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Heterogeneity in the Strehler-Mildvan General Theory of Mortality and Aging

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Abstract This study examines and further develops the classic Strehler-Mildvan (SM) general theory of mortality and aging. Three predictions from the SM theory are tested by examining the age dependence of mortality patterns for 42 countries (including developed and developing countries) over the period 1955–2003. By applying finite mixture regression models, principal component analysis, and random-effects panel regression models, we find that (1) the negative correlation between the initial adulthood mortality rate and the rate of increase in mortality with age derived in the SM theory exists but is not constant; (2) within the SM framework, the implied age of expected zero vitality (expected maximum survival age) also is variable over time; (3) longevity trajectories are not homogeneous among the countries; (4) Central American and Southeast Asian countries have higher expected age of zero vitality than other countries in spite of relatively disadvantageous national ecological systems; (5) within the group of Central American and Southeast Asian countries, a more disadvantageous national ecological system is associated with a higher expected age of zero vitality; and (6) larger agricultural and food productivities, higher labor participation rates, higher percentages of population living in urban areas, and larger GDP per capita and GDP per unit of energy use are important beneficial national ecological system factors that can promote survival. These findings indicate that the SM theory needs to be generalized to incorporate heterogeneity among human populations.

Keywords Strehler-Mildvan theory · Ecological system · Population heterogeneity · Age of expected zero vitality

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Introduction

About 50 years ago, Strehler and Mildvan (SM; 1960) published a general theory of mortality and aging in *Science* that provides a biological and physical science underpinning for the Gompertz (1825) law of exponential increases in adult mortality long used by demographers. SM theory presents an elegant biodemographic model of the age dependence of human mortality that can be used, among other things, to rank countries by the extent to which their social and physical environments promote survival. However, the empirical estimates used by Strehler and Mildvan to test their theory were based on a cross-sectional sample of a limited number of countries (32 countries) with data from the mid-1950s.

With the passing of more than four decades of contemporary history and the associated expanded database, it is possible to further examine and develop the SM theory by analyzing data from a larger number of countries over a longer period. It is also possible to study and identify those aspects of social, economic, and physical environments that promote longevity. These are the objectives of the present study. In the context of aging societies, this research contributes to the scientific understanding of aging patterns and causes of different aging patterns across countries and time periods. In addition, this research has implications for public policy. Understanding the relationships between socioeconomic and other environmental factors and aging is essential for developing better economic, environmental, and social policies to enhance population health.

The Strehler-Mildvan Theory and Subsequent Studies

Strehler and Mildvan's (1960) general theory of mortality and aging synthesized the following: (1) *Gompertz's Law of Mortality*—the exponential increase of adult human mortality with age t, $R_t = R_0 e^{\alpha t}$; (2) a *linear decline of vitality index* $V_t = V_0(1-Bt)$ with increasing age, where vitality V_t is the capacity of an individual organism to stay alive at age t, and the *attrition coefficient* B is the fractional loss each year of original vitality V_0; and (3) *parameters measuring environmental stresses*—a measure of the frequency of environmental variations K and a measure of their average magnitude ${}_{\varepsilon}D$.

This theory and the empirical assessment thereof by Strehler and Mildvan (1960) led to three important predictions and properties: First, the intercept $\ln R_0$ and the slope α of the logarithm of the Gompertz mortality curve are negatively correlated, the so-called *SM correlation* (i.e., $\ln R_0 = -\frac{1}{B}\alpha + \ln K$); in other words, if population 1 has a lower initial mortality rate than population 2, it should have a higher rate of increase in mortality. Strehler and Mildvan predicted that this correlation should be applicable to any human mortality situation regardless of living standards, health care, and other factors.

Second, the fractional loss of vitality coefficient $B = b + f(D)$ is a function of both a normal aging component b, and the impact $f(D)$ on B of environmental factors as measured by the summary measure of relative environmental deleteriousness, D. Based on mortality data from 32 countries from the mid-1950s, Strehler and Mildvan (1960:16–17) concluded that B "appears to be nearly constant regardless of the

environment.…It thus appears that B is dominated by [the normal aging process] b, or in other words that the rate of loss of vitality during the aging process is largely independent of the environment."

Third, the inverse of the fractional loss of vitality (i.e., $1/B$) constitutes the SM estimate of maximum lifetime attainable—life span—in a homogeneous population to which the population average parameters are applicable.

The SM theory is deterministic in the tradition of classic life table and stable population theory in demography. For empirical applications, it thus can be regarded as providing expected values around which observed human populations are distributed. In any case, all parameters in the theory are population-level estimators or population averages and thus do not take into account individual-level variation within a population. This is why it is important to reexamine SM theory with longitudinal, over-time data on a larger set of countries and to explore possible heterogeneity in these parameters and correlation patterns among them.

This theory has stimulated many subsequent studies, some of which have suggested that the SM correlation pattern was stable for adult mortality patterns from 1900 to 1986 in the United States (e.g., Riggs 1992) and other developed countries, including overall mortality trends in industrialized countries (e.g., Prieto et al. 1996; Riggs and Millecchia 1992), and for such specific causes of death as stroke (e.g., Riggs and Myers 1994), colon cancer (e.g., Riggs 1993), malignant brain tumors (e.g., Riggs 1994), and multiple myeloma mortality (e.g., Riggs 1995). But other studies have found that either the period SM correlation or the cohort SM correlation is not stable in France, Japan, Sweden, and the United States and have suggested further extensions of the SM theory (Yashin et al. 2000, 2001, 2002a,b).

The debate over limits to human life span is even more intense and conflicting. Different from the SM predictions (i.e., the Gompertz Law and limited life span), in some studies of humans (Horiuchi and Wilmoth 1998; Kannisto et al. 1994; Lynch and Brown 2001), medflies, Mexican fruit flies, Drosophila, bruchid beetle (Callosobruchus *maculates*), nematode worms, and even automobiles (Vaupel 1997), it has been found that mortality tends not to accelerate at the oldest ages, but rather to decelerate. Moreover, gains of survival over age 80 and even over age 90 began accelerating after 1960 (Wilmoth 1997) and continued to increase for several countries through 2004 (Rau et al. 2008). The population heterogeneity hypothesis argues that the deceleration of mortality at the oldest ages is a statistical effect of compositional change that results from the higher early-life mortality of the frail decreasing the rate of increase in the age trajectory of mortality (Kowald and Kirkwood 1993). But others have argued that it is implausible that all the observed deceleration of mortality at older ages is an artifact of heterogeneity. Rather, "some of the observed deceleration is due to behavioral and physiological changes that occur with age and that are associated either with declines in reproductive activity or with repair mechanisms that compensate for damage at younger ages" (Vaupel 1997: 26). In addition, the maximum age at death for some national populations has risen and continues to rise in a steady, almost linear fashion; if it approached a fixed upper bound, the trend should have slowed down or decelerated, but such a slowing is not evident (Wilmoth and Lundstrom 1996).

