Influence of Cause of Death Structure on Age-Patterns of Mortality

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Variation in social, environmental, and medical conditions tends to affect the death rates of all age and sex groups. As a result of their mutual responsiveness, the death rates of any two of these groups are closely related to one another when data from a wide variety of populations are examined. Fifteen years ago, the demographer could refer to only one empirical description, or "model", of these mortality relationships; today, he confronts 13 one-parameter models and four two-parameter models.[1]*

The growth of such models reflects primarily the development of techniques for utilizing census data to infer vital rates not provided by the registration system. A well-known technique requires as a first step that one find a mortality function (death rate expressed as a function of age) that is broadly consistent with observed inter-censal survivorship.[2] Death and birth rate estimates are then readily calculated. In this context, the model of mortality relationships provides a set of candidates from which the best-fitting function can be selected.

Unfortunately, mortality relationships have not proved to be identical in all parts of the world and during all periods. Nor can discrepancies be attributed to random error. Coale and Demeny have identified four distinct and persistent age-patterns of mortality corresponding to four geographical regions of Europe.[3] Age patterns in certain less developed

* Footnotes appear at the end of the paper.

countries fail to conform to any existing models.[4] In this paper we will attempt to trace the source of discrepancy among age curves of mortality to differences in the structure of causes of death. The age-incidence of death varies among causes, and environmental conditions affect the intensity of various causes. Therefore, it is reasonable to expect that many pertinent environmental features will be transmitted to the age curve of mortality via the cause-of-death structure. In more formal terms, we attempt to show that the most general parametrization of the age curve of mortality should contain as many parameters as there are statistically unique causes of death. The aim is principally to unearth the foundation of existing models, rather than to add to their number. However, we consider briefly how information on cause-of-death structure might be utilized to fashion model mortality patterns suitable for use in a particular country or region.

Nature of the Data Base

This analysis utilizes data collected and tabulated by the author in collaboration with Nathan Keyfitz, Verne E. Nelson, and Robert Schoen.[5] Data on causes of death by age and sex were gathered and processed for 183 populations, representing 48 nations in the period 1861 to 1964. The principal criterion for inclusion in the collection was simply the availability of data on causes and numbers of persons in the appropriate age-sex categories, a criterion which weighted the sample heavily in favor of European countries. Except for the period 1959-64, when a nation was allowed to be represented twice, it rarely appeared in the collection more than once per decade. Twenty of the original 183 populations are excluded from most of the present analysis, primarily because of apparent peculiarities in the method of assigning cause of death.[6] In particular, the number of deaths in the category, "other and unknown causes," was unusually large in most of the excluded populations. This screening process does not imply, of course, that data for the remaining populations are free of error or peculiarity.

For each population and with distinction by sex, all of

the standard life table parameters were computed, together with crude, age-specific and age-standardized death rates from all causes combined and from twelve mutually exclusive and exhaustive cause of death groupings. The causes of death, and their corresponding numbers in the A-or B-list of the 7th Revision of the International Classification of Causes of Death, are[7]: respiratory tuberculosis (B1); other infectious and parasitic diseases (B2-17); malignant and benign neoplasms (B18-19); cardiovascular disease (B22, 24-29; A85, 86); influenza, pneumonia, bronchitis (B30-32); diarrhea, gastritis, enteritis (B36); certain chronic diseases (B20, 33, 37, 38) (these numbers represent, respectively: diabetes mellitus, ulcer of stomach and duodenum, cirrhosis of liver, and nephritis and nephrosis); maternal mortality (B40); certain diseases of infancy (B42-44); motor vehicle accidents (BE47); all other accidents and violence (BE48-50); and all other and unknown causes.

Procedures

The question that concerns us can be rephrased in the following way: "How much more accurately can age-curves of mortality be predicted on the basis of rates of death from various causes than on the basis of death rates from all causes combined?" Cause of death rates would offer no additional information if age-curves of mortality from the various causes were identical in shape, varying only in level, or if the level of mortality from each cause were perfectly correlated with the level of mortality from every other cause. (These conditions will be made more precise momentarily.) Figure I presents evidence on the first of these conditions in the form of average age curves of mortality in the 183 female populations from 10 of the 12 causes.[8] Their substantial diversity supports the potential value of decomposing mortality into causes of death.

In order to measure the added predictive value of cause-of death structure, we must first see how well age-patterns can be predicted in the absence of such information. Lacking a theoretically-sound and empirically-verified "law" of

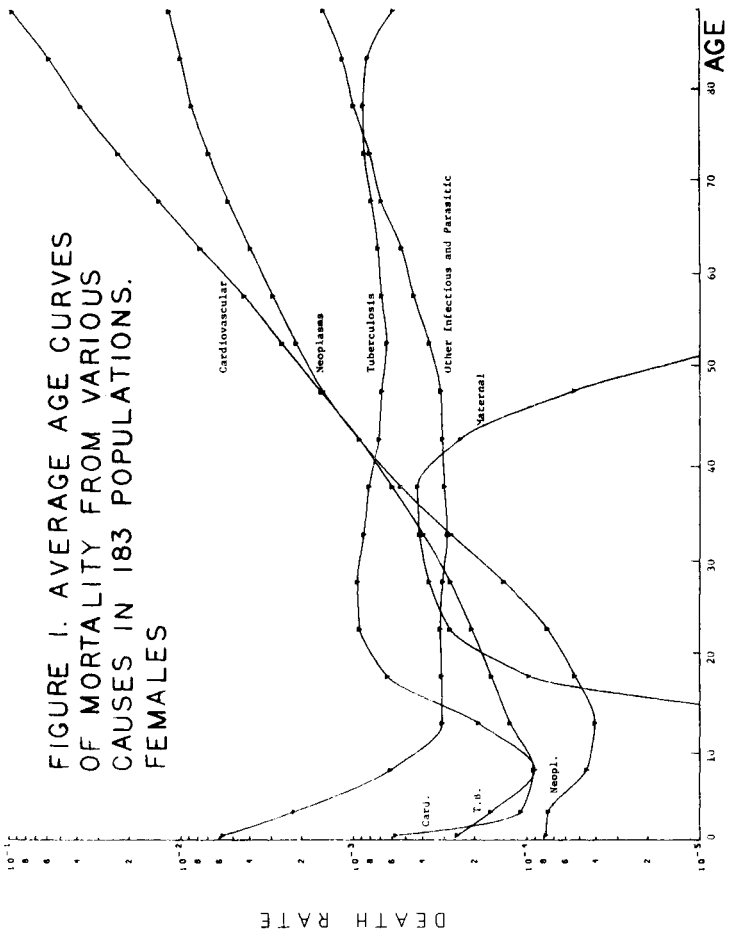


FIGURE I. AVERAGE AGE CURVES
OF MORTALITY FROM VARIOUS
CAUSES IN 183 POPULATIONS.
FEMALES
Cardiovascular
Neoplasms
Tuberculosis
Other Infectious and Parasitic
Maternal
Card.
T.B.
Neopl.
AGE
DEATH RATE

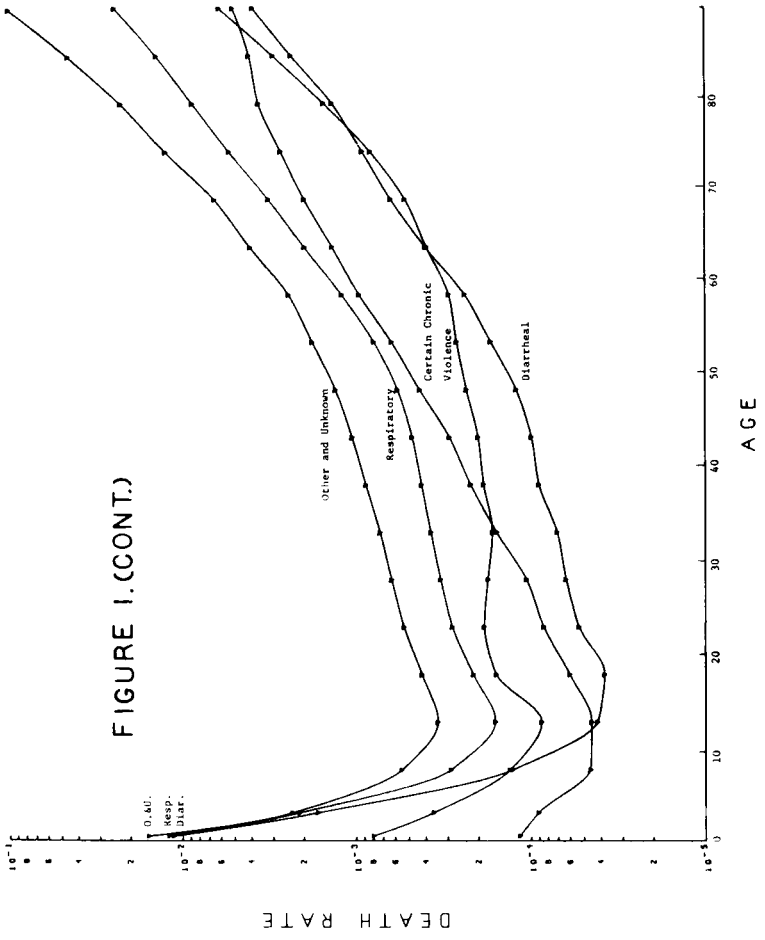


FIGURE I.(CONT.)
DEATH RATE
AGE
Other and Unknown
Respiratory
Certain Chronic
Violence
Diarrheal
0.&U.
Resp.
Diar.

human mortality, we must rely upon observation to indicate the general nature of the relationship among death rates at various ages. The United Nations model patterns employ second-degree polynomial regressions to summarize the relationship between death rates in adjacent age intervals, but the contribution of the second-degree term is typically quite small. In their reformulation of the U. N. models, Gabriel and Ronen ignore the second-degree term altogether, and Ledermann's relationships are linear except for those involving ${}_4q_1$. Even here the nonlinearity is virtually invisible to the naked eye.[9]

If the death rate at each age is linearly related to the death rate at every other age, then it is linearly related to a linear combination of death rates at all ages. We will measure mortality "level" by such a linear combination of age-specific death rates, in particular by what is commonly termed an age-standardized crude death rate:

$$\text{ASCDR} = \sum_i c_i^s \cdot M_i \ ,$$

where c_i^s = proportion of the standard population in the ith age interval

M_i = death rate in the ith age interval

Any linear combination will do; in practice we choose one which gives smoothly-descending weights to death rates at higher and higher ages.[10] Note that the expectation of life is not a linear combination of age-specific death rates. Instead, the e_o^o implicitly assigns to death rates at the several ages weights which vary from population to population, depending on the mortality function itself. To demonstrate the differential responsiveness of the two measures to a change in mortality level, we write the death rate at each age x, μ_x, as a linear function of the death rate at one particular age, z .

$$\mu_x = a_x + b_x \mu_z$$

$$\text{ASCDR} = \int_0^\infty c_x^s \cdot \mu_x dx$$

$$= \int_0^\infty c_x^s (a_x + b_x \mu_z) dx$$

$$= \int_0^\infty c_x^s a_x dx + \mu_z \int c_x^s b_x dx$$

$$\frac{d\text{ASCDR}}{d\mu_z} = \int_0^\infty c_x^s b_x dx \quad .$$

Obviously the ASCDR is a linear function of the mortality level, as measured by μ_z .

