variance γ_0 and $W(0)$ equal to zero. Note that two parameters $a_1(x)$ and $a_1'(x)$ are used in the formulation. The idea is that both these parameters are non-negative and that $a_1(x)$ (along with $a_0(x)$) represents the effects of debilitation whereas $a_1'(x)$ represents the homeostatic healing and recuperation. This is a simple expedient, but effective, at least for exposition and for gaining insights into the effects of debilitation vs. recuperation.

It follows from Vaupel et al. (1979) and Yashin (1986) that even in the case of changing individual indisposition, the observed population trajectory of the force of mortality is given by

$$\bar{\mu}(x) = E(\mu(x,Y) \mid X > x) \tag{30}$$

and hence by

$$\bar{\mu}(x) = \mu_0(x) + \lambda(x)\bar{z}(x) \tag{31}$$

where $\bar{z}(x)$ is the average frailty (i.e., squared indisposition) at age x among those surviving. As noted earlier, the result

$$\bar{z}(x) = m^2(x) + \gamma(x) \tag{32}$$

holds for *any* process $Y(x)$ such that

$$z(x) = Y^2(x) \quad .$$

When $Y(x)$ is described by (29) and when $Y(0)$ is normally distributed with mean m_0 and variance γ_0, then, as shown by Yashin et al. (1985),

$$\frac{dm(x)}{dx} = a_0(x) + (a_1(x) - a_1'(x))m(x) - 2\lambda(x)m(x)\gamma(x) \tag{33}$$

and

$$\frac{d\gamma(x)}{dx} = 2(a_1(x) - a_1'(x))\gamma(x) + b^2(x) - 2\lambda(x)\gamma^2(x) \quad . \tag{34}$$

The previous equations for $\frac{dm(x)}{dx}$ and $\frac{d\gamma(x)}{dx}$ given in (19) and (20) and (25) and (26) can be seen to be special cases of (33) and (34).

Equations (33) and (34) can be solved in various special cases, but in general the values of $m(x)$ and $\gamma(x)$ have to be calculated using numerical approximation methods. This is readily done with the help of a personal computer using different equations to calculate the values of $m(x + \Delta)$ and $\gamma(x + \Delta)$, for some sufficiently small increment Δ, given the values of $m(x)$ and $\gamma(x)$.

A DECOMPOSITION OF THE FORCE OF MORTALITY

The model leads to a two-stage decomposition of the force of mortality $\bar{\mu}(x)$. First, the baseline mortality rate $\mu_0(x)$ can be separated from the excess mortality rate given by $\lambda(x)\bar{z}(x)$. Second, formulas (32), (33), and (34) imply that the relative rate of change in this excess mortality rate can be decomposed into four components:

$$\frac{\frac{d\lambda(x)\bar{z}(x)}{dx}}{\lambda(x)\bar{z}(x)} = \rho_v(x) + \rho_d(x) + \rho_r(x) + \rho_s(x) \quad , \tag{35}$$

where

$$\rho_v(x) = \frac{\frac{d\lambda(x)}{dx}}{\lambda(x)} \quad , \tag{36}$$

$$\rho_d(x) = 2a_1(x) + \frac{2a_0(x)m(x) + b^2(x)}{m^2(x) + \gamma(x)} \quad , \tag{37}$$

$$\rho_r(x) = 2a_1'(x) \quad , \tag{38}$$

and

$$\rho_s(x) = 2\lambda(x)\gamma(x)\left\{1 + \frac{m^2(x)}{m^2(x) + \gamma(x)}\right\} \quad . \tag{39}$$

In this decomposition $\rho_v(x)$ captures the impact of change over age (or time) in vulnerability, $\rho_d(x)$ captures the impact of debilitation, $\rho_r(x)$ captures the impact of recuperation, and $\rho_s(x)$ captures the impact of selection. Note that if a_0, a_1, and b equal zero, there is no debilitation and $\rho_d(x)$ equals zero. On the other hand, if $\lambda(x)$ is zero or if the population is homogeneous (i.e., $\gamma(x)$ equals zero) then there is no selection and $\rho_s(x)$ is zero. We will refer to the ρ's as the forces of vulnerability, debilitation, recuperation, and selection.

Because both $\rho_d(x)$ and $\rho_s(x)$ depend on $m(x)$ and $\gamma(x)$, the two processes interact. There can be selection with no debilitation—if μ_0, a_1, and b are zero and γ_0 is positive. This is the familiar case of a heterogeneous population with fixed frailty. There can also be debilitation with no selection—if a_0 or a_1 are positive and both γ_0 and b are zero. This is the case when the population is homogeneous in frailty at all ages. But if frailty is changing in a heterogeneous population, then debilitation at any age will affect selection, i.e., $\rho_s(x)$, at later ages and selection at any age will affect debilitation, i.e., $\rho_d(x)$, at later ages.

Given the formulation of the model, the force of recuperation affects $m(x)$ and $\gamma(x)$, as described in (33) and (34), and thus affects the force of debilitation and selection. On the other hand, since the force of recuperation, as given by (38), depends only on $a_1'(x)$, this force is not directly affected by the force of debilitation or selection. At some level not explicitly included in the model there could, however, be some linkage. For instance, a disaster that causes debilitation might invoke social aid that increases the value of $a_1'(x)$ and hence fosters recuperation.

VARIETIES OF DISASTROUS EXPERIENCE

To gain some insights about the model, we wrote a simulation program that runs on an IBM PC. Table 1 summarizes the parameters of 10 mortality regimes that govern the life chances of a hypothetical cohort as it ages over time. In every regime, $\mu_0(x)$, the baseline force of mortality, is assumed to be the same. The various parameters are given in the table and in the notes to the table. The regimes were selected to illustrate ideas rather than to replicate empirical observations.

Table 1. Alternative mortality regimes.