Rather than entering into the intense debate over whether the maximum human life span is fixed or malleable, we instead use the term *age of expected zero vitality*

to refer to the SM expected (population average) maximum survival age. Since the SM model is deterministic, the implied age of zero vitality, estimated by $1/B$, should be regarded as an expected value for a specific human population. In empirical applications to human populations, however, there will be stochastic variability around this age that should be taken into account. Within the context of the SM model, the reason for this is that even at an age of zero vitality, individuals must be confronted with challenges to molecular bonds from the environment that arrive according to a Maxwell-Boltzmann (exponential) frequency distribution—challenges of sufficient severity to destroy the molecular bonds and cause death. Because such challenges are variable in their arrival times, the SM implied expected age of zero vitality for the population as a whole does not imply that it is impossible for an individual in the population to live beyond that age.

Ecological Demography and Relative Environmental Deleteriousness

The SM model uses the overall environmental deleteriousness parameter D to rank countries by the extent to which their social and physical environments promote survival, but it does not specify what national sociodemographic and economic characteristics account for levels and variations in D. The "environment" construct used in the SM model is more like the concept of an "ecological system" used in ecological demography, which is very broad and goes beyond any specific characteristics of the "environment." *Ecological demography* studies how the ecological system affects demographic processes. One classical model in ecological demography, termed the POET model (Duncan 1959), identifies four elements of the ecological system: *population, organization, environment,* and *technology*—where the breadth of "environment" is smaller than the one used in the SM model.[1]

Organization is the fundamental element of the ecological system. The notion of organization does not refer just to the division of labor, but is multifaceted (Poston and Frisbie 2005:608). A major dimension of sustenance organization is *sustenance differentiation*—that is, the "arrangement of differentiated parts suited to the performance of a given function or set of functions" (Hawley 1950:178). Sustenance differentiation is a major component of the division of labor. A second dimension is *functional interdependence*, which can form the other side of the division of labor by combining with sustenance differentiation (Gibbs and Poston 1975). A third dimension is the *volume of sustenance* produced by the population—that is, "the degree of productivity of the particular configuration of sustenance activities" (Poston and Frisbie 2005:609), which may include agricultural productivity, retail services productivity, wholesale services productivity, personal services productivity, and so on. A fourth dimension is the extent of *efficiency of the sustenance organization*, which can be measured by the ratio of the amount of sustenance produced to the amount of inputs needed in the production process. A bigger ratio indicates higher efficiency. A final dimension is "the degree to which population members are engaged in sustenance-related pursuits" (Poston and Frisbie 2005:610).

[1] Henceforth, we use *ecological system* to refer to what Strehler and Mildvan (1960) termed the "environment"; we use the term *environment* for one of the four elements in ecological demography.

Related measures of this dimension can be the labor participation rate in the population and the extent to which the social status is ascribed or achieved.

Population structure also has very significant consequences on demographic processes (i.e., migration, fertility, and mortality). In the POET model, population structure refers not only to the age, race, and sex composition of population but also to the spatial location of population.

Among the four basic ecological elements, *technology*, as the primary driving force for social change, has the most critical role regarding the adaptation and growth of human populations because it sets the boundaries for possible social and economic development (Poston and Frisbie 2005:611). Technology has three dimensions: "*material features* (tools, capital equipment, machines); *information* (knowledge, techniques, scientific discovery); and *energy*" (Poston and Frisbie 2005:612). Gross domestic product (GDP) per capita often is taken as an overall index of the hardware, capital equipment, and information dimension of technology of a society.

The *environment* concept in ecological demography does not comprise everything external to the phenomenon under investigation, "but only those externalities that, by virtue of the limits they set on the acquisition of sustenance, affect the life chances of an organized population with a given technological repertoire" (Poston and Frisbie 2005:615). Thus, the *environment* concept in ecological demography is a set of conditions serving as limiting (or enabling) resources for the adaption and growth of populations, whose breadth depends on "the technological devices and modes of organization that prevail in a given population" (Schnore 1958:628). Based on this definition, social and economic activities (e.g., unemployment rates, industry structure, levels of education and income) are not part of the environment. Rather, the environment concept in ecological demography has two broad and distinct dimensions: the physical and the social. The *physical environment* refers to climate, natural resources, topography, and so on. The *social environment* includes such things as size, proximity to the nearest metropolitan areas, and government influences.

Research Questions

The foregoing, briefly summarized concepts of ecological demography will be used to generalize SM theory. To motivate this, we commence with an empirical evaluation of SM theory by examining the age dependence of male mortality patterns for 42 countries[2] (including developed and developing countries) over a 50-year period. Specifically, this research addresses several questions derived from the three predictions of the SM theory mentioned above:

1. Are the initial mortality rate $\ln R_0$ and rate of increase in mortality α negatively correlated across the 42 countries and the period 1955–2003?

[2] These 42 countries include two regions: Hong Kong and Puerto Rico. For the sake of simplicity, we will refer to them as countries in this article.