Letting $p(x)$ equal the probability of surviving from birth to age x, we define life expectancy in the conventional way as

$$e_0^0 = \int_0^\infty p(x)dx = \int_0^\infty e^{-\int_0^x \mu_t dt} dx$$

$$= \int_0^\infty e^{-\int_0^x [a_t + b_t \mu_z] dt} dx$$

$$\frac{de_0^0}{d\mu_z} = \int_0^\infty e^{-\int_0^x a_t dt} \cdot -\left[\int_0^x b_t dt\right] \cdot e^{-\mu_z \int_0^x bt\, dt} dx$$

$$= -\int_0^\infty p(x) \left[\int_0^x b_t dt\right] dx \quad .$$

Thus the change in life expectancy per unit change in μ_z depends upon the initial level of mortality itself. The two changes are not linearly related. As a result, the observed relationship between the death rate at a particular age and life expectancy is curvilinear, as noted empirically by Ledermann.[11]

We can therefore expect the relationship between death rates at a particular age and overall mortality level to be satisfactorily represented by a linear function on the age-standardized crude death rate. Linear correlation and regression analysis is a convenient means of deriving the coefficients of the relationships and measuring their goodness-of-fit. Examination of residuals from the regressions indicates that the error assumptions of classical least squares regression are not severely violated in our data.

Before proceeding to the calculations, we modify the dependent variable slightly by first noting that the probability of survival between age x and age $x + n$ can be written

$$ {}_nP_x = e^{-\int_x^{x+n} \mu_t \, dt} . $$

Thus,

$$ - \log {}_nP_x = \int_x^{x+n} \mu_t \, dt . $$

Since mortality at all ages is assumed to be a linear function of the age-standardized crude death rate, and since the natural logarithm of the probability of survival is simply a sum of age-specific death rates, it is also a linear function of the age-standardized crude death rate.

$$ \log {}_nP_x = {}_nA_x + {}_nB_x \cdot \text{ASCDR} . \tag{1} $$

Equation (1) provides a convenient means of relating the index of mortality, ASCDR, to life table parameters, and will be utilized throughout.

Coefficients of the relationships between $\log_n p_x$ in various age intervals and the age-standardized crude death rate are displayed in Table I. These coefficients are calculated by classical least squares methods applied to the 163 observations. For convenience, we deal throughout with six age intervals. It should be noted that the values of mortality parameters for one of these age intervals are based upon mortality rates in intervals of 5 years or less. Regardless of the width of the interval displayed, its parameters are not subject to age distributional disturbances.

The model of mortality relationships contained in Table I is very similar to that of the United Nations' model life tables and the Coale-Demeny "West" pattern. Both of these represent "average" patterns, the former based upon all populations examined and the latter upon all populations remaining after anomalous cases had been removed. Table II compares the female survivorship columns of the three models at four different levels of mortality. The three models are in close correspondence, especially when contrasted with other regional families. They are virtually identical at the lowest level of mortality. At the next lowest level, Table I's survivorship column is always intermediate between the other two, despite their crossing. Thereafter a slight tendency emerges for Table I's pattern to display lower death rates at older ages, and the U. N. pattern slightly higher rates at those ages. In short, the equations of Table I produce models of mortality relationships quite similar to those of two other "average" patterns, despite differences in the sample of populations and in the specification of the relationships.[12] We can be confident that neither our sample nor our specification substantially misrepresents average mortality relationships.

Table I also presents the coefficients of linear correlation between the death rate in various age intervals ($-\log_n p_x$) and the age-standardized death rate at all ages combined. Since all but two of the coefficients lie between .90 and .95, they imply that 80-90% of the variance in the death rate at a particular age could be "explained" by variation in the death rate at all ages combined.[13] For some purposes, this is an acceptable degree of predictability. For use in inferring vital

Table I. Regression Equations Relating Life Table Functions to Level of Mortality

$$\log(\ell_{x+n}/\ell_x) = a_x + b_x \cdot \text{ASCDR} + \varepsilon$$

Dependent Variable	a_x	b_x	Correlation Coefficient	Cause of Death Having Highest Partial Correlation with $\log(\ell_{x+n}/\ell_x)$, Holding Constant ASCDR	
			FEMALES		
$\log(\ell_1/\ell_0)$	.05356	-9.8722 (.2947)	-.935	Cert. Dis. of Infancy	(-.606)
$\log(\ell_5/\ell_1)$	.05977	-7.7134 (.2243)	-.938	Diarrheal Diseases	(-.582)
$\log(\ell_{20}/\ell_5)$	.02982	-4.5684 (.1162)	-.952	Infect. and Parasitic	(-.499)
$\log(\ell_{40}/\ell_{20})$	.05411	-9.9101 (.2642)	-.947	Tuberculosis	(-.661)
$\log(\ell_{60}/\ell_{40})$	.00036	-13.5567 (.4520)	-.921	Cert. Chronic Dis.	(-.506)
$\log(\ell_{80}/\ell_{60})$	-.44342	-40.0032 (1.2358)	-.931	Cardiovascular	(-.434)

Table I Continued

Dependent Variable	a_x	b_x	Correlation Coefficient	Cause of Death Having Highest Partial Correlation with $\log(\ell_{x+n}/\ell_x)$, Holding Constant ASCDR	
			MALES		
$\log(\ell_1/\ell_0)$	.09451	-11.5953 (.3482)	-.934	Cert. Dis. of Infancy	(-.609)
$\log(\ell_5/\ell_1)$	.07836	- 7.5193 (.2717)	-.909	Infect. and Parasitic	(-.624)
$\log(\ell_{20}/\ell_5)$	.03007	- 3.8824 (.1467)	-.902	Infect. and Parasitic	(-.694)
$\log(\ell_{40}/\ell_{20})$	.06562	- 9.5712 (.3104)	-.925	Tuberculosis	(-.475)
$\log(\ell_{60}/\ell_{40})$	.00675	-15.8733 (.4794)	-.934	Violence	(-.422)
$\log(\ell_{80}/\ell_{60})$	-.67160	-34.2633 (1.8687)	-.822	Cardiovascular	(-.610)

Table II. Proportions Surviving from Birth to Age x In Various Model Female Life Tables at Four Levels of Mortality

Age (x)	Coale-Demeny "West", $e_0^0 = 72.5$	Computed from Table I ASCDR=.00806	U.N., $e_0^0 = 73.09$	Coale-Demeny "West", $e_0^0 = 60.0$	Computed from Table I ASCDR=.0133	U.N., $e_0^0 = 60.17$
0	1.000	1.000	1.000	1.000	1.000	1.000
1	.977	.974	.978	.929	.926	.918
5	.973	.972	.973	.899	.886	.886
20	.966	.966	.965	.870	.860	.858
40	.943	.941	.939	.800	.795	.793
60	.849	.844	.846	.656	.664	.669
80	.392	.392	.399	.230	.250	.253

Age (x)	Coale-Demeny "West", $e_0^0 = 47.5$	Computed from Table I ASCDR=.0205	U.N., $e_0^0 = 47.63$	Coale-Demeny "West", $e_0^0 = 35.0$	Computed from Table I ASCDR=.0300	U.N., $e_0^0 = 34.31$
0	1.000	1.000	1.000	1.000	1.000	1.000
1	.868	.862	.860	.786	.784	.791
5	.797	.781	.795	.672	.661	.681
20	.743	.733	.743	.597	.594	.601
40	.634	.631	.635	.459	.465	.449
60	.469	.478	.478	.292	.310	.274
80	.125	.135	.131	.050	.060	.046

Sources: Ansley J. Coale and Paul Demeny, Regional Model Life Tables and Stable Populations. Princeton University Press, 1966. pp. 8, 13, 18, 23.
United Nations, Department of Social Affairs. Age and Sex Patterns of Mortality. Population Study No. 22, 1955, pp. 20-21.
Table I.

rates from census data, however, one seeks greater accuracy. Even relatively minor variations among model age curves of mortality can produce major differences in estimates of demographic parameters. Their sensitivity results in large part from the fact that, in census age distributions, a recent infant death is indistinguishable from a birth that never occurred. For example, United Nations Manual IV shows that stable estimates of birth rate in a hypothetical population vary from 44.5 to 50.1/1000, depending upon whether the "West" or "South" model mortality patterns are used.[14]

How much of the unexplained variance in age specific death rates is caused by differences in the cause-structure of mortality at a particular level? Table I permits some initial observations on this point. Partial correlation coefficients between $\log_n p_x$ and age-standardized death rates from particular causes ($ASCDR_i$) are displayed, with ASCDR held constant. The causes of death that are most closely associated with age-specific death rates for both sexes are, starting at the lowest ages: certain diseases of early infancy; infectious and parasitic diseases; tuberculosis; and cardiovascular disease. It is almost superfluous to note that a disease proves to be significant at an age where one expects its relative incidence to be greatest.[15] This result offers the first indication of the importance of cause of death structure for age-curves of mortality.

Model Age-Patterns of Mortality Incorporating Causes of Death

In order to introduce causes of death into model age-patterns of mortality, we require information on the manner in which age-specific death rates from a particular cause are related to one another among populations. In this matter we have no previous studies available for reference, and we must rely upon the cause of death data forming the basis of this study. Unfortunately, there are 3,672 individual relationships between cause-specific death rates at two different ages to be examined, with 163 observations on each. As a result, it was necessary to draw samples both from the

total number of possible relationships and from the number of observations available on each. In the process of plotting these relationships, it quickly became apparent that linearity continued to be a good approximation of the shape of these relationships. For several causes of death, most notably other infectious and parasitic diseases, the constant terms were typically quite close to zero. Some evidence on the nature of these cause-specific relationships will be presented below.