	Parameters						
				during disaster			
Regime	γ_0	b	a_1'	λ_0	a_0	a_1	b
i	0	0	0	100	0	0	0
ii	1	0	0	100	0	0	0
iii	0	0	0	1	.1	0	0
iv	1	0	0	1	.1	0	0
v	1	0	0	1	0	.1	0
vi	0	0	0	1	0	0	1
vii	0	0	.05	1	0	0	1
viii	0	0	0/.05	1	0	0	1
ix	0	0	0	1	.05	.05	.5
x	0	.5	0	1	.1	0	.5

Notes: In all instances m_0 is one, $\mu_0(x)$ is given by $.0001e^{.1x} + .01e^{-x}$, and $\mu(x,Y)$ is given by $\mu_0(x) + \lambda(x)Y^2(x)/\bar{z}_0$, where $\lambda(x)$ equals $5\lambda_0\mu_0(x) + .002$ and where $\bar{z}_0$ is a scaling factor equal to $m_0^2 + \gamma_0$. This scaling insures that $\bar{\mu}(0)$ is the same in all the regimes. In regime viii, a_1' is zero before age 20 and .05 afterwards. In all regimes, a_0 and a_1 are zero and λ_0 is one, except during a disaster. Disasters last from age 10 through age 19.

Figure 1 presents eight pairs of mortality trajectories, labelled (i) to (viii), that correspond to the first eight mortality regimes listed in Table 1. In each case, the solid curve gives the trajectory when there is no disaster and the dotted curve gives the trajectory when there is a disaster. As noted in Table 1, a disaster starts at age 10 and lasts through age 19.

Figure 1(i) illustrates the consequences of a disaster in a mortality regime where there is no heterogeneity, no debilitation, and no recuperation. The disaster comes and goes, with severe immediate effects but no aftermath.

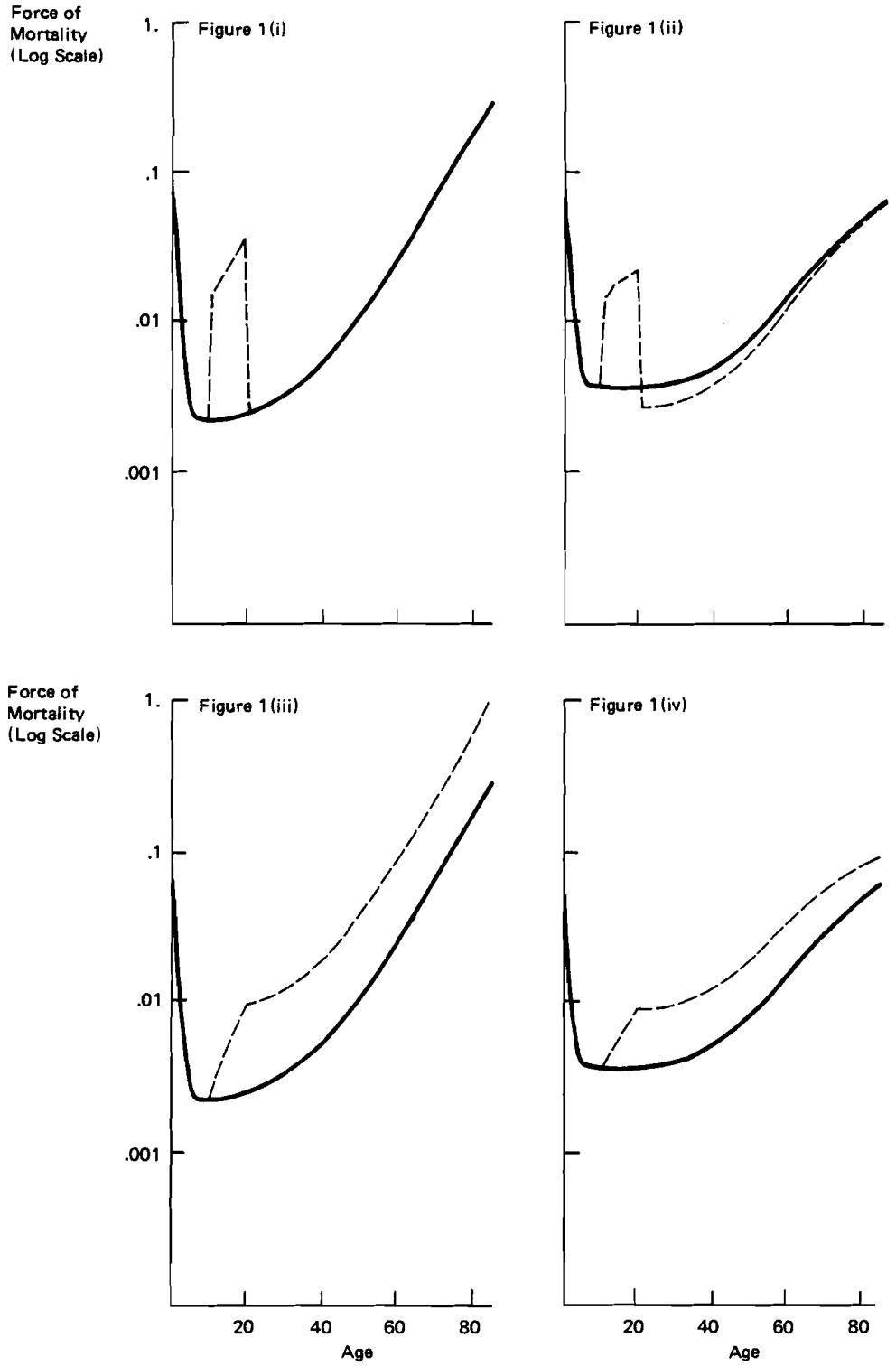
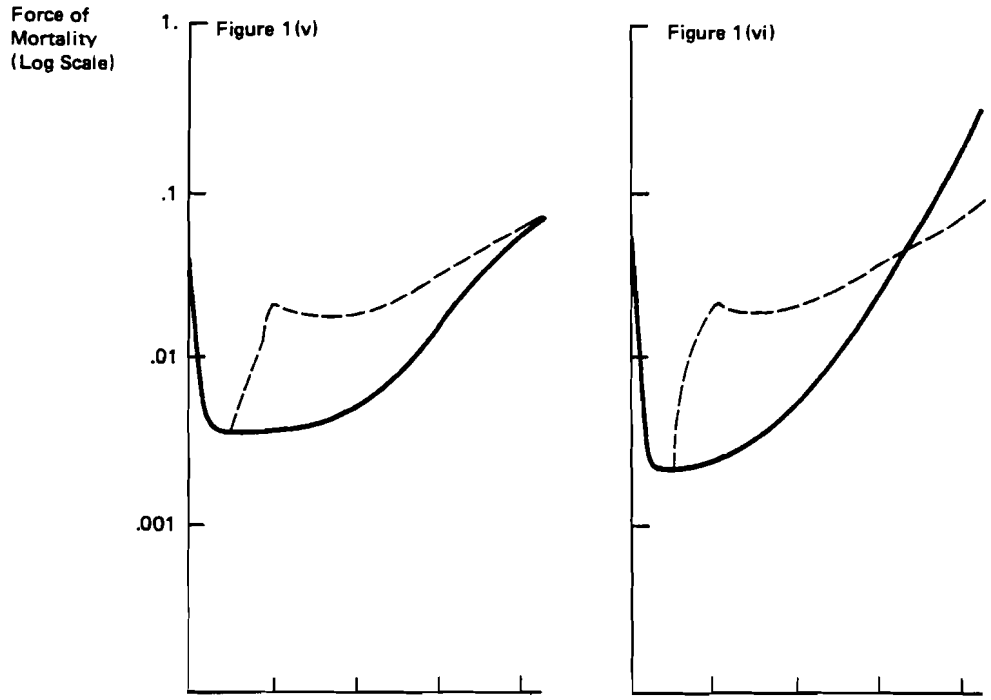
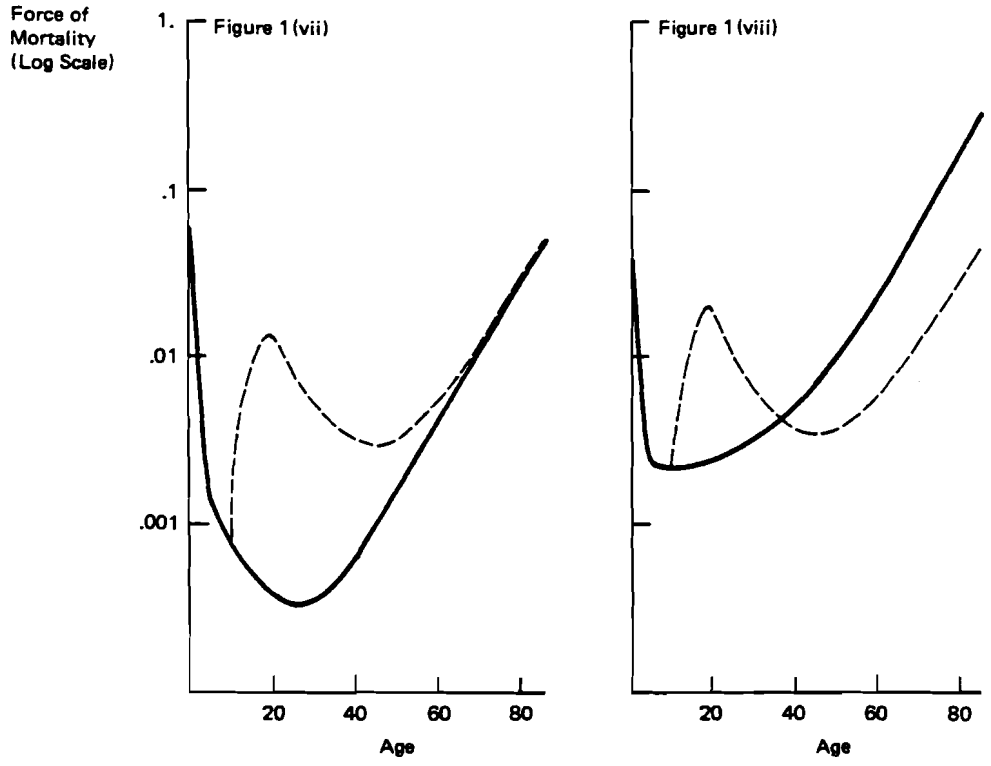