2. Is the fractional loss of vitality B constant—that is, is it dominated by normal aging process b and independent of the ecological system $f(D)$?
3. Correspondingly, does a constant, or relatively constant, age of expected zero vitality ($1/B$) exist? Or do the SM estimated ages of expected zero vitality change over time?
4. If the estimated ages of expected zero vitality are not constant over time, do all 42 countries have the same trajectory of changes?
5. If there is hidden heterogeneity in the form of detectable differences of trajectories of change over time in ages of expected zero vitality, what accounts for it? Can any measure in the POET model in ecological demography account for the variations in deleteriousness D?[3]

Methods

Data

The country-period-age-specific male mortality data set for 42 countries analyzed herein was compiled from the *Demographic Yearbook* 1955–2003, published annually by the Department of Economics and Social Affairs of the United Nations.[4] In order to directly test the SM theory, we organized this data set into a pooled country-period data design. Each country-period case has the following five estimated SM parameters: the logarithm of initial mortality rate $\ln R_0$, the slope α of the logarithm of the Gompertz mortality curve, attrition coefficient B, age of expected zero vitality $1/B$, and the relative deleteriousness of national ecological system D.[5]

National ecological system data were complied from World Development Indicators (WDI), published by the World Bank Group. We collected several indicators of the elements of the ecological system as summarized earlier: the *physical environment/climate* of a country as measured by its regional location; *organization* as measured by the share of a country's biological water pollutants accounted for by the food industry, its total labor force participation rate, and male labor force participation rate; *population structure* as measured by a country's population density (people per square km), its percentage of population in the largest city, and percentage of population in urban agglomerations greater than 1 million; and *technology* as measured by a country's gross domestic product per capita (in

[3] It must be emphasized that the objective of this paper is *not* to conduct a systematic test of the POET model in ecological demography. Rather, we study whether the POET model can be used to enrich the concept of the environment in SM theory and to account for hidden heterogeneity and variations in the environmental deleteriousness measure D.

[4] Following Strehler and Mildvan (1960), our primary analyses focus on five-year male mortality rates beginning at age 30, 30–34, 35–39, . . ., 80–84, and then the open-ended interval ages 85+. We similarly analyzed five-year female mortality rates from age 40 to 85+. The initial age is higher for females because of the relatively high risk of maternal mortality in the childbearing ages in developing countries. This can confound estimates of the parameters of the Gompertz function. For the most part, findings are similar for female mortality rates as for male rates. Any differences are noted below.

[5] Formulas for the calculation of these parameters are given in Strehler and Mildvan (1960) and will not be repeated here.

constant 2000 U.S. dollars), and its GDP per unit of energy use (constant 2000 purchasing power parity [PPP] dollars per kg of oil equivalent).

Analytic Methods

We examine the five research questions stated earlier by using descriptive analyses and estimates of finite-mixture and random-effects regression models. The descriptive analyses are used to address the first three questions.

The finite-mixture regression model is used to examine the fourth question. The finite-mixture regression model has come to be known as the *semi-parametric group-based trajectory model* or the *latent class trajectory model* (Jones et al. 2001; Land et al. 1996; Nagin 1999; Nagin and Land 1993). Differing from the hierarchical model (Bryk and Raudenbush 1987, 1992; Goldstein 1995) and the latent growth curve model (McArdle and Epstein 1987; Meredith and Tisak 1990; Muthen 1989; Willett and Sayer 1994), which are based on continuous multivariate density functions to calibrate the variation in the average developmental trajectory within the population, the finite-mixture model uses a multinomial mixture modeling strategy and is designed to identify relatively homogeneous clusters of trajectories of development or change over time in the presence of repeated observations on analytic units, and thus temporal interdependence (Jones et al. 2001; Nagin 1999).

Since the SM estimate of age of expected zero vitality ($1/B$) is a continuous variable, we used the censored normal (CNORM) model version of the finite-mixture model. We estimated this model by application of the SAS TRAJ package (Jones et al. 2001), first to identify latent trajectories of changes in longevity patterns among these countries across the latter half of the twentieth century, and then to identify risk factors that are predictive of membership in the different trajectories.

We conducted principal component analysis and estimated random-effects panel regression models to address the fifth question—that is, to ascertain what, if any, national organizational, population, and technological factors account for variation in the overall ecological system deleteriousness parameter D. To explore some possible national-level covariates that might account for cross-national and temporal variation in D, in addition to *year*, we study the influences of a limited number of regressors that were identified from the ecological demography theory and cited above. Before conducting a random-effects panel regression analysis, we applied principal component analysis to these eight variables (excluding region) to minimize collinearity problems, simplify the dimensionality of the regressor space, and thus reduce the likelihood of partialling fallacies (i.e., the allocation of all explained variance to one of two regressors, both of which are more highly correlated with each other than with the outcome variable; see Land et al. 1990).

Random-effects panel regression models generally are more statistically efficient than pooled OLS or fixed-effect models (Wooldridge 2002). In random-effects panel regression models, the cross-sectional error terms c_i are assumed independent of the longitudinal error terms u_{it} and the values of the explanatory variables/regressors X_{it}, which are also independent of each other for all i and t. To test the assumption of orthogonality of the random effects with respect to the regressors, we applied the Hausman test for comparisons of estimates of random- and fixed-effects panel

regression models (Wooldridge 2002:288–291). The random-effects panel model is specified by a serial of equations:

$$D_{it} = \beta_0 + \beta_1 Year_t + \beta_2 X2_{it} + \beta_3 X3_{it} + \beta_4 X4_{it} + \beta_5 X5_{it} + c_i + u_{it}$$

where c_i is unobserved in all periods but constant over time, and u_{it} is a time-varying idiosyncratic error. Note, specifically, that the c_i are included in order to account for unobserved properties of units, countries in the present case, which are constant over time and thus may cause temporal interdependence of repeated observations for the same unit over time. The composite error is defined as $v_{it} = c_i + u_{it}$.

Results

We begin our discussion of the results by addressing each of our five research questions in turn.

1. Are $\ln R_0$ and α negatively correlated? An examination of patterns of change in estimates of the logarithm of the initial mortality rate ($\ln R_0$) and the rate of increase of mortality with age (α) for individual countries for each five-year period from 1955 to 2000 and then 2003 shows that some trend up, others go down, and still others appear to be quadratic, cubic, and even irregular. Four types of temporal trends are illustrated in more detail in Fig. 1: (1) $\ln R_0$ linearly declines and α linearly increases over time (e.g., the Netherlands and most of the countries); (2) $\ln R_0$ and α follow a quadratic and even cubic function (e.g., Denmark, New Zealand, and Switzerland); (3) $\ln R_0$ and α fluctuate irregularly over time (e.g., the United States and Portugal); and (4) both $\ln R_0$ and α decline over time (e.g., Argentina).