Let us suppose, then, that for each cause of death i, for every age x and every age interval θ,

$$\mu_x^i = a_{x,\theta}^i + b_{x,\theta}^i \cdot \mu_{x+\theta}^i \quad .$$

Then

$$\mu_x^i = A_x^i + B_x^i \cdot \text{ASCDR}^i \quad .$$

Thus the cause-specific death rate at each age x is a linear function of the age-standardized death rate from the cause. And since

$$\mu_x = \sum_i \mu_x^i \quad ,$$

$$\mu_x = \sum_i A_x^i + \sum_i B_x^i \cdot \text{ASCDR}^i$$

$$\log {}_n p_x = A'_x + \sum_i B_x^{i'} \cdot \text{ASCDR}^i \quad . \tag{2}$$

Equation (2) is the form in which we shall develop model age curves of mortality that incorporate causes of death. Note that if age curves of mortality or of mortality variation from the various causes were identical to one another, the B_x^i's would be equal for all i at a given x and equation (2)

would reduce to equation (1). Similarly, if causes of death were always found in some linear combinations, then for all k

$$\begin{aligned} ASCDR^k &= P^1 + Q^1 \cdot ASCDR^1 \\ &= P^2 + Q^2 \cdot ASCDR^2 \\ &\vdots \\ &= P^{12} + Q^{12} \cdot ASCDR^{12} \end{aligned}$$

and equation (2) would once again reduce to (1). Death rates from various causes would have no added value if either the causes' age curves were identical or their rates were always found in linear combination. We have already seen evidence that these conditions do not apply.

We first estimate the parameters of equation (2) with all twelve causes of death as independent variables, and then attempt to reduce the number of causes without sacrificing substantial explanatory power. A stepwise regression procedure is used, in which the next variable entering the equation is that having the highest partial correlation (at that stage) with the dependent variable. With this procedure, we can determine the most significant causes at each age, information vital to the reduction process. Tables III-a and III-b designate the first five causes of death entering each equation, in order, and present their coefficients after five causes have entered. They also present the correlation coefficients that result when all twelve causes have entered.

The coefficient of every cause of death in each equation of Table II is at least twice the size of its standard error, many exceeding it by a factor of 10 to 15. Assuming that errors are normally distributed, each of the coefficients is significant at a 1% level. Once again, causes of death are generally significant at ages where their incidence is highest (except for the few causes with positive coefficients, appearing late in the step-wise procedure, for which exactly

the reverse is true). Under certain circumstances, the B_x^i coefficients would have a very direct interpretation. Suppose that all relationships are deterministic and that in every population the age-curve of death rates from a cause is some constant multiple (varying from population to population) of a standard age curve. Then

$$\mu_x^i = K^i \cdot \mu_x^{iS}, \quad \text{where } S \text{ denotes the standard curve}$$

$$ASCDR^i = K^i ASCDR^{iS}$$

$$\mu_x = K^1 \mu_x^{1S} + K^2 \mu_x^{2S} + \ldots + K^{12} \mu^{12S}$$

$$= \frac{ASCDR^1}{ASCDR^{1S}} \cdot \mu_x^{1S} + \frac{ASCDR^2}{ASCDR^{2S}} \cdot \mu_x^{2S} + \ldots + \frac{ASCDR^{12}}{ASCDR^{12S}} \cdot \mu_x^{12S}$$

$$= \frac{\mu_x^{1S}}{ASCDR^{1S}} \cdot ASCDR^1 + \frac{\mu_x^{2S}}{ASCDR^{2S}} \cdot ASCDR^2 + \ldots$$

$$+ \frac{\mu^{12S}}{ASCDR^{12S}} \cdot ASCDR^{12} \quad .$$

That is, the coefficient of $ASCDR^i$ at age x would equal the death rate in the standard population from cause i at age x, divided by the age-standardized death rate from cause i in the standard population. Since the divisor remains the same from age to age, the age-sequence of coefficients for a particular cause would uniquely provide the age-curve of mortality from that cause in the standard population.[16] The standard age-curve of mortality implicit in the data could be directly inferred from the Beta-coefficients, a neat and time-saving procedure.

For certain major causes of death this device works reasonably well. The age-sequence of Beta coefficients for

respiratory disease in Table III, for example, is U-shaped, like the typical age curve of mortality from the cause. For many causes, however, the age-sequence of Beta-coefficients is quite erratic. The standard age-patterns of mortality from a cause must in general be developed by more laborious procedures.

The pattern of causes of death depicted in Table III may illuminate the matrix of intercorrelations among age-specific death rates (all causes combined), shown in Table IV. The death rate at a particular age is most highly correlated with death rates at adjacent ages. An obvious explanation, verified in Table II, is that causes of death which are important for one age also tend to be important for adjacent ages. The highest correlation for both sexes is between death rates at ages 5-20 and 20-40, ages whose significant causes of death show a high degree of concordance. On the other hand, two of the three leading causes of death at infancy appear also at ages 1-4 but never again through the remainder of the age span. This may account for the relatively low correlations between infant mortality and death rates at ages over 5, also noted elsewhere. However, the male death rate at ages 60-80 is more closely correlated with infant mortality than with mortality in middle-life, a feature also uncovered by Coale and Demeny for both sexes in the "West" and "North" patterns.[17] Table II indicates that the explanation may lie in the pre-eminent importance at both ages of respiratory disease, a major cause of death with a sharply-angled, U-shaped age curve.

The central question is the gain in predictability of age-specific rates from the introduction of information on causes of death. We shall measure the gain by the proportionate reduction in the amount of variance left unexplained by the age-standardized crude death rate. Table V presents these figures for the age intervals which we have distinguished. We succeed in removing between 41% and 72% of the variance previously left unexplained by the death rate from all causes combined, with a mean of 60% and a median of 62%. The largest gains occur at ages under 5, where certain diseases of infancy and diarrheal deaths are of greatest importance; at

Table III-a. Parameters of Linear Regressions after Five Causes of Death Have Entered the Equations. Females. N = 163.

Dependent Variable	Constant	First Cause of Death Entering Equation, Its Coefficient and Standard Error After Five Causes Have Entered	Second Cause of Death	Third Cause of Death
$\log(\ell_1/\ell_0)$	.0024	Respiratory	Cert. Dis. of Infancy	Diarrheal
		-12.135	-70.099	-25.660
		(1.861)	(5.218)	(2.224)
$\log(\ell_5/\ell_1)$	.0039	Infectious and Parasitic	Diarrheal	Respiratory
		-21.277	-21.661	-7.234
		(1.909)	(1.813)	(1.308)
$\log(\ell_{20}/\ell_5)$	.0085	Infectious and Parasitic	Tuberculosis	Other and Unknown
		-13.198	-13.335	-3.633
		(1.008)	(1.002)	(.439)
$\log(\ell_{40}/\ell_{20})$	.0004	Tuberculosis	Maternal	Other and Unknown
		-36.848	-100.554	-5.355
		(2.235)	(15.786)	(.824)
$\log(\ell_{60}/\ell_{40})$	-.0711	Maternal	Respiratory	Cert. Chronic Diseases
		-161.886	-15.525	-50.745
		(41.366)	(3.466)	(7.571)
$\log(\ell_{80}/\ell_{60})$	-.3070	Respiratory	Cardiovascular	Other and Unknown
		-64.628	-92.206	-54.822
		(7.202)	(7.074)	(4.937)

Table III-a Continued

Fourth Cause of Death	Fifth Cause of Death	Multiple Correlation After 5 Causes are Entered	Multiple Correlation After all 12 Causes are Entered
Tuberculosis	Infectious and Parasitic	.974	.979
-16.076	7.028		
(2.657)	(2.406)		
Cert. Dis. of Infancy	Cert. Chronic Diseases	.972	.982
-14.400	8.243		
(4.229)	(2.633)		
Cert. Chronic Diseases	Cardiovascular	.979	.981
-4.984	-1.660		
(1.377)	(.520)		
Cert. Chronic Diseases	Infectious and Parasitic	.983	.985
-22.745	-12.443		
(3.114)	(2.081)		
Infectious and Parasitic	Tuberculosis	.947	.958
-25.081	-21.922		
(4.445)	(5.712)		
Other Violence	Automobile Accidents	.954	.965
-272.815	672.210		
(63.882)	(173.757		

Table III-b. Parameters of Linear Regressions after Five Causes of Death Have Entered the Equations. Males. N = 163

Dependent Variable	Constant	First Cause of Death Entering Equation, Its Coefficient and Standard Error After Five Causes Have Entered	Second Cause of Death	Third Cause of Death
$\log(\ell_1/\ell_0)$	.0136	Respiratory	Cert. Dis. of Infancy	Diarrheal
		-10.747	-68.517	-18.106
		(1.632)	(4.843)	(2.486)
$\log(\ell_5/\ell_1)$	.0051	Infectious and Parasitic	Diarrheal	Cert. Dis. of Infancy
		-22.565	-17.255	-18.418
		(2.059)	(2.010)	(4.222)
$\log(\ell_{20}/\ell_5)$	.0001	Infectious and Parasitic	Tuberculosis	Other Violence
		-14.324	-4.979	-5.451
		(1.100)	(1.016)	(1.790)
$\log(\ell_{40}/\ell_{20})$	-.0210	Tuberculosis	Infectious and Parasitic	Other Violence
		-31.158	-14.750	-40.886
		(2.367)	(2.373)	(3.539)
$\log(\ell_{60}/\ell_{40})$	.0006	Respiratory	Tuberculosis	Other Violence
		-18.758	-41.842	-53.522
		(2.975)	(5.385)	(7.786)
$\log(\ell_{80}/\ell_{60})$	-.0978	Respiratory	Cardiovascular	Other and Unknown
		-57.419	-102.091	-84.496
		(6.878)	(6.630)	(6.331)

Table III-b Continued

Fourth Cause of Death	Fifth Cause of Death	Multiple Correlation After 5 Causes are Entered	Multiple Correlation After all 12 Causes are Entered
Other and Unknown -4.842 (.984)	Tuberculosis -10.778 (2.634)	.975	.978
Respiratory -4.421 (1.253)	Cert. Chronic Diseases 7.186 (2.581)	.960	.967
Other and Unknown -1.914 (.445)	Cert. Dis. of Infancy -6.329 (2.166)	.960	.962
Cancer 11.666 (3.232)	Respiratory -4.405 (1.433)	.970	.974
Cardiovascular -20.142 (2.342)	Other and Unknown -11.628 (2.032)	.945	.962
Cancer -141.854 (21.468)	Cert. Chronic Diseases -93.258 (17.678)	.928	.936

ages 20-40, where tuberculosis can disturb normal mortality patterns; and ages 60-80 for males, where cardiovascular disease behaves in a largely autonomous fashion. It appears that a good deal of the variability in age curves of mortality can be attributed to cause of death structures which differ among populations at the same level of mortality.