Figure 1. Varieties of disastrous experience. (See text for explanation.)


Force of Mortality (Log Scale)
1.
.1
.01
.001
Figure 1(v)
Figure 1(vi)


Force of Mortality (Log Scale)
1.
.1
.01
.001
Figure 1(vii)
Figure 1(viii)
20
40
60
80
Age
20
40
60
80
Age

If there is heterogeneity in frailty in the population at birth, as is the case in Figure 1(ii), then both with and without a disaster, the effects of selection reduce the level of observed mortality. A disaster raises mortality levels and hence accelerates the death of frailer individuals. As a result, the increase in mortality rates during the disaster is somewhat moderated. In addition, the level of mortality after the disaster is lowered. Because selection now operates more rapidly in the advantaged cohort, the two mortality trajectories gradually converge.

If the disaster raises everyone's indisposition by the same amount and if the population is homogeneous, the case in Figure 1(iii), then the result of the disaster is a permanent increase in the level of mortality. If, however, the population is heterogeneous, as in Figure 1(iv), the increased force of selection after the disaster results in some convergence in the mortality trajectories.

Figure 1(v) also describes mortality trajectories for a heterogeneous cohort, but now the disaster does not raise each individual's indisposition by the same absolute amount but by the same proportion. The disaster, in increasing the variance in indisposition among individuals as well as the level of indisposition, substantially accelerates the selection process. The mortality trajectories, as a consequence, show marked convergence.

The cohorts whose mortality is described in Figure 1(vi) are both initially homogeneous. The cohort affected by the disaster becomes heterogeneous as a result of the disaster: the disaster can be thought of a time of turbulence producing random changes in indisposition. The selection caused by the acquired heterogeneity, coupled with the existence of fortunate individuals whose indisposition is reduced during the disaster, so markedly affects the subsequent mortality trajectory of the stricken cohort that after age 70 or so this cohort has a more favorable mortality experience.