   At the aggregate level of all 42 countries for 1955–2003 (N = 462 country-period observations), Fig. 2 clearly indicates that $\ln R_0$ and α are negatively correlated ($r = -.92$). We also calculated the correlation between $\ln R_0$ and α for each country over the 11 time periods and found that it is negative for all 42 countries except Argentina and France.[6] On average, this correlation is −.752 among the other 40 countries, with a range from −.064 to −.993. In brief, we conclude that there is evidence that the SM correlation exists, but it is not constant and shows considerable heterogeneity among countries.
2. Is B constant; that is, is B dominated by b and independent of the ecological system? When the aggregate ($\ln R_0$, α) inverse relationship is decomposed by countries and periods, heterogeneity in estimates of the slope ($-1/B$) also becomes evident. Figure 3 exhibits plots of $\ln R_0$ against α across the years 1955–2003. Four patterns are evident. First, some patterns have a nearly constant slope over the roughly 50-year period (e.g., the Netherlands and Greece). Second, some patterns have typical "hooks" corresponding to recent

[6] While *period-to-period changes* in $\ln R_0$ and α for Argentina and France are negatively correlated across the 11 time periods of the study, the correlation of the period-specific values across the 11 periods is slightly positive for France (.009) and substantially positive for Argentina (.487) because of relatively high $\ln R_0$ values in 1955. Peculiarities in the age structures of these two countries in the immediate post–World War II decades likely account for these differences.

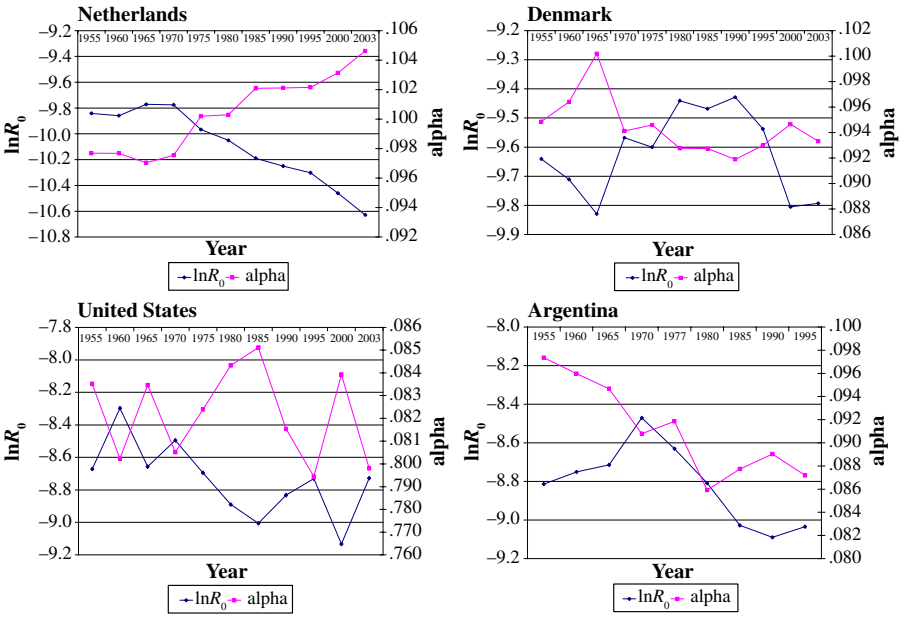

Fig. 1 Trajectories of $\ln R_0$ and α for the Netherlands, Denmark, the United States, and Argentina: 1955–2003

changes in survival (e.g., Japan and Norway). As observed in Yashin et al. (2001), for different countries, these hooks emerge in different places on the ($\ln R_0$, α) plane. Third, some patterns show the negative relationship between $\ln R_0$ and α, but the y-intercept decreases over time; in addition, the temporal trajectories of $\ln R_0$ and α are irregular for some countries (e.g., the United States and France). Fourth, some patterns show very unstable slopes (i.e., $-1/B$) (e.g., Australia and the United Kingdom).

The above analyses all point to the conclusion that the vitality attrition coefficient B is not constant either over years or across countries. This raises the question, Why is B not constant? Is it because the normal aging process parameter b is changing, or is it due to changes in the relative ecological system deleteriousness parameter D? Since it is difficult to estimate changes in the normal aging process b from an aggregate demographic data set, we focus the analysis on how the ecological system D may affect B.

Panel a of Fig. 4 shows that B ranges from 0.007 to 0.011; in other words, estimates of the rate of loss of vitality vary from 0.7% to 1.1% per year of age. Panel a also suggests that B is not independent of relative ecological system deleteriousness D.[7] That is, these two variables have a linear relationship. But

[7] Strehler and Mildvan (1960:18) noted that in order to compare countries with respect to the relative deleteriousness of their ecological systems, a "base" country must be chosen with its D parameter set to a specific number—say, 100. They recommended using the country with the most favorable national ecological system for this base, and then measuring the deleteriousness of the other countries relative to this base. For the data in our analysis, as plotted in Fig. 4, Malta in 1985 has the least deleterious ecological system and thus it is used as the base, as indicated on the horizontal axes in Fig. 4.

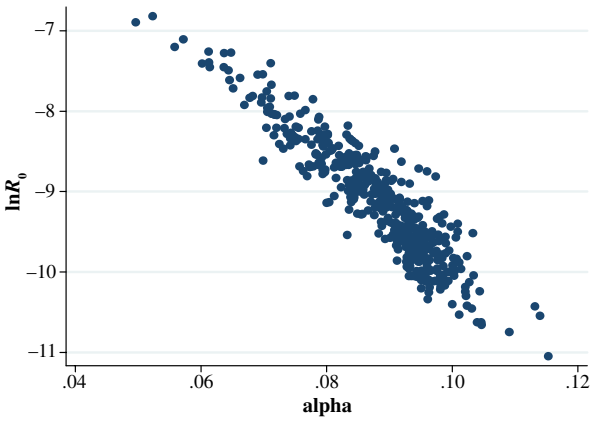

Fig. 2 The inverse relationship between $\ln R_0$ and α for 42 countries, 1955–2003

when a national ecological system becomes worse (the D value increases further from 100), the rate of physiological decline B also, surprisingly, decreases, thus implying higher ages of expected zero vitality. This is consistent with the "survival trade-off" theory (Kirkwood 1990, 1996; Yashin et al. 2002b), which postulates that individuals may "increase adaptive capacity (e.g., the rate of DNA and protein repair)" in response to the magnitude of environmental stresses "at the expense of a reduction in robustness" (Yashin et al. 2002b:213). In the present case, as the value of D increases, B decreases.