Reducing the Number of Causes

The number of causes can be reduced by half without forfeiting a substantial amount of explanatory power. Causes which rarely or never appear in Table III — automobile accidents, other violence, cancer, certain chronic diseases - need not be enumerated individually. Groups of causes that are persistently important in the same age intervals - certain diseases of infancy and diarrheal disease, or cardiovascular disease, cancer, and "other and unknown" - can, in practice, be successfully combined.

After a certain amount of trial and error, the 12 cause of death categories were collapsed into 6 for males and 7 for females. The new categories are:

I. Tuberculosis

II. Other Infectious and Parasitic Diseases

III. Respiratory Disease

IV. Diarrheal Disease and Certain Diseases of Infancy

V. All Violence

VI. Maternal Complications (Females Only)

VII. All Other and Unknown

Diarrheal diseases are combined with certain diseases of early infancy not simply because of the similarity of their age patterns of influence. Under the 6th and 7th Revisions

of the International List of Causes of Death, deaths from diarrhea in the first four weeks of life are assigned to CDEI, an assignment not operative in other Revisions. Moreover, deaths from "toxicosis" under age one are assigned to CDEI, although the principal cause of this condition is diarrhea. According to Verhoestraete and Puffer, this important coding peculiarity in Latin America frequently prevents diarrhea from emerging as the leading cause of death in infancy, and improprerly projects CDEI into this position.[18]

The new cause-of-death regressions are presented in Table VI. The correlation coefficients are such that an average of 82% of the gain in explanatory power that resulted from using all 12 causes of death is retained. Thus the structure of important causes of death can be simplified considerably at little cost. Four of the remaining six causes for males are diseases that are themselves frequently grouped under the title "infectious and parasitic diseases." However, these four contain quite different implications for age curves of mortality, and such a grouping is clearly inappropriate in this context.

Application to Actual Populations

The results indicate that discrepancies in age patterns of mortality are frequently produced by differences in the structure of causes of death. In this section we offer additional evidence for this proposition by comparing specific populations having the same level of mortality but divergent age curves. Of special interest are representatives of the several regional groups identified by Coale and Demeny. England and Wales and Italy provide the earliest data on causes of death of any populations in these groups. England and Wales belongs to the "West" regional group and Italy to the "South."[19] The "South" pattern is characterized by high mortality under age 5, low mortality between ages 40 and 60, and high mortality above age 65.[20] The "West," of course, shows no systematic pattern of deviation.

Table VII presents the male age-standardized crude death rates from the six causes for England and Wales in

Table IV. Matrix of Correlation Coefficients Between Death Rates in Various Age Groups

	$\log(1_1/1_0)$	$\log(1_5/1_1)$	$\log(1_{20}/1_5)$	$\log(1_{40}/1_{20})$	$\log(1_{60}/1_{40})$	$\log(1_{80}/1_{60})$
			FEMALES			
$\log(1_1/1_0)$	1.000	.913	.868	.867	.827	.813
$\log(1_5/1_1)$	.915	1.000	.919	.856	.796	.823
$\log(1_{20}/1_5)$	.878	.917	1.000	.963	.877	.842
$\log(1_{40}/1_{20})$	.891	.845	.936	1.000	.942	.836
$\log(1_{60}/1_{40})$	.829	.765	.792	.889	1.000	.853
$\log(1_{80}/1_{60})$	.634	.613	.577	.615	.792	1.000
			MALES			

Table V. Reduction in Unexplained Variance from Introducing Data on Causes of Death

	r_1^2 with ASCDR*	r_2^2 with ASCDR$_i$**, i=1...12	$\frac{(1-r_1^2)-(1-r_2^2)}{(1-r_1^2)}$ Proportionate Reduction in Unexplained Variance
		FEMALES	
$\log(\ell_1/\ell_0)$	.874	.958	.670
$\log(\ell_5/\ell_1)$	.880	.964	.703
$\log(\ell_{20}/\ell_5)$	.906	.962	.598
$\log(\ell_{40}/\ell_{20})$	.897	.970	.711
$\log(\ell_{60}/\ell_{40})$	.848	.918	.458
$\log(\ell_{80}/\ell_{60})$	.867	.931	.484
		MALES	
$\log(\ell_1/\ell_0)$	.872	.956	.659
$\log(\ell_5/\ell_1)$	.826	.935	.626
$\log(\ell_{20}/\ell_5)$	.814	.925	.600
$\log(\ell_{40}/\ell_{20})$	.856	.949	.645
$\log(\ell_{60}/\ell_{40})$	.872	.925	.416
$\log(\ell_{80}/\ell_{60})$	.676	.876	.618

*Source: Table I

**Source: Table III

Table VI. Coefficients of Regression Equations with Reduced Number of Causes of Death

	Constant	Coefficient of Tuberculosis	Coefficient of Infectious and Parasitic	Coefficient of Respiratory
				FEMALES
$\log(\ell_1/\ell_0)$	.00866	-9.9285	10.8098	-14.9013
		(3.3306)	(2.7983)	(2.0959)
$\log(\ell_5/\ell_1)$	.02004	-6.1873	-20.3757	-4.2076
		(2.3737)	(1.9943)	(1.4937)
$\log(\ell_{20}/\ell_5)$	.01265	-14.0136	-13.8533	.5256
		(1.1926)	(1.0540)	(.7608)
$\log(\ell_{40}/\ell_{20})$	.02142	-34.1307	-10.0233)	
		(2.4470)	(2.6321)	
$\log(\ell_{60}/\ell_{40})$	.00902	-15.1575	-15.0771	-11.0574
		(5.5709)	(4.6805)	(3.5057)
$\log(\ell_{80}/\ell_{60})$	-.21010	25.3880	-47.3659	-81.5535
		(15.9044)	(13.3625)	(10.0084)
				MALES
$\log(\ell_1/\ell_0)$	.02454	-10.7699	-1.6411	-13.5127
		(3.1820)	(2.9641)	(2.0318)
$\log(\ell_5/\ell_1)$	.02610	-.9512	-22.3760	-2.6001
		(2.1811)	(2.0317)	(1.3927)
$\log(\ell_{20}/\ell_5)$	.00579	-5.3421	-16.6080	-.3039
		(1.2358	(1.1511)	(.7891)
$\log(\ell_{40}/\ell_{20})$	.02524	-31.6330	-18.0716	-2.5905
		(2.5301)	(2.3569)	(1.6156)
$\log(\ell_{60}/\ell_{40})$	.03922	-36.1242	-10.8434	-15.5975
		(4.7289)	(3.8911)	(2.8852)
$\log(\ell_{80}/\ell_{60})$	-.18828	37.1778	19.9587	-65.3533
		(15.6107)	(14.5417)	(9.9678)

Table VI. Continued

Coefficient of Diarrheal and Cert. Dis. of Infancy	Coefficient of Violence	Coefficient of Other and Unknown	Coefficient of Maternal	R^2
-30.5294	8.4661	-2.5468	-58.7727	.937
(2.4384)	(11.8654)	(1.1025)	(21.2818)	
-20.0184	4.3736	-2.0869	31.3303	.947
(1.7378)	(8.4561)	(.7858)	(15.1670)	
-2.3515	2.9396	-2.5863		.955
(.9293)	(4.4175)	(.4138)		
-3.0013	-8.2513	-4.9133	-132.1588	960
(1.7830)	(9.2271)	(.8587)	(15.7858)	
7.1795	-79.2064	-11.5012	-217.8493	.908
(4.0784)	(19.8461)	(1.8441)	(35.5961)	
10.0419	-113.3955	-74.5970	331.710	.912
(11.6435)	(56.6589)	(5.2648)	(101.6238)	
-31.9127	-17.4076	-1.8465		.929
(2.6373)	(4.5058)	(1.1565)		
-16.8333	6.0128	-2.6772		.925
(1.8078)	(3.0885)	.7927		
-1.8474	-4.9403	-1.1097		.910
(1.0242)	(1.7499)	(.4491)		
-4.8679	-37.5044	-2.0634		.935
(2.0971)	(3.5827)	(.9196)		
	-56.8239	-15.8312		.916
	(6.7189)	(1.7143)		
8.9527	20.0802	-98.5621		.848
(12.9386)	(22.1050)	(5.6738)		

Table VII. Age-Standardized Crude Death Rates from All Causes and Specific Causes in Five Populations

Disease	England and Wales, Males, 1871. $e_0^0 = 39.16$	Italy, Males, 1891 $e_0^0 = 38.48$	United States, Females, 1940 $e_0^0 = 65.43$	Norway, Females, 1930, $e_0^0 = 65.78$	Portugal, Females, 1960, $e_0^0 = 66.72$
Respiratory Tuberculosis	.00260	.00091	.00033	.00120	.00024
Other Infectious and Parasitic	.00389	.00355	.00028	.00055	.00021
Influenza, Pneumonia, Bronchitis	.00400	.00338	.00083	.00095	.00096
Diarrheal and Cert. Dis. of Infancy	.00232	.00449[1]	.00057	.00047	.00134[2]
Violence	.00125	.00067	.00063	.00017	.00027
Maternal	--	--	.00012	.00009	.00005
All Other	.01335	.01436	.00883	.00702	.00695
All Causes	.02741	.02736	.01159	.01045	.01002

1. Diarrheal = .00313; Cert. Dis. of Infancy = .00136
2. Diarrheal = .00067; Cert. Dis. of Infancy = .00067

Source: Samuel H. Preston, Nathan Keyfitz, and Robert Schoen, Causes of Death: Life Tables for National Populations. Seminar Press. New York. 1972.

1871 and for Italy in 1891. The life expectancies of the two populations are quite close at 39.16 and 38.48. The cause-of-death structure differs substantially in the two cases, however, with England and Wales exhibiting higher death rates from tuberculosis and other infectious and parasitic diseases. Italy's death rate is much higher from diarrheal diseases/certain diseases of infancy, of which the former entity is the more important.

The age sequence of death probabilities in the two populations are presented graphically in Figure II.[21] The pattern of deviations agrees with that noted by Coale and Demeny. More important, the deviations are predicted quite successfully by the cause-structures in the two populations. Cause of death rates displayed in Table VII are substituted into the male equations in Table VI, and the resulting predictions shown in Figure II, joined by a dotted line. Since the actual age-curves are predicted quite accurately, so are differences between them.