As illustrated by these six figures, disasters can be captured either by changes in the vulnerability function λ or by changes in the parameters of the indisposition process, α_0, α_1, or b. The lingering mortality consequences of a disaster produced in any of these ways can be moderated by homeostasis or recuperation, as represented by the parameter α_1'. Figures 1(vii) and (viii) illustrate this in the case of the kind of disaster portrayed in Figure 1(vi), a time of turbulence that results in acquired heterogeneity.

If homeostasis operates from birth on, then, as shown in Figure 1(vii), the mortality curve for the cohort not suffering the disaster is substantially lower than the curves shown in previous figures. The effect of the homeostasis parameter is to gradually reduce everyone's indisposition from its initial level of one toward the optimal level of zero. This might reflect health progress made over the course of the cohort's life. A disaster that creates substantial heterogeneity increases the level of mortality, but as a result of homeostasis (or recuperation) there is rapid convergence of the new mortality trajectory toward the trajectory of the fortunate cohort.

In creating Figure 1(viii) it was assumed that recuperation follows a disaster, being produced both by natural physiological recovery and by various social interventions. Hence, the cohort not afflicted by the turbulent times of the disaster does not benefit from recuperation. Its trajectory is the same as the trajectory for the advantaged cohort in Figure 1(vi). The afflicted cohort benefits so substantially from the force of recuperation that its mortality trajectory falls below the other cohort's trajectory less than twenty years after the disaster. The effects of selection, recuperation, and random lowering of indisposition for some fortunate individuals during the disaster combine to yield a very favorable mortality trajectory from age 40 on.

DISENTANGLING DEBILITATION AND SELECTION

As noted earlier, demographers for many years have been interested in the effects of debilitation vs. selection. Confusion here is easy given the intricate interaction of debilitation and selection. To gain some insights into the nature of this interaction, it is useful to carefully dissect the immediate and lingering effects of a disaster. Figures 2(i) through (vii) provide an illustration for mortality regime ix in Table 1. In this mortality regime, there is no homeostasis and the vulnerability parameter λ_0 is constant and equal to one. These simplifications facilitate comprehension of the effects of debilitation and selection resulting from a combination of absolute, proportional, and random changes in indisposition in a heterogeneous population.

Figure 2(i) displays the mortality trajectories with and without the disaster. The effects of the debilitation caused during the disaster and the selection following the disaster are substantial. Note that the mortality trajectories converge, but that there is no crossover. Differential selection necessarily produces some

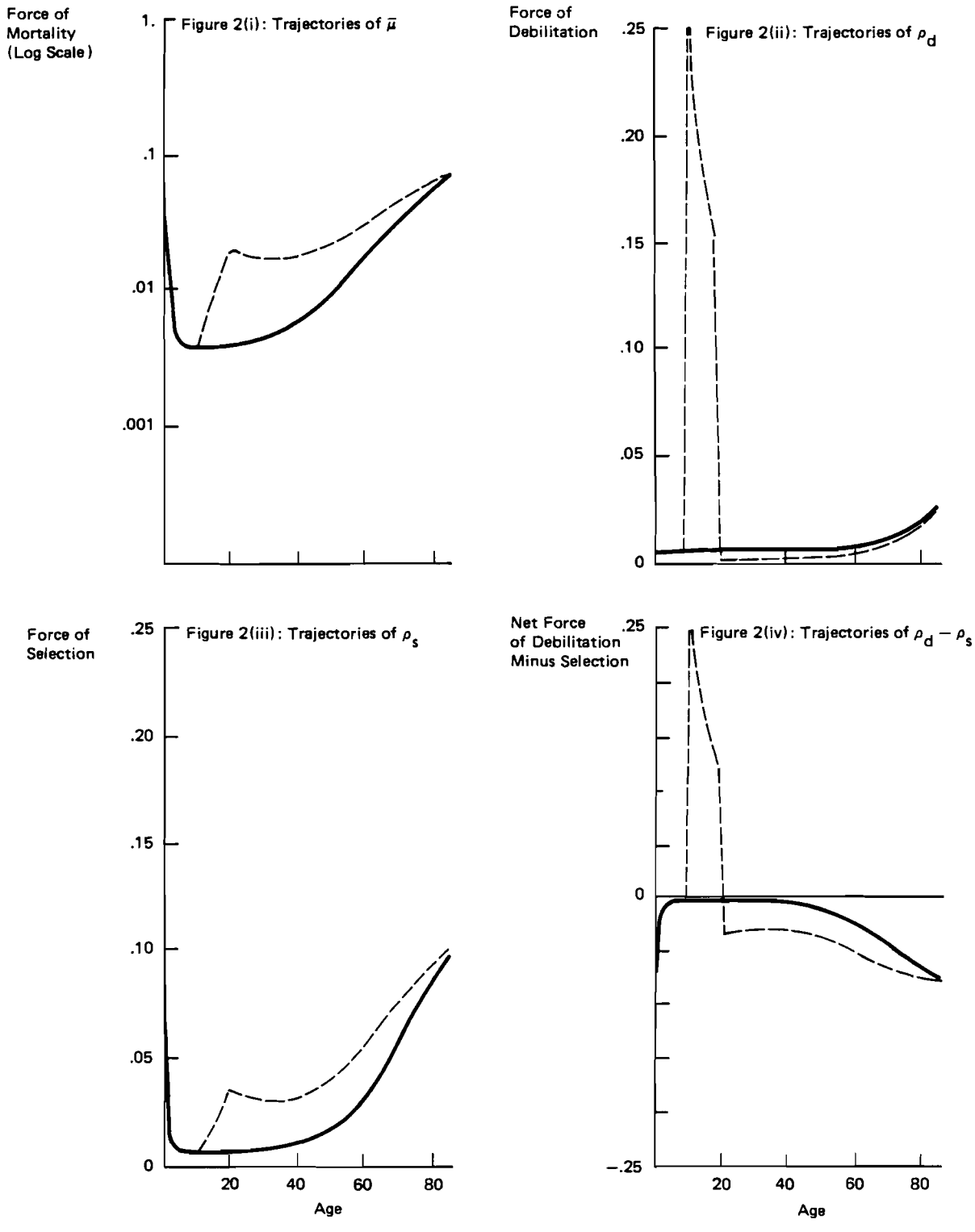


Figure 2. Aspects of disaster.
Figures 2(i)-(vii) display the trajectories of $\bar{\mu}$, ρ_d, ρ_s, $\rho_d - \rho_s$, m, γ, and $m^2+\gamma$, respectively.