However, this is not the only pattern observed. In some developed countries (e.g., the United States, Australia, and Canada), the relationship between D and B is positive; that is, when the ecological system D becomes less deleterious (i.e., the value of D becomes smaller), the fractional loss of vitality becomes smaller. When the full set of countries in Panel a of Fig. 4 is disaggregated into subsets of developed and developing countries,[8] as in Panels b and c, it is evident that the correlation between B and D is negative in the developing countries but is essentially null in the developed countries.

3. Does a constant, or relatively constant, age of expected zero vitality ($1/B$) exist? Figure 5 shows the trajectory of the mean of $1/B$ among all 42 countries for each period from 1955 to 2003. This trajectory of means of $1/B$ is a clear indication that the age of expected zero vitality is not stable but varies over time. In fact, it increases from 1955 to 1960 and then decreases until the early 1970s, after which it continually increases. The general upward trend in the trajectory also suggests that, contrary to the SM prediction of a fixed maximum life span, the age of expected zero vitality is not fixed.
4. Do all countries have the same trajectory of changes of SM estimates of age of expected zero vitality? If not, how do variations in national ecological systems account for the different trajectories? The foregoing analyses point to the inescapable conclusion that longevity trajectories are not homogeneous among these countries. An identification of different clusters of longevity trajectories sheds more light on this pattern. For this, we estimated the semiparametric

[8] The set of 42 countries is divided into developed and developing based on per capital gross domestic product, with developed countries at or above $10,000 per capita.

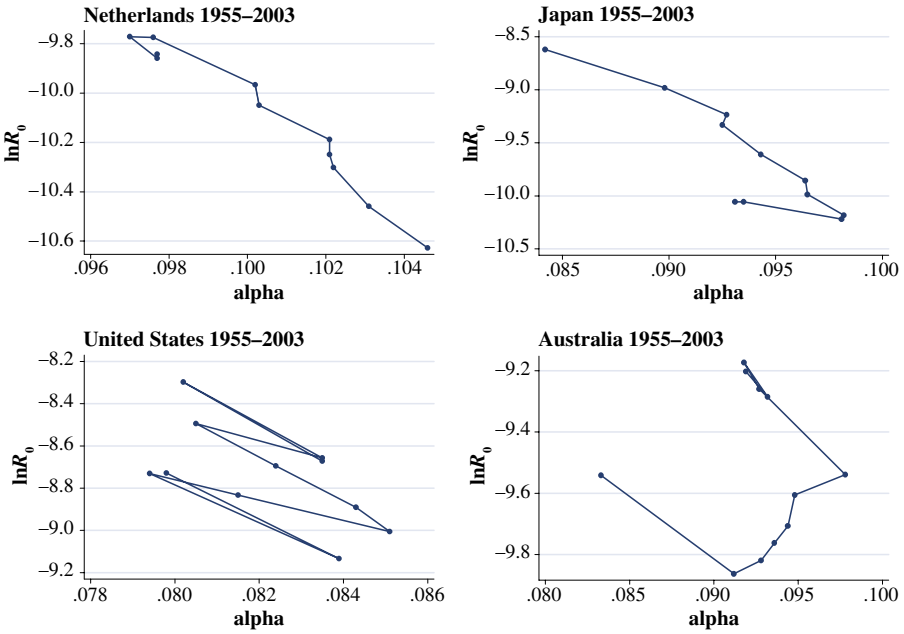

Fig. 3 Plots of $\ln R_0$ against α for the Netherlands, Japan, the United States, and Australia: 1955–2003

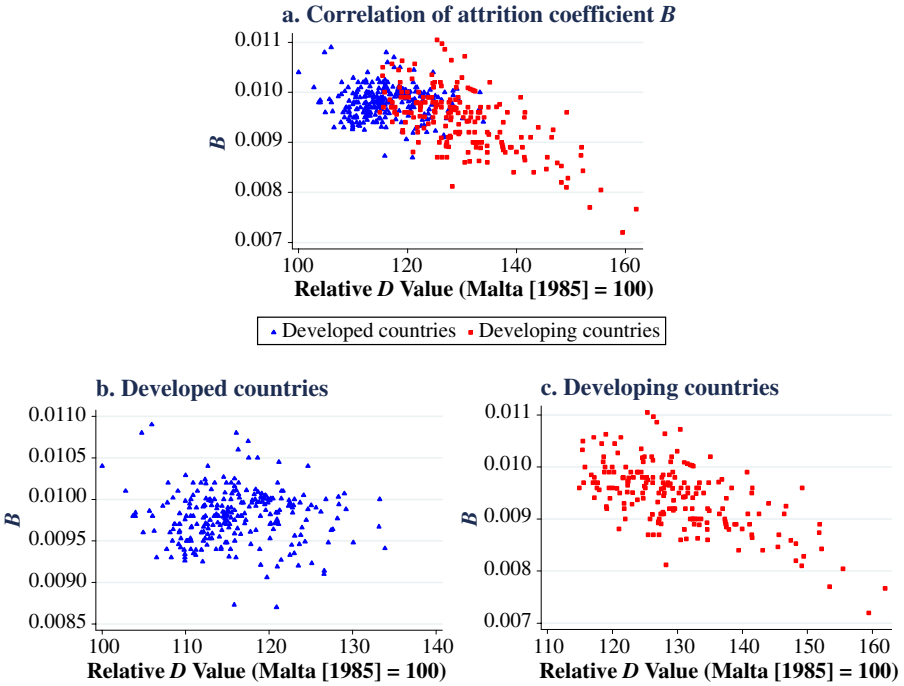

Fig. 4 The correlation of the attrition coefficient B and the national ecological system parameter D for developed and developing countries. B is calculated from the inverse relationship between $\ln R_0$ and α by assigning a value of K ($K = 1$) (Strehler and Mildvan 1960)