At a higher level of life expectancy, representatives from three of the four regional patterns can be compared. Table VII presents the female age-standardized death rates from the seven causes in the United States, 1940, Norway, 1930, and Portugal, 1960. The life expectancies in these populations are, respectively, 65.43, 65.78, and 66.72. The Northern representative, Norway, displays the highest death rate from tuberculosis and other infectious and parasitic diseases. The Southern country again displays the highest death rate from diarrheal/certain diseases of infancy. The United States, from the "West", is intermediate in these important causes.

Probabilities of dying for the three countries are plotted in Figure III and compared to the probabilities predicted on the basis of a country's cause-structure in combination with the "female" equations on Table VI. Once, again, divergencies are predicted rather well. The ranking of the three countries is predicted correctly at all ages except infancy, where the order of the United States and Norway is reversed. Thus eventeen of the eighteen rankings of death probabilities in any pair of these three countries are preserved in the predictions.

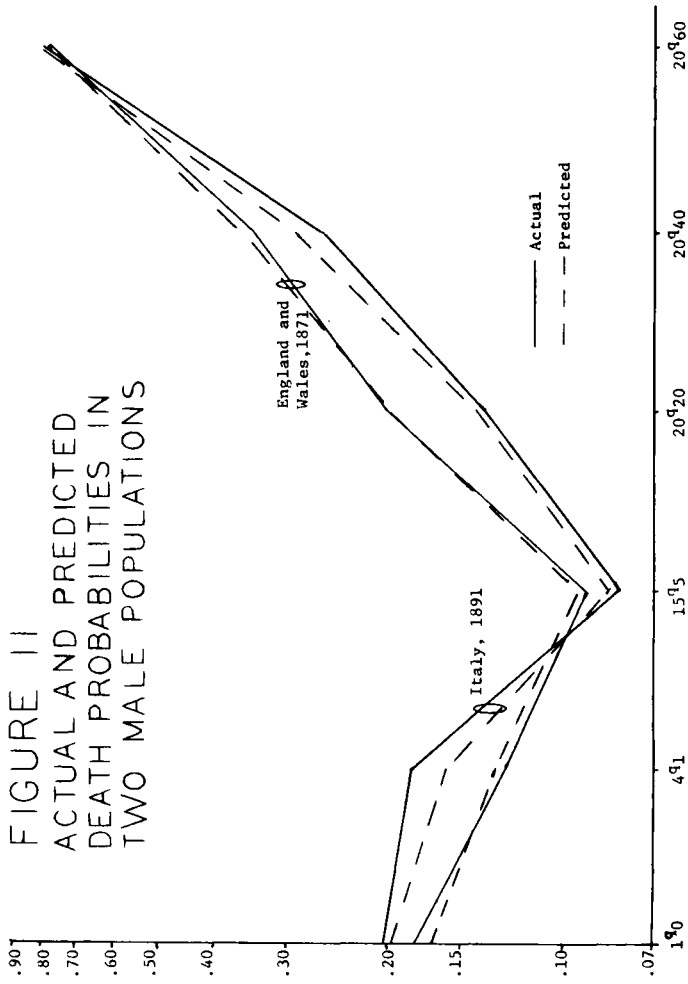


FIGURE 11
ACTUAL AND PREDICTED
DEATH PROBABILITIES IN
TWO MALE POPULATIONS
.90
.80
.70
.60
.50
.40
.30
.20
.15
.10
.07
$_{1}q_{0}$
$_{4}q_{1}$
$_{15}q_{5}$
$_{20}q_{20}$
$_{20}q_{40}$
$_{20}q_{60}$
England and Wales,1871
Italy, 1891
Actual
Predicted

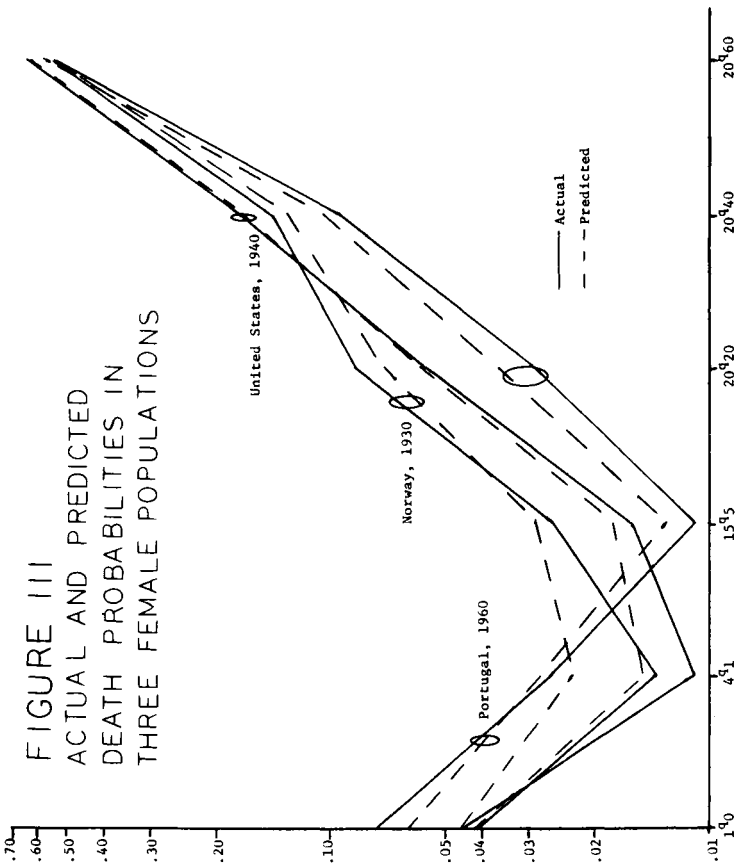


FIGURE III
ACTUAL AND PREDICTED
DEATH PROBABILITIES IN
THREE FEMALE POPULATIONS
.70
.60
.50
.40
.30
.20
.10
.05
.04
.03
.02
.01
$_1q_0$
$_4q_1$
$_{15}q_5$
$_{20}q_{20}$
$_{20}q_{40}$
$_{20}q_{60}$
United States, 1940
Norway, 1930
Portugal, 1960
Actual
Predicted

Many other examples of such comparisons could be presented. Czechoslovakia in 1934 is the only available representative of the "East" pattern before World War II. When compared to New Zealand, 1881, at the same general level of mortality, Czechoslovakia had higher death rates in infancy and ages 60-80 (typical of the East group) and much higher death rates from respiratory and diarrheal diseases. Consequently, their differences in cause of death structure are predicted quite accurately. In more recent years, however, members of the "East" family show infant mortality rates substantially higher than predicted levels.

The age structure of mortality in many non-Western nations can be predicted as accurately as that of western countries at the same level of life expectancy. For recent years, the male populations of Chile, Colombia, Costa Rica, Mexico, Panama, Trinidad and Tobago, Venezuela, and the Union of South Africa (coloured) all show predictions in close accord with reality. One important exception is Guatemala, where predicted death rates at infancy exceed the actual death rate, itself quite high, by about 50%. The reason may be an over-recording of deaths to diarrheal diseases, or gastroenteritis. A review of coding accuracy determined that the number of deaths returned in this category was about three times higher than it should have been among adults in Guatemala City in 1962-64.[22] Predictions of infant mortality are also substantially too high in Taiwan, perhaps because of problems in reporting ages of infants.

Cause of death structures in the non-western nations contained in this analysis are, as a group, remarkably similar to those of western nations at equivalent levels of mortality.[23] The two persistent differences are relatively high death rates from diarrheal diseases in the non-western areas, and low death rates from cardiovascular diseases. The relatively high death rates for diarrheal diseases in non-western areas probably represent a slower improvement in nutritional standards and sanitation, of central importance in the etiology of deaths from these diseases, than in medical technology, principally influential against the infectious diseases caused by specific and identifiable micro-organisms.[24]

Reduced death rates from cardiovascular disease are not important until the later ages, and create only minor disturbances in age-patterns. However, diarrheal deaths are heavily concentrated at the crucial ages under five. Since Southern European countries share abnormally high rates of death from diarrheal diseases, they will typically provide a more accurate model of mortality relations for non-western areas than will other European regions.[25] U. N. Manual IV notes that Mexico, with virtually complete vital registration, has a mortality pattern much closer to the "South" than to the "West".[26] Sullivan confirms the importance of diarrheal disease for age patterns of mortality by tracing changes in the Taiwanese pattern between 1957 and 1968 to a rapid reduction in diarrheal death rates.[27]

Causes of Death and the "African Standard"

Just as the cause-structure of mortality can be used to predict the age pattern, the age pattern implies a special cause of death structure. We illustrate the usefulness of this reversal by reference to Brass' "African standard" mortality schedule.[28] This standard incorporated what were considered characteristic features of tropical African mortality. In particular, the pattern exhibits relatively high death rates between ages 5 and 20 relative to those at ages below 5, and exceedingly high death rates above age 20 relative to those below 20. Brass and Coale cautiously state that "Whether these common features are a characteristic bias in African data or a characteristic feature of African mortality is a matter of conjecture."[29]

One gauge of the authenticity of the "African standard" is the plausibility of the cause of death structure which it implies. A portion of the African standard $p(x)$ function is presented in Table VIII. $p(1)$ (for both sexes) is .880, which corresponds approximately to the $p(1)$ for ASCDR = .0205 in Table II. The typical cause of death structure at an ASCDR = .0205 is determined by computing linear regressions relating $ASCDR_i$ to ASCDR, and substituting ASCDR = .0205 into the resulting equations.[30] Predicted death rates

for males are as follows: Tuberculosis = .00127, Other Infectious and Parasitic = .00142; Respiratory = .00306; Diarrheal and Cert. Dis. of Infancy = .00189; Violence = .00106; Other and Unknown = .01130. The age-curve implied by this combination of causes, when substituted into male equations of Table VI, is also shown in Table VIII. In order to reproduce very closely the "African standard" p(x) function, we can multiply the "average" death rate from tuberculosis by 2.00, from infectious and parasitic diseases by 2.75, and from diarrheal and certain diseases of infancy by .6, and once again apply equations in Table VI. The resulting p(x) column does not differ from the African standard by more than .006 through age 60.