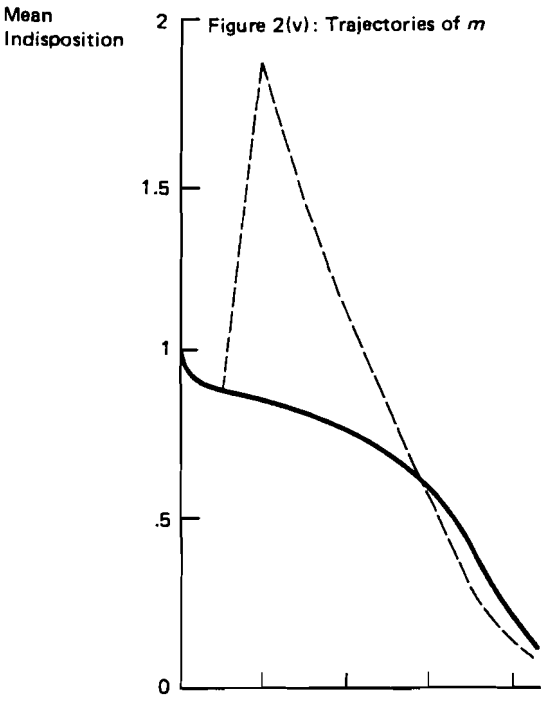


Figure 2(v): Trajectories of m

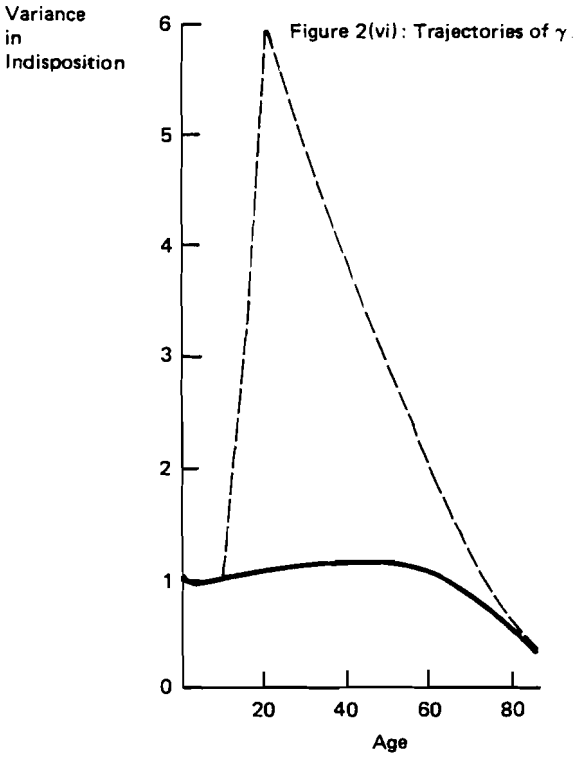


Figure 2(vi): Trajectories of γ

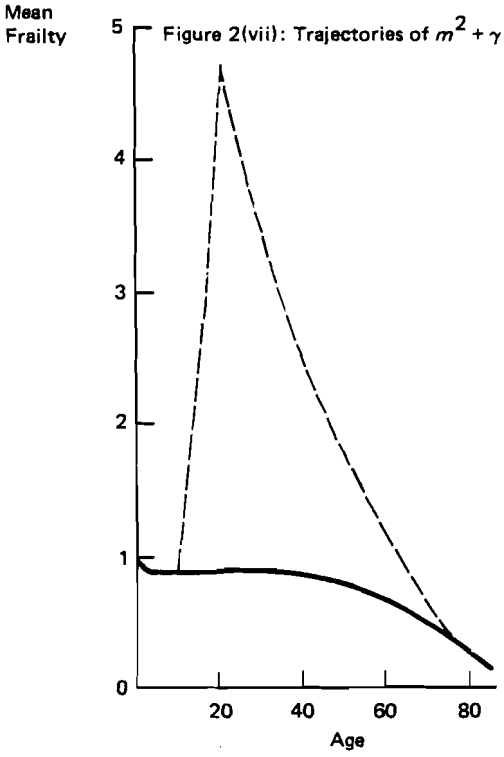


Figure 2(vii): Trajectories of $m^2 + \gamma$

convergence, but it does not have to result in a crossover (Vaupel and Yashin 1985).

Figure 2(ii) plots the force of debilitation, as defined by formula (37) over age. For both cohorts, there is some debilitation before the disaster, resulting from the random changes in indisposition produced by the positive value of the parameter b. The strongest period of debilitation is confined to the decade of the disaster, but there is some debilitation thereafter produced by random changes in indisposition. Since the value of the parameter b is constant after the disaster, formula (37) implies that the increase in the force of debilitation is attributable to the declining value of mean frailty (i.e., of $m^2+\gamma$). Similarly, the low value of the force of debilitation for the afflicted cohort, especially in the years immediately following the disaster, is attributable to the high value of mean frailty during this period.

As shown in Figure 2(iii), for both cohorts there is some selection before the disaster, largely in infancy when the mortality rate is high: this selection results from the initial heterogeneity of the population, as implied by the positive value of the parameter γ_0. Selection accelerates during the disaster as population heterogeneity increases, and selection continues to operate after the disaster, with increasing force as the level of mortality increases with age. The high mortality rate suffered by the afflicted cohort gradually reduces the difference in mean frailty between the two cohorts: this differential selection produces the convergence with age in the forces of selection for the two cohorts.