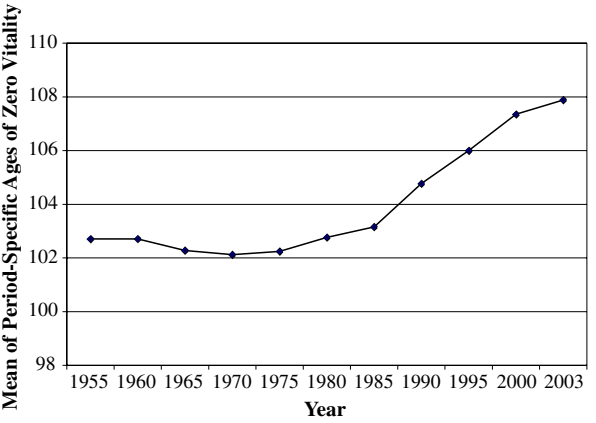

Fig. 5 The trajectory of the mean of period-specific ages of zero vitality among 42 countries, 1955–2003

group-based trajectory model, yielding three distinct longevity trajectories, as shown in Fig. 6. All three trajectories are quadratic in shape (BIC =−1158.28).[9] Of the 42 countries, 64% and 19.3% belong to quadratic trajectories 1 and 2, respectively, wherein the mean age of expected zero vitality shows declines during the 1960s and then steadily increases from 1970 onward. The difference between these two trajectories is that the first trajectory has a higher starting age of expected zero vitality than the second one. Nearly 16.7% of the countries belong to trajectory 3, where age of expected zero vitality is higher than those of the other two, decreases after 1960, and then increases again from 1980 onward. More specifically, trajectory 1 contains Norway, the United States, Australia, Austria, Belgium, Canada, Chile, Denmark, Finland, France, Germany, Italy, Japan, New Zealand, Portugal, Sweden, Switzerland, Trinidad and Tobago, Venezuela, Cuba, Hong Kong, Israel, Bulgaria, Greece, Hungary, Poland, and Romania; trajectory 2 contains Argentina, Egypt, Ireland, the Netherlands, Mauritius, Singapore, Malta, and the United Kingdom; and trajectory 3 contains Costa Rica, Mexico, Panama, Puerto Rico, Ecuador, Philippines, and Thailand.[10]

The three trajectories provide an intriguing classification of the 42 countries, since those belonging to trajectory 3 are concentrated in Central America and Southeast Asia. Reestimating the finite-mixture trajectory

[9] We dropped 1955 mortality data to match with national ecological system data available from the World Bank Group. Three quadratic trajectories are consistent with the trajectory of the mean of $1/B$ from 1960 to 2003, as shown in Fig. 5.

[10] The findings are based on male mortality data. An analysis of female age of zero vitality trajectories shows some similarities and some differences in group memberships from the male results. Among the 42 countries, 26 countries have the same group membership for male and female: highest trajectory (Panama, Mexico, Ecuador, Philippines, and Thailand), middle trajectory (the United States, Australia, Belgium, Canada, Chile, Denmark, France, Japan, New Zealand, Sweden, Switzerland, Venezuela, Cuba, and Hong Kong), and lowest trajectory (Argentina, Egypt, Ireland, Mauritius, Singapore, Malta, and the United Kingdom). But, in 13 countries, and especially in Europe (Norway, Austria, Finland, Germany, Italy, Portugal, Bulgaria, Greece, Hungary, Poland, Romania, Trinidad and Tobago, and Israel), women in these countries move to the lowest trajectory while their counterparts belong to the middle trajectory. One exception in Europe is the Netherlands, where women move to the middle trajectory while their counterparts belong to lowest trajectory. Moreover, women in Costa Rica and Puerto Rico move to the middle trajectory while their counterparts belong to the highest trajectory. Further research should study these male-female differences in group memberships.

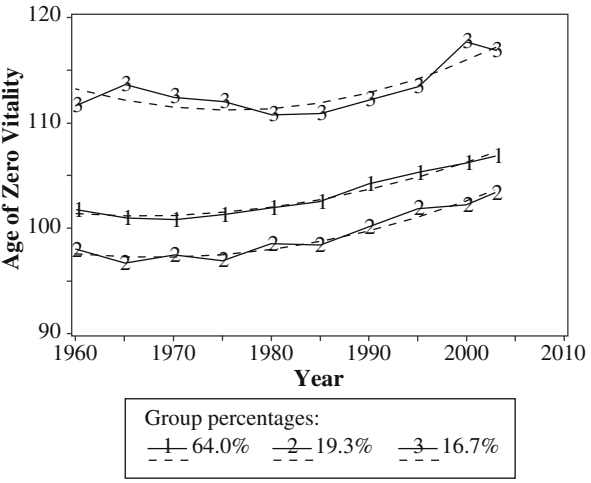

Fig. 6 Three distinct ages of zero vitality trajectories estimated by the semiparametric group-based trajectory model. Expected trajectories are indicated by dashed lines and observed trajectories, by solid lines

models by adding time-constant risk factors (in this case, the risk factor is region), we find that the region variable has a strongly significant effect on membership belongings. As shown in Table 1, region (Central American and Southeast Asian countries = 1, others = 0) significantly distinguishes trajectory 3, but not trajectory 2, from trajectory 1 (the reference group).

This raises the question, Why does region have such a strong effect on trajectory membership? Is it because Central American and Southeast Asian countries have similar ecological system characteristics—POET characteristics—that promote survival? Or is it because their populations have gene structures or expressions that may result in similar normal aging processes? Or is it an interaction of both? Recall that the vitality attrition coefficient B is determined by the normal aging process parameter b and the relative ecological system deleteriousness parameter D. As noted earlier, variations in the aging process parameter b cannot be estimated from this aggregate demographic data set. Therefore, we next examine whether the Central American and Southeast Asian countries have similar advantageous ecological systems with respect to the deleteriousness parameter D.

5. Do any national organizational, population, and technological characteristics account for variations in D? The SM model uses the overall relative ecological system deleteriousness parameter D to rank countries by the extent to which their ecological systems promote survival, but it does not specify what national organizational, population, and technological characteristics account for D. We address this limitation by using random-effects panel regression models for the merged panel data.[11]

[11] The country-period mortality data and national ecological system data were merged. We applied the Hausman test to determine if it is statistically justifiable to use random-effects instead of fixed-effects regression models. The results show that the coefficients estimated by the efficient random-effects estimator are the same as those estimated by the consistent fixed-effects estimator (Prob > chi-squared = .539); therefore, the random effects for the country-specific intercepts can be assumed to be independent of the regressors. Accordingly, we use the coefficient estimates from the random-effects panel model.