In other words, the African standard implies death rates from tuberculosis and infectious and parasitic diseases which are approximately 2-3 times higher than normal for a population at that approximate level of mortality. In view of the over-riding importance of malaria in tropical Africa, this implication seems quite acceptable.[31] In fact, malaria has an age curve of mortality intermediate between that of tuberculosis and other infectious and parasitic diseases, excluding both malaria and tuberculosis. For example, of these three categories in Thailand, 1964 — the country with highest malarial death rates in World Health Organization statistics of that year — "other infectious and parasitic" has the highest death rate under age 15, malaria between ages 15 and 25, and tuberculosis thereafter.[32] Except at ages 15-25, malaria's rates are always intermediate.

By the same token, unusually high death rates from infectious diseases cannot account for the African standard's exceptionally high death rate at ages 60-80. This feature could be produced by an increment in the "Other and Unknown" death rate (which, in Table VI, has an impact on death rates 6 times greater in this age interval than in any other). On the other hand, it could easily be bogus. There is surely very little solid information upon which to base estimates of death rates for this group in Africa, in light of pervasive mis-reporting of age and relatively small numbers of persons. Whether or not this particular characteristic of

Table VIII. Comparison of Three Survivorship Curves (Probability of Surviving from Birth to Age x)

Age (x)	Cause-of-Death Regressions, Average Structure of Causes Corresponding to ASCDR = .0205. Males[1]	Cause of Death Regressions, Structure of Causes Same as (1) But Death Rate from Tuberculosis Multiplied by 2.00, Infectious and Parasitic 2.75, and Diarrheal and Cert. Dis. of Inf. by .6. Males[1]	African Standard (both sexes)[2]
	(1)	(2)	(3)
0	1.000	1.000	1.000
1	.876	.882	.880
5	.816	.786	.786
20	.779	.715	.713
40	.690	.584	.590
60	.506	.398	.398
80	.127	.109	.076

1. Source: Table VI and text
2. Source: William Brass and Ansley J. Coale, "Methods of Analysis and Estimation: in William Brass, et al., The Demography of Tropical Africa. Princeton University Press. 1968. p.133.

the "African standard" is appropriate, the fundamental point is that cause of death mortality patterns provide a means of testing the plausibility of any particular age pattern of mortality by tying its features more directly to local epidemiologic conditions.

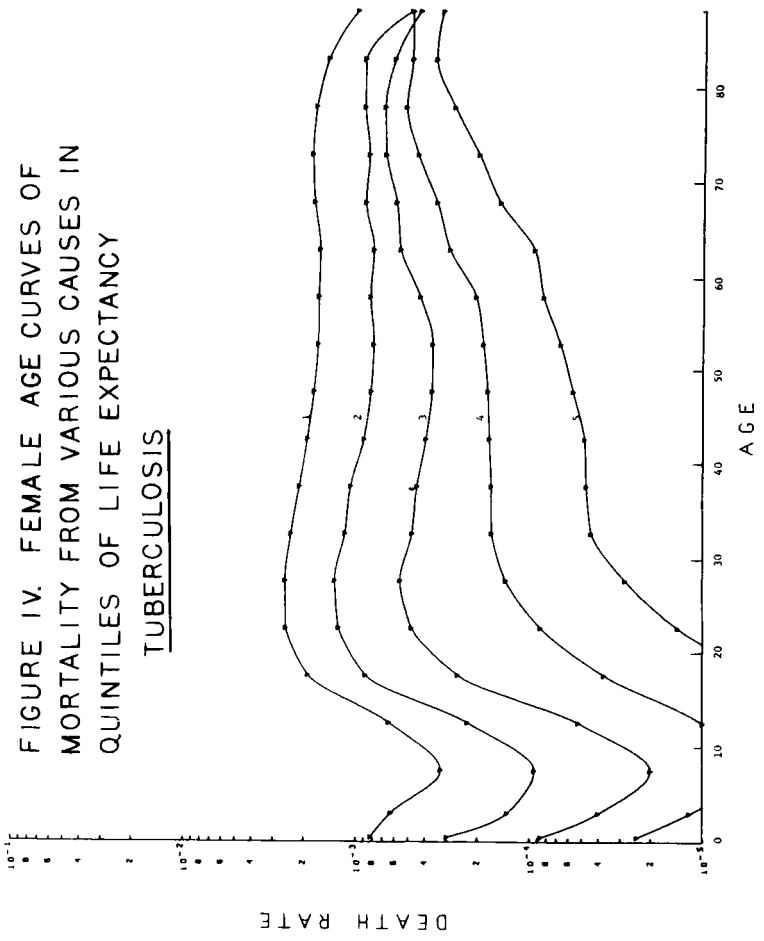
Constructing a Set of Model Mortality Patterns Based on Estimated Cause of Death Structure

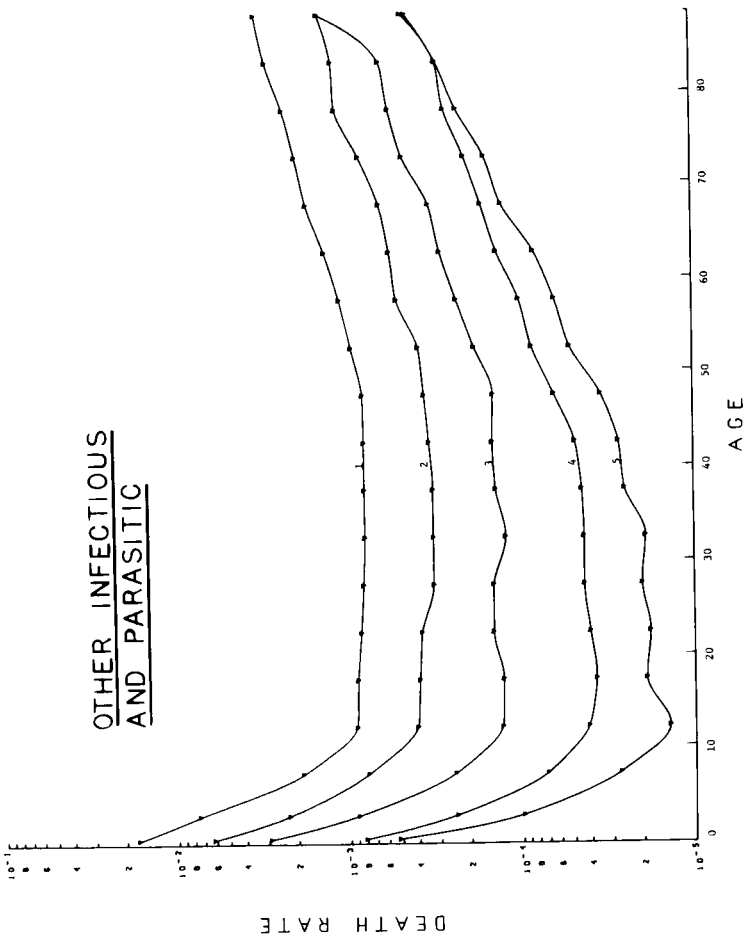
It is uncommon to have better data on causes than ages of death, although John Graunt faced this situation in preparing what is sometimes considered the world's first life table.[33] The obvious way to proceed to a "model" in such a case is to adopt a standard age curve of mortality from each pertinent cause, set its level in accordance with estimated disease intensity, produce the "all cause" curve as a sum of curves of individual causes, and let the aggregate vary proportionately at all ages as mortality level varies. The problem is that, for many causes, there is no one standard curve that can be allowed to vary proportionately; instead, the shape of the age-curve itself changes as mortality level changes. These variations are demonstrated in Figure IV for the seven causes distinguished for females. Equi-proportionate variation should produce, on semi-log paper, parallel age curves. Tuberculosis, respiratory disease, and "other and unknown" have age curves which clearly converge at higher ages (reflected in Table VI as high constant terms in the regression equations for ages 60-80). Age curves from violence cross, with the ranking of the 5 curves exactly reversed at the extremes of age, although the curves themselves are close enough together that parallelism is not a bad approximation.

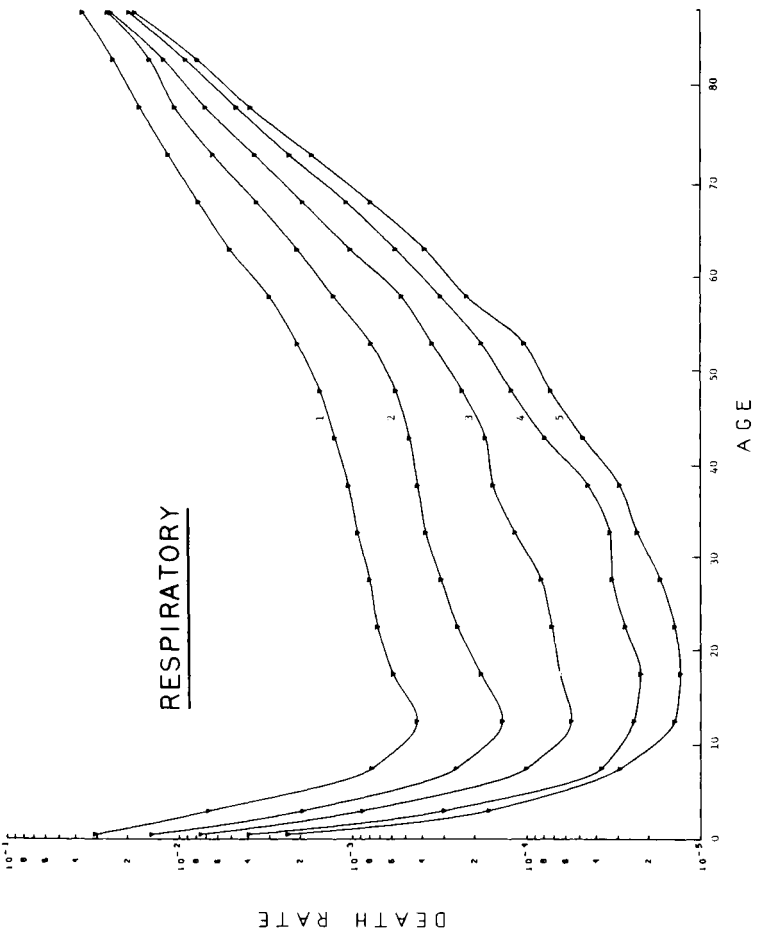
In the face of such level/slope relationships, one is forced to construct model age curves for each cause that vary with the level of that cause. In using the tables, the tedious process of interpolating at various ages between levels of mortality from all causes combined would have to be repeated for each cause of death. It is unlikely that even the most diligent demographer would find much use for model life tables developed in this fashion.