Figure 2(iv) displays the difference between the force of debilitation and the force of selection, for the two cohorts. When this difference is positive, it can be said that debilitation predominates; when it is negative, selection predominates. For the cohort that does not suffer the disaster, selection predominates at all ages, although the forces of selection and debilitation are in rough balance (and are both small) from childhood through age 40 or so. For the cohort that suffers the disaster, debilitation predominates only during the decade of the disaster, and, as a result of this debilitation, the force of selection is substantial at all ages after the disaster. As this example makes transparent, selection should be thought of not as an alternative to debilitation, but as a consequence of any debilitation that increases population heterogeneity.

Figures 2(v), (vi), and (vii) show the change with age in m, the mean level of indisposition, γ, the variance in indisposition, and $\bar{z}$, the mean level of frailty (i.e., indisposition squared). Mean indisposition falls somewhat during the first decade

as a result of selection in the initially heterogeneous population. For the afflicted cohort, the mean almost doubles during the disaster but, as a result of rapid selection, the mean falls below one again around age 40 and falls below the mean for the advantaged cohort around age 60. Variance in indisposition shows a similar pattern, reaching a peak of 8 for the afflicted cohort, although without a crossover. As a result, the trajectory of mean frailty, which equals $m^2+\gamma$, shows an analogous pattern for the advantage cohort of steady decline and for the afflicted cohort of a sharp rise to a peak, this time close to 5, and then a somewhat less rapid fall as the frail victims of the disaster die.

Figures 3(i) through (vii) provide a second set of illustrations of the interactions of various factors in the mortality model. As discussed above, the importance of what might be called a cohort's memory of past disasters, as reflected in current and future mortality rates, is reduced by selection and by homeostasis or recuperation. Stochastic change in individuals' indisposition also leads to forgetfulness, because the more turbulent these changes are, the less correlation there will be between an individual's indisposition at two different ages. To explore this phenomenon, mortality regime x in Table 1 was used to derive the diagrams in Figure 3. In this regime, b, the parameter of stochasticity, is set at a value of .5 at all ages. The disaster is modeled by setting a_0, the drift parameter, equal to .1; this kind of disaster was previously analyzed in Figures 1(iii) and (iv).

Note in Figure 3(i) that in contrast to the trajectories in Figure 1(iii) and (iv) the mortality trajectory of the afflicted cohort is only modestly higher, and only for a relatively short period, than the mortality trajectory for the advantaged cohort. Also note that the two mortality trajectories are qualitatively similar to previous mortality trajectories: the substantial turbulence in this mortality regime is not apparent in the trajectories of population force of mortality.

Figures 3(ii) and (iii) display the trajectories of the forces of debilitation and selection, respectively. Debilitation increases as a result of the disaster and then falls below that for the advantaged cohort, for the same reasons discussed earlier. What is new here is the very rapid increase in the force of debilitation with age, although the underlying cause of this, the decline in the value of mean frailty, is the same as discussed before. The force of selection also rises very rapidly with age, and for both the force of debilitation and selection the afflicted and advantaged cohort's trajectories quickly converge.

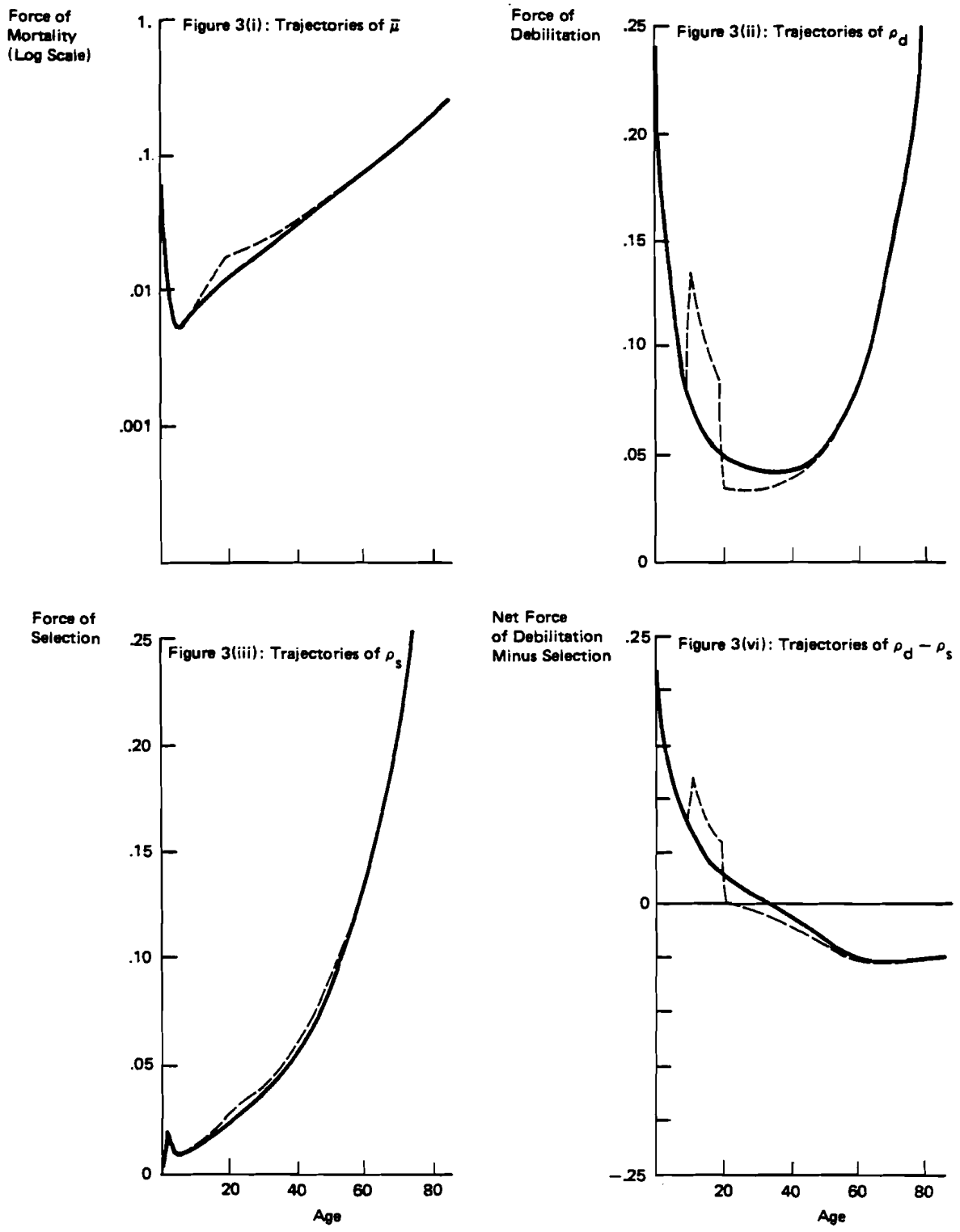


Figure 3. Aspects of mortality in a turbulent regime.
Figures 3(i)-(vii) display the trajectories of $\bar{\mu}$, ρ_d, ρ_s, $\rho_d - \rho_s$, m, γ, and $m^2+\gamma$, respectively.