Table 1 Relationship of regional location of countries to trajectory group membership

Trajectory	Parameter	Estimates	SE	*t* Tests	*p* Values
2	Constant	−1.2541	0.51485	−2.436	0.0153
	Region	0.13132	1.28226	0.102	0.9185
3	Constant	−2.457	0.75118	−3.271	0.0012
	Region	2.73695	1.03041	2.656	0.0082

Before conducting random-effects panel regression analysis, we applied principal component analysis to the structural covariates of our regressor space to ascertain whether this would give some insight into how many relatively independent sources of variation there are in this space. Application of this method to the eight explanatory variables identified earlier (excluding the regional location identifier) reveals three clusters across all the time periods, as shown in Table 2.[12] The first is the *employment rate*, consisting of the total labor force participation rate (% of total population ages 15–64) and the male labor force participation rate (% of male population ages 15–64). The second is *population structure*, composed of population density (people per sq. km), percentage of population in the largest city, and percentage of population in urban agglomerations greater than 1 million. The third is *technology*, consisting of gross domestic product per capita (in constant 2000 U.S. dollars), and GDP per unit of energy use (constant 2000 PPP dollars per kg of oil equivalent). The "shares of food industry in BOD (biochemical oxygen demand) emissions"[13] indicator does not cluster with other variables and separately represents *industry structure*. A larger share of food industry in BOD emissions indicates a larger share of agricultural and food productivities in the industrial composition of a country. Because of large amounts of missing data in WDI for developing countries and for earlier time periods, we were not able to include other potentially important indicators of the four elements of the POET system. Nonetheless, these empirically derived clusters are consistent with the theoretical classification based on the POET model. They were used to form composite indices for these three ecological system dimensions.

Table 3 presents estimates of the resulting random-effects regression models with the ecological system deleteriousness coefficient D as the outcome variable. As shown in Model 1, the estimated coefficient for year is a statistically significant −0.110. This implies that the D value decreases over time, or the average national ecological system across the 42 countries became less deleterious over the period 1960–2003.

[12] Only the components accounting for substantial variance in the regressor space and having substantial component loadings for two or more regressors were identified. When calculating the component score, we included only those variables that possessed component loadings of .5 or greater across all time periods.

[13] Emissions of organic water pollutants are measured by biochemical oxygen demand (BOD), which refers to the amount of oxygen that bacteria in water needs to break down waste. This is a standard water-treatment test for the presence of organic pollutants (World Development Indicators 2008).

Table 2 Structural indices and covariates

Structural Index / Covariate	Variable
Industry Structure	Water pollution, food industry (% of total biochemical oxygen demand [BOD] emissions)
Employment Rate	Labor force participation rate, total (% of total population ages 15–64), and male (% of male population ages 15–64)
Population Structure	Population density (people per sq. km), population in the largest city (% of urban population), and population in urban agglomerations > 1 million (% of total population)
Technology	GDP per capita (constant 2000 U.S. dollars) and GDP per unit of energy use (constant 2000 PPP dollars per kg of oil equivalent)

Table 3 Random-effects model estimates of relationships of POET covariates to *D* (standard errors in parentheses)

	Model					
Structural Index/ Covariate	1	2	3	4	5	6
Intercept	339.520***	335.766***	293.494***	334.560***	301.019***	227.362**
	(61.314)	(61.430)	(63.119)	(62.340)	(61.784)	(72.464)
Year	−0.110***	−0.110***	−0.086**	−0.106***	−0.090**	−0.053
	(0.031)	(0.031)	(0.032)	(0.032)	(0.031)	(0.037)
Region (Central America and Southeast Asian countries = 1, others = 0)		10.120***	11.495***	11.943***	13.872***	12.654***
		(2.984)	(3.082)	(3.076)	(2.938)	(2.761)
Water Pollution, Food Industry (% of total BOD emissions)			−0.099*	−0.097*	−0.118**	−0.114**
			(0.044)	(0.043)	(0.042)	(0.042)
Employment Rate				−1.267**	−1.218**	−1.165**
				(0.399)	(0.389)	(0.389)
Population Structure					−2.200***	−1.829**
					(0.656)	(0.633)
Technology						−1.359*
						(0.671)
N	159	159	159	159	159	159
Wald χ^2	12.83	24.24	29.56	41.23	55.30	62.14

Sample sizes are relatively small after all the missing values in all variables are deleted

*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$

The other nested Models 2–6 yield several findings. First, larger shares of the food industry in emissions of organic water pollutants than other industries (e.g., chemical, clay and glass, metal, paper and pulp, textile, and others) have a negative relationship with *D*; in other words, larger shares of food industry in BOD (biochemical oxygen demand) emissions are good for national ecological system. This indicates that agricultural and food productivities are beneficial for a country's ecological system to promote survival. Second, the employment rate, which indicates the degree to which population members are engaged in sustenance-related pursuits, also has a negative relationship with *D*; that is, the higher the employment rate, the less deleterious the ecological system. Third, population structure, which includes population density and the concentration of population members in urban areas, makes for a less deleterious national ecological system. Fourth, technology, which includes GDP per capita and GDP per unit of energy use, drives a better ecological system. When the technology index is included in the regression, year becomes nonsignificant, which implies that improving technology explains why national ecological systems became less deleterious over the years 1960 to 2003. We conclude that national organizational, population, and technological characteristics do account for substantial variations in *D*.

Explaining the Independent Effect of Regional Location

An intriguing finding from Table 3 is the consistently statistically significant effect of *regional location* across all five models. That is, geographical location in Central America and Southeast Asia increases the expected value of environmental deleteriousness *D* from the estimated regression models by 10 to 14 points. This means that geographical location in these regions, net of the other regressors that measure and control for specific aspects of the population, organization, environment, and technology aspects of the ecological systems of the countries in the analysis, makes for a substantially more deleterious environment. On the other hand, the 11 countries in this regional grouping tend to have higher expected ages of zero vitality $1/B$ than other countries and smaller estimated fractional losses of vitality *B*. These exceptional countries account for the negative correlation of *B* and *D* in Panel c of Fig. 4.

Figure 7 further demonstrates the negative relationship between *B* and *D* in the trajectory 3 countries, which are concentrated in Central America and Southeast Asia, compared with other countries belonging to trajectory 1 or 2. This raises the question, Why do Central American and Southeast Asian countries have relatively unfavorable national ecological systems but higher estimated ages of expected zero vitality than other countries? A related but distinct question is, Why is the correlation between *B* and *D* negative *within* this set of countries?