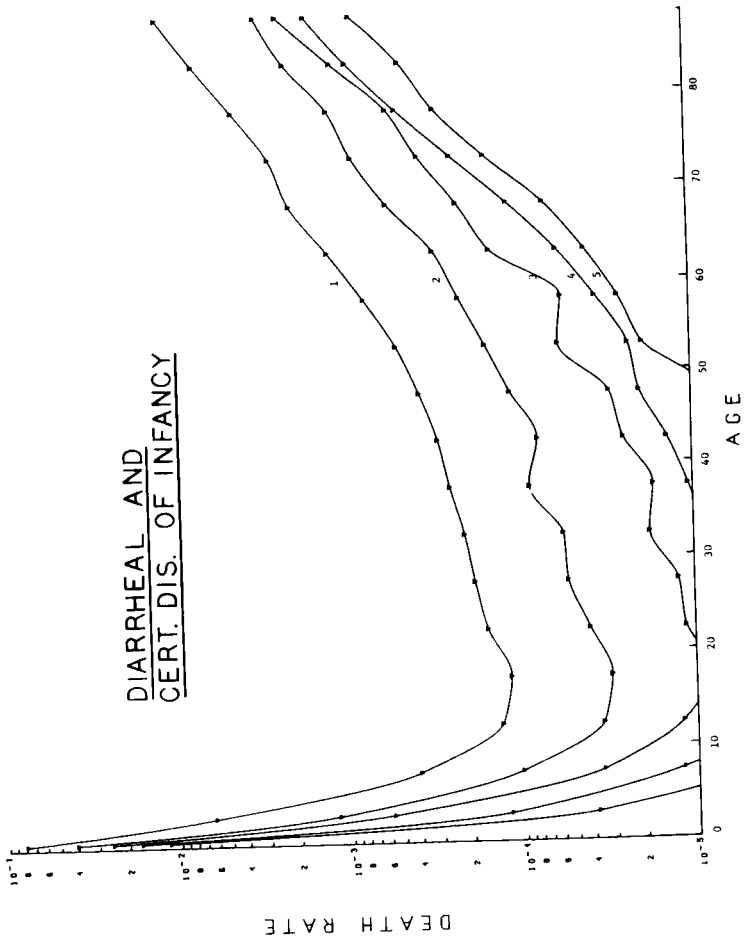
FIGURE IV. FEMALE AGE CURVES OF MORTALITY FROM VARIOUS CAUSES IN QUINTILES OF LIFE EXPECTANCY

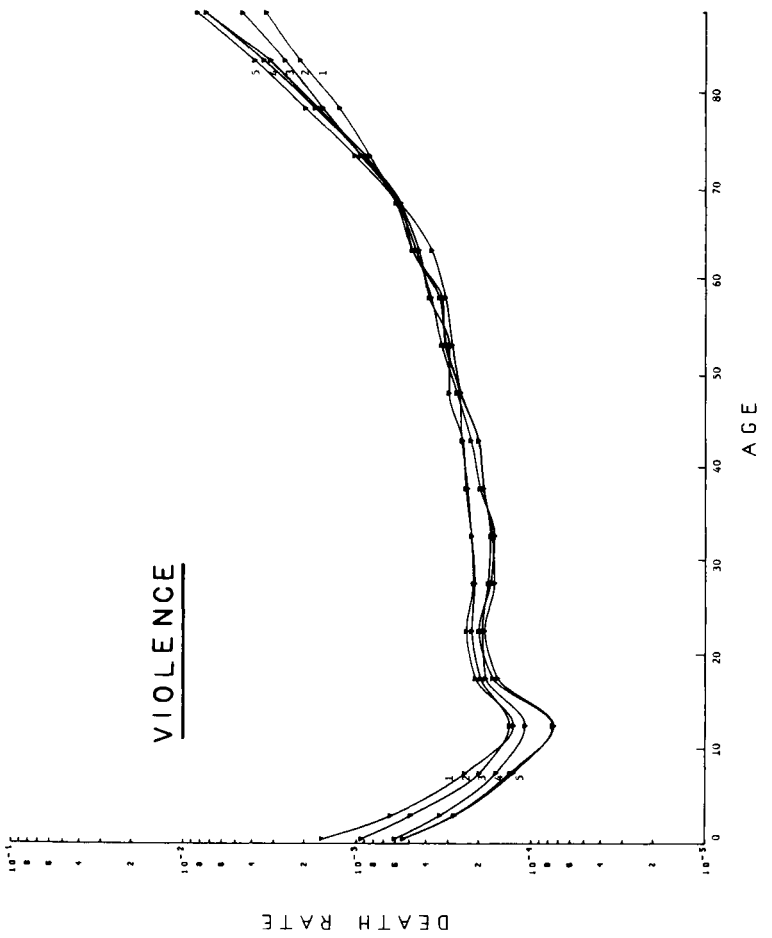
TUBERCULOSIS

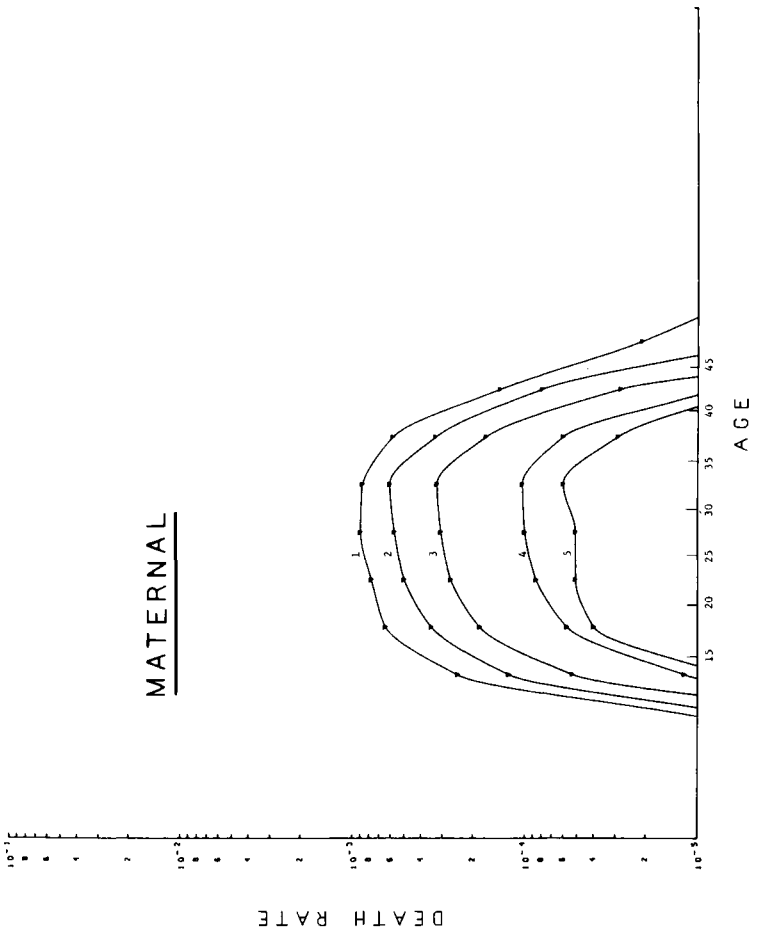


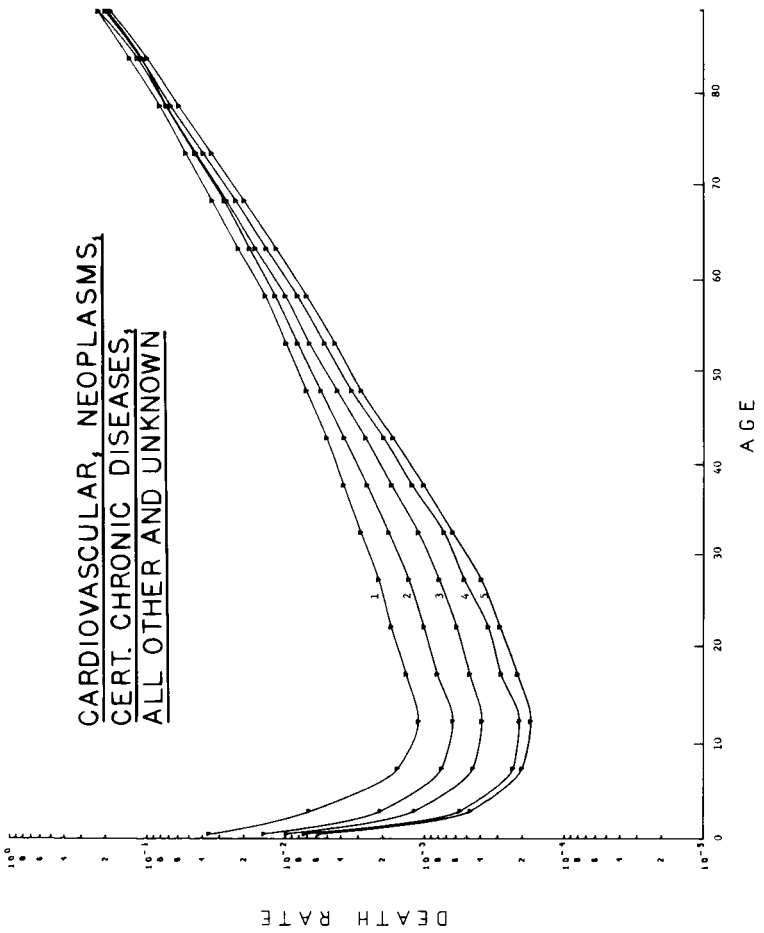

OTHER INFECTIOUS
AND PARASITIC
DEATH RATE
AGE
1
2
3
4
5


RESPIRATORY
DEATH RATE
AGE
1
2
3
4
5
0
10
20
30
40
50
60
70
80


DIARRHEAL AND
CERT. DIS. OF INFANCY
DEATH RATE
AGE
1
2
3
4
5
0
10
20
30
40
50
60
70
80


VIOLENCE
DEATH RATE
AGE
0
10
20
30
40
50
60
70
80
1
2
3
4
5


MATERNAL
DEATH RATE
AGE
1
2
3
4
5
15
20
25
30
35
40
45


CARDIOVASCULAR, NEOPLASMS,
CERT. CHRONIC DISEASES,
ALL OTHER AND UNKNOWN
1
2
3
4
5
DEATH RATE
AGE
0
10
20
30
40
50
60
70
80

An alternative is to exploit regression equations of the sort provided in Table VI. The most sensible way of proceeding is to estimate the level of overall mortality for a population (ASCDR), estimate the average structure of causes of death at that level by consulting the companion paper by Preston and Nelson,[34] modify that structure in accordance with distinctive features of local conditions, and substitute the modified cause-of-death rates into equations in Table VI. One obtains thereby a series of $\log_n \hat{p}_x$:

$$\log_n \hat{p}_x = A_x + \sum_i B_x^i \, \hat{ASCDR}^i \, .$$

Mortality level can now be allowed to vary by multiplying all $ASCDR^i$ by a constant factor, K, which is equivalent to holding the cause structure of mortality constant as its level varies. (This condition is likely to apply only within a limited range, and if mortality level proves to fall outside of that range, the process must be started all over again.)