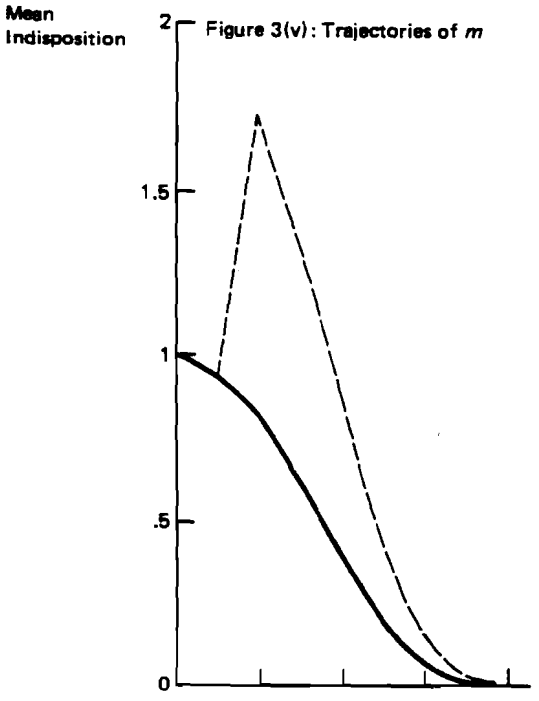


Figure 3(v): Trajectories of m

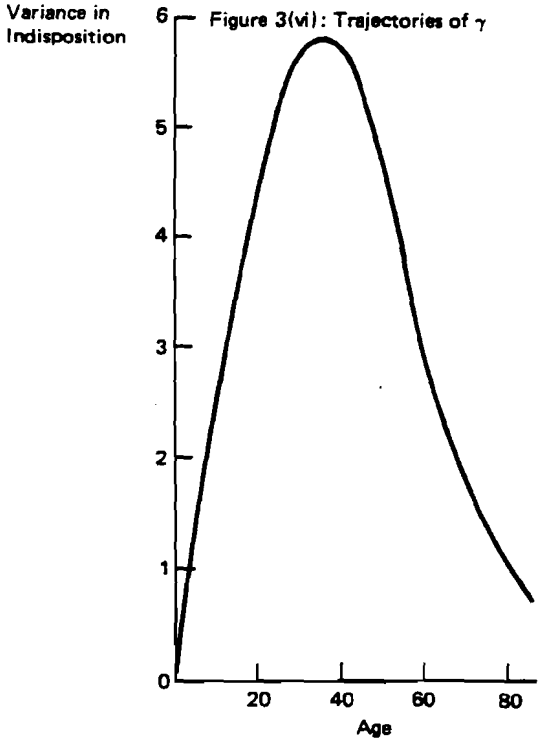


Figure 3(vi): Trajectories of γ

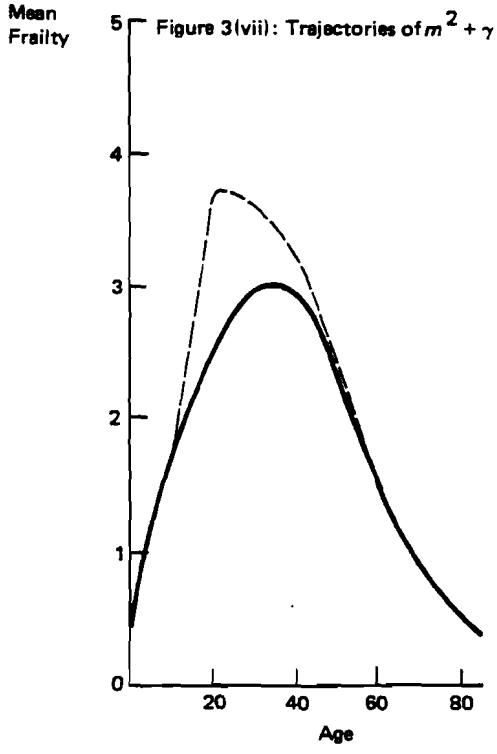


Figure 3(vii): Trajectories of $m^2 + \gamma$

As shown in Figure 3(iv), the force of selection manages, after age 20 or 30, to keep ahead of the force of debilitation, so that the rapid increase in both forces results in a balance that somewhat favors selection. The rapid increase in the force of selection can be interpreted as resulting from the rapid increase in the force of debilitation, but the force of selection does not lag behind the force of debilitation, but stays ahead of it.

The strength of the force of selection drives the mean value of indisposition to zero, as shown in Figure 3(v). The variance in indisposition rises to a peak but then falls off as the force of selection exceeds the force of debilitation, as shown in Figure 3(vi). There is only a single curve in this figure because the disaster does not affect the variance in indisposition but only the level of indisposition. The combination of these two trajectories, in the form of $m^2+\gamma$, produces the trajectories for mean frailty displayed in the final figure, Figure 3(vii). In this turbulent mortality regime, memory of the disaster is no longer apparent after age 50.

EXTENSIONS AND OTHER APPLICATIONS

The model of mortality presented and illustrated above can be extended in various ways and applied to other kinds of population problems. This section adumbrates a few possibilities.

Our concern here has been conceptual advance and insight, rather than statistical estimation and inference. Elsewhere, however, we discuss how Woodbury-Manton stochastic models can be used in empirical studies (see, e.g., Woodbury et al. 1979, Manton and Stallard 1984, and Yashin et al. 1985). In many of these applications it is appropriate to distinguish several different, interacting stochastic processes related to various physiological, behavioral or environmental factors that may be continuously observed, partially observed, or unobserved. Fortunately, the univariate-process model described here is readily generalized to a multivariate-process model with various kinds of data (Woodbury and Manton 1977, Yashin et al. 1985).

Our focus has been on disasters, but the model could also be used to study other aspects of mortality, including the typical shape of mortality trajectories. A remarkable feature of most human mortality curves is the bump in mortality rates, usually centered around age 20 or 25. In developed countries today this bump is greater for males than for females and can be largely explained by violent deaths

resulting from accidents, homicide, and suicide. But a bump also appears in mortality trajectories of cohorts born more than a century ago and in mortality trajectories for less developed countries (see, e.g., Preston et al. 1972). The model presented here can be specified to produce such as bump. By choosing less extreme parameter values, the very prominent bumps in some of the figures discussed above can be reduced to realistic size. It seems plausible that at least some of the excess mortality bump is caused by a kind of debilitation that occurs during the adolescent and early adult years as a result of individuals being confronted with environments that they are not fully prepared to deal with. For various physiological, behavioral, and environmental reasons, as an individual reaches maturity a gap may develop between external demands and internal capabilities and inclinations, a gap that for most individuals is reduced with age, partially as a result of learning and the acquisition of wisdom and caution. It may prove informative to apply the kind of stochastic model presented here to analyze the bump in mortality in various countries and times.

In addition, the model could be used to analyze the effects of lifetime deprivation and the effects of progress over time in reducing mortality levels. For a wide variety of different specifications of the model, the mortality trajectory of a disadvantaged cohort will converge toward the trajectory of an advantaged cohort. Similarly, equal rates of progress at all ages in reducing the underlying force of mortality on the individual level will result in declining rates of progress with age in reducing the observed, population force of mortality. Thus, there will appear to be convergence between the mortality trajectory of a cohort not benefitting from mortality progress and the trajectory of a cohort that does benefit. Essentially what is needed to produce these patterns of convergence is a higher level of mortality, at least before some age, for the disadvantaged cohort and some heterogeneity in frailty, either innate or acquired with age.

This brings us to the question of how useful fixed frailty models are. Much of the theoretical work on heterogeneous population (as reviewed by Vaupel and Yashin 1985) as well as nearly all the empirical work (including Manton et al. 1981 and 1986, Heckman and Singer 1984, and Trussel and Richards 1985), has resorted to the simplifying assumption that an individual's frailty (or relative risk) does not vary, at least over the period being studied. That is, frailty is not necessarily assumed to be fixed from birth, but frailty is assumed to be constant after, say, age 65 if the analysis focuses on mortality rates at older ages. This may be realistic. Furthermore, as our model suggests (and as discussed by Vaupel and Yashin

1985), the assumption of fixed frailty may be a reasonable expedient if everyone's frailty changes by roughly the same absolute or proportional amount: much of this effect can be captured in the baseline force of mortality $\mu_0(x)$. Finally, if the action of homeostasis is to quickly restore individuals to their base level of frailty, deviations from this base may not be significant.

The key question then is whether substantial stochastic changes in frailty influence mortality trajectories in a way that cannot be captured by models that assume fixed frailty. Our model, and the various analytical explorations we have engaged in using it, indicate that debilitation associated with stochastic changes in frailty produces subsequent selection that helps counter-balance the effects of the debilitation. Furthermore, the main qualitative results in the theory of heterogeneity, concerning mortality convergence and the deviation of population trajectories from underlying individual trajectories, remain valid in most cases even given substantial stochastic debilitation. Nonetheless, stochastic changes in frailty can, as illustrated by the figures presented above, produce mortality trajectories quite different from the trajectories produced when frailty is fixed. Thus, mortality analysts, especially in their empirical research, may find it useful to employ a stochastic-process model when they have reason to believe that individual frailty may be changing turbulently.

DISCUSSION

Does a cohort remember mortality past? Are current and future mortality rates experienced by a cohort influenced by previous mortality rates or, more directly, by previous rates of morbidity and deprivation? Is there, in short, a cohort effect distinct from age and period effects, and if so, what is the nature of this effect? These questions have long been important in demographic thought and remain of central concern (Ryder 1965, Hobcraft, Menken, and Preston 1982). The model presented here is useful in conceptualizing and comprehending the complex nature of possible cohort effects and age-period-cohort interactions. The underlying age-pattern of mortality is captured by the baseline mortality function, μ_0. The vulnerability, drift, homeostasis, and stochasticity parameters capture effects that occur at specific ages or times. Because our focus was on a single cohort, we did not distinguish between age and time in our analysis, but it is readily possible to explicitly make these parameters functions of age and of time.

Our model and illustrative results suggest that it may not be productive to conceptualize or model age, period, and cohort mortality effects as three independent factors and that it is particularly questionable to assume that these effects are not only independent but also constant for each age, period, or cohort. Time-specific incidents of high mortality or morbidity probably affect cohorts of different ages differently and these incidents probably have lingering consequences that gradually decay as the result of recuperation, selection, and stochastic changes in individual frailty. Furthermore, debilitation and selection should not be thought of as independent factors. Debilitation that increases population heterogeneity will result in subsequent selection; selection, by altering the distribution of population heterogeneity, will influence the impact of debilitating events.

Correctly conceptualizing these factors is important for demographic theory and for understanding historical patterns of mortality. In addition, appropriate models of how cohorts remember past mortality and morbidity can contribute to public-health decisionmaking. If, for instance, high levels of morbidity in childhood can be linked to high levels of mortality at older ages, then efforts to reduce morbidity (and associated mortality) in childhood will have the double benefit of an immediate effect and a delayed effect. Understanding the magnitude of lingering mortality effects can thus help in determining the benefits of alternative public-health interventions and in targeting these interventions to achieve maximum benefits.

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