Once the series of $\log_n p_x$'s is determined for the value of $K = 1$, all other series are readily calculated by simply multiplying the summation term by K. Because of the A_x terms, large at high ages, the series will converge at these ages. But the prolonged sequence of steps still required to fashion such a model mortality pattern discourages their further elaboration. Probably their major function is to expose the epidemiologic foundations and implications of existing models and guide the user in choosing among them.

FOOTNOTES

1. The earliest set of model life tables appears in United Nations, Department of Social Affairs, Population Branch, Age and Sex Patterns of Mortality. Population Study No. 22. New York: 1955. Additional models have been provided in K. R. Gabriel and Ilana Ronen, "Estimates of Mortality from Infant Mortality Rates", Population Studies. 1958. Vol. 12, pp. 164-69; Ansley J. Coale and Paul Demeny, Regional

Model Life Tables and Stable Populations. Princeton University Press. Princeton: 1966; Sully Ledermann, Nouvelles Tables-Types de mortalité. Institut national d'études démographiques. Travaux et Documents. Cahier no. 53. 1969; Norman Carrier and John Hobcraft, Demographic Estimation for Developing Societies. Population Investigation Committee, London School of Economics. London: 1971.

2. United Nations, Department of Economic and Social Affairs, Methods of Estimating Basic Demographic Measures from Incomplete Data. Manual IV. Population Study No. 42. New York: 1967. Chapter I.

3. Ansley J. Coale and Paul Demeny, Op. cit.

4. Demeny and Shorter present evidence that even the model mortality pattern embodying highest mortality below age 5 — the "South" pattern of Coale and Demeny — severely underestimates the child mortality rates in Turkey at a given level of adult mortality. Paul Demeny and Frederic C. Shorter, "Estimating Turkish Mortality, Fertility, and Age Structures: Application of Some New Techniques." Publication No. 218. Faculty of Economics, University of Istanbul: 1968. William Brass is forced to develop a hypothetical "African standard" model pattern for use in tropical Africa, since none of those based upon reliable data embody sufficiently high mortality beyond age 20 . William Brass and Ansley J. Coale, "Methods of Analysis and Estimation", in William Brass, et al., The Demography of Tropical Africa. Princeton University Press. Princeton: 1968. pp. 132-33.

5. To be published in Samuel H. Preston, Nathan Keyfitz, and Robert Schoen, in collaboration with Verne E. Nelson, Causes of Death: Life Tables for National Populations. Seminar Press, New York: 1972.

6. The excluded populations are: Chile (1909), Ceylon (1960), El Salvador (1950), France (1926, 1931, 1936), Greece (1928), Japan (1899), Mauritius (1960, 1964), Philippines (1964),

Portugal (1920), Taiwan (1920), Union of South Afriça (Asians), 1941, 1951, and 1960. In addition, West Berlin (1960, 1964) was excluded because of its sub-national status, and the United States (1920, 1950) was excluded in favor of representing white and nonwhite for these dates separately. The remaining 163 populations are: AUSTRALIA: 1911, 1921, 1933, 1940, 1951, 1960, 1964; AUSTRIA: 1961, 1964; BELGIUM: 1960, 1964; BULGARIA: 1964; CANADA: 1921, 1931, 1941, 1951, 1960, 1964; CHILE: 1920, 1930, 1940, 1950, 1959, 1964; COLOMBIA: 1960, 1964; COSTA RICA: 1960, 1964; CZECHOSLOVAKIA: 1934, 1960, 1964; DENMARK: 1921, 1930, 1940, 1960, 1964; ENGLAND AND WALES 1861, 1871, 1881, 1891, 1901, 1911, 1921, 1931, 1940, 1951, 1960, 1964; FINLAND: 1951, 1960, 1964; FRANCE: 1951, 1960, 1964; GERMANY(F. R.): 1960, 1964; GREECE: 1960, 1964; GUATEMALA: 1961, 1964; HONG KONG: 1961, 1964; HUNGARY: 1960, 1964; ICELAND: 1964; IRELAND: 1951, 1961; ISRAEL (JEWISH POP): 1951, 1960, 1964; ITALY: 1881, 1891, 1901, 1910, 1921, 1931, 1960, 1964; JAPAN: 1908, 1940, 1951, 1960, 1964; MALTA AND GOZO: 1964; MEXICO: 1960, 1964; NETHERLANDS: 1931, 1940, 1950, 1960, 1964; NEW ZEALAND: 1881, 1891, 1901, 1911, 1921, 1926, 1936, 1945, 1951, 1964; NORTHERN IRELAND: 1960, 1964; NORWAY: 1910, 1920, 1930, 1946, 1951, 1960, 1964; PANAMA: 1960, 1964; POLAND: 1960, 1964; PORTUGAL: 1930, 1940, 1960, 1964; PUERTO RICO: 1960, 1964; SCOTLAND: 1951, 1960, 1964; SOUTH AFRICA (COLOURED AND WHITE): 1941, 1951, 1960; SPAIN: 1930, 1940, 1960; SWEDEN: 1911, 1920, 1930, 1940, 1951, 1960, 1964; SWITZERLAND: 1930, 1940, 1951, 1960, 1964; TAIWAN: 1930, 1936, 1960, 1964; TRINIDAD AND TOBAGO: 1963; U.S.A.: 1900, 1910, 1930, 1940, 1960, 1964; U.S.A. (WHITE AND NONWHITE): 1920, 1950; VENEZUELA: 1960, 1964; YUGOSLAVIA: 1961, 1964.

7. World Health Organization, Manual of the International Statistical Classification of Disease, Injuries, and Causes of Death. Volume I. Geneva: 1957.

8. The age "curve" of mortality from certain diseases of infancy is scarcely of interest; the death rates from

automobile accidents are typically the lowest of any cause (excluding certain diseases of infancy) and would, if included in Figure I, require a change in scale.

9. Sully Ledermann, Op. cit., p. 20.

10. The weights are provided by the Coale and Demeny "West" female stable population with $e_0^0 = 65.00$ and $r = .01$. Op. cit., p. 62.

11. Sully Lederman, Op. cit., Section 2-10.

12. The United Nations tables are based upon relationships of the following form:

$$q_x = a_x + b_x \cdot q_{x*} + c_x \cdot (q_{x*})^2 \quad ,$$

while Coale and Demeny combine two different functional forms in their final product:

$$q_x = a_x + b_x \cdot e_{10}^0$$

$$\log q_x = a'_x + b'_x \cdot e_{10}^0 \quad .$$

13. For comparison, the correlations were also computed between $\log(l_{x+n}/l_x)$ and e_0^0. At the six ages listed in Table I, they were the following:

Females: .942, .929, .950, .956, .926 .900
Males: .958, .929, .935, .948, .904, .720

In 7 of 12 cases, these are higher than the corresponding coefficients in Table I. The level of performance of the two indices is approximately equal; the non-linearity of life expectancy is counter-balanced by the information which it provides on the age structure of death, information not present in the age-standardized crude death rate.

14. Op. cit., p. 42.

15. The reader may suspect that, for "certain diseases of early infancy", age and cause of death are indecomposable. However, this cause of death represents an average of only 37.0% of all infant deaths for males and 35.0% for females. Consequently, it need not be a significant contributor to age patterns of mortality. In fact the death rate from respiratory disease has a higher zero-order correlation with the infant mortality rate than does "certain diseases of early infancy", as will be demonstrated below.

16. $$-\log {}_{n}p_{x} = \int_{x}^{x+n} \mu(t)dt = \bar{\mu}(t) \cdot n \ ,$$

where $\bar{\mu}(t)$ is the mean value of the force of mortality function in the interval x to $x + n$. Therefore, the age sequence of coefficients of $\log {}_{n}p_{x}$ for a cause must be divided by n to yield the standard age-curve of mortality from that cause.

17. Ansley J. Coale and Paul Demeny, Op. cit., p. 15.

18. Louis J. Verhoestraete and Ruth R. Puffer, "Diarrheal Disease with Special Reference to the Americas" Bulletin of the World Health Organization. Vol. 19. 1958. p. 27.

19. Coale and Demeny, Op. cit., p. 14.

20. Ibid., p. 14.

21. From Preston, Keyfitz, and Schoen, Op. cit.

22. Ruth Rice Puffer and G. Wynne Griffith, Patterns of Urban Mortality. Pan American Health Organization. Publication No. 151. Washington: 1967, pp. 238, 327.

23. Documentation is provided by Samuel H. Preston and

Verne E. Nelson, "Structure and Change in Causes of Death An International Summary", Unpublished, University of Washington, 1972.

24. The slow decline in mortality from diarrheal diseases, in the face of rapid extension of public health programs, has been noted by several authors, among them Helen Moore, et al., "Diarrheal Disease Studies in Costa Rica: 1. Plan and Method of Investigation". American Journal of Public Health, Vol. 56(2). 1966. pp. 376-86. See also Preston and Nelson, Op. cit.

25. Preston and Nelson, Op. cit.

26. Op. cit., p. 72.

27. Jeremiah M. Sullivan, "Shifts in the Age Pattern of Mortality in Taiwan During a Period of Rapid Mortality Decline". Population Studies Center, University of Michigan, 1972.

28. Brass and Coale, Op. cit., p. 132-3.

29. Ibid., p. 133.

30. Regression coefficients are presented in Preston and Nelson, Op. cit.

31. See J. Bonte and A. Kühner, "Recent Levels, Characteristics and Trends of Mortality in Africa" United Nations Economic and Social Council. Economic Commission for Africa. African Population Conference. Accra, Ghana. Dec. 9-18, 1971.

32. World Health Organization, World Health Statistics Annual. 1964. Geneva. pp. 126-7, 260-61. While malaria is nominally a member of our "other infectious and parasitic" category, its death rates are typically too low to have influenced the characteristics of this aggregate.

33. David Glass, "Graunt's Life Table" Journal of the Institute of Actuaries. LXXVI. 1950. pp. 60-64.

34. Op. cit.

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