One possible explanation, as mentioned above, is *survival tradeoff theory*. This theory suggests that individuals may "increase adaptive capacity (e.g., the rate of DNA and protein repair)" to the magnitude of environmental stresses "at the expense of a reduction in robustness," which can increase life span (Yashin et al. 2002b:213). This explanation can simultaneously answer the above two questions. However, if this explanation is applicable, the question remains, Why do the beneficial national ecological systems of some other countries, such as Belgium, Canada, and Germany, also reduce the fractional loss of vitality coefficient *B* and thereby promote survival?

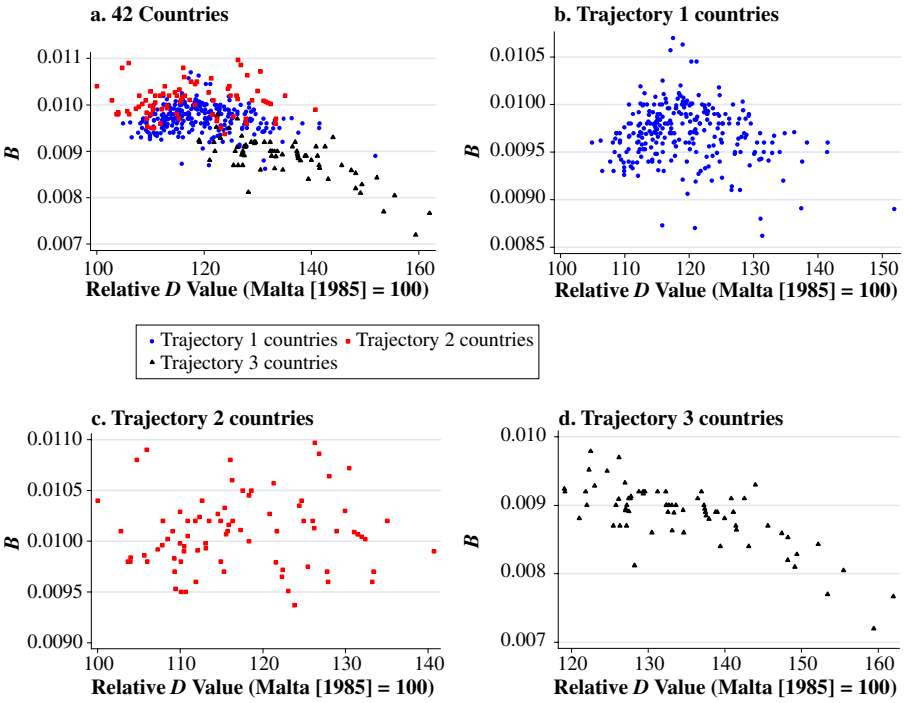

Fig. 7 The correlation of the attrition coefficient B and the national ecological system parameter D for the entire set of countries and the three trajectory groups

Another possible explanation is *heterogeneous frailty effects*; that is, a more deleterious environment D will kill more frail individuals at early ages and leave relatively more strong and fit persons at older ages. This, in turn, could contribute to higher longevity for the elderly. As shown in Fig. 8, when D increases (i.e., the ecological system becomes more deleterious), the initial mortality rate increases.[14] Therefore, it is possible that a deleterious ecological system kills more young frail Central American and Southeast Asian men and leaves relatively more robust adults than in other countries. Since the Central American and Southeast Asian countries are identified separately from the other countries in Fig. 8, and since they indeed are concentrated at the higher end of the D axis as well as at the upper reaches of the $\ln R_0$ axis, Fig. 8 provides evidence in support of this explanation. Figure 9 further illustrates the much higher initial mortality rates in the trajectory 3 countries than for those in trajectories 1 or 2.

Note, however, that our mortality data begins with age 30. This may generate artificially higher ages of expected zero vitality in the Central American and Southeast Asian countries than in others. To assess this possibility, we conducted sensitivity analyses and found that the group membership basically is not changed by choosing different starting ages (age 25 or age 30). Only two exceptions happen when the starting age changes from 30 to 25: (1) Venezuela moves from trajectory 1

[14] We also can obtain the positive relationship between D and $\ln R_0$ from the equations for B and D. D is the standardized B/α. To simplify, $D = B/\alpha$. Since $B = -\alpha/\ln R_0$, therefore, $D = -1/\ln R_0$, or $\ln R_0 = -1/D$. Thus, as D increases, $\ln R_0$ also increases.

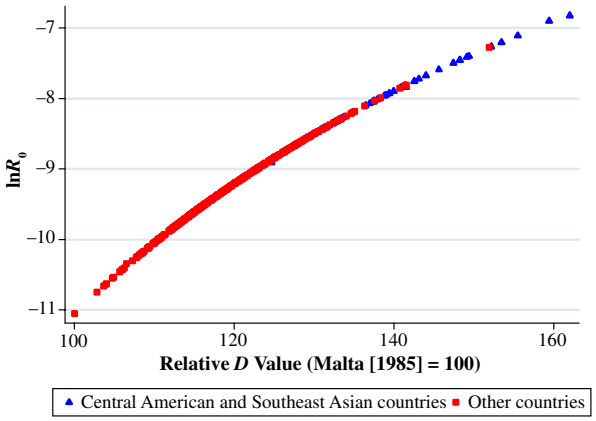

Fig. 8 The correlation of the logarithm of initial mortality rate $\ln R_0$ and the national ecological system parameter D

(the middle trajectory) to trajectory 3 (the highest trajectory); and (2) Hungary moves from trajectory 1 (the middle trajectory) to trajectory 2 (the lowest trajectory). Even if the heterogeneous frailty effects explanation can answer the above two questions, it cannot explain the nonsignificant relationship between B and D *within* trajectory 1 and 2 countries.

A third possible explanation is *position on the epidemiological transition*. Most trajectory 1 and trajectory 2 countries have gone through to the fourth stage of the epidemiological transition; afterward, increases in the age of zero vitality respond slowly to the changes in ecological system. This notion can explain the lack of a nonzero regression relationship between D and B in Fig. 7 for the trajectory 1 and 2 countries. By contrast, trajectory 3 countries are more likely to be in the second or third stage of the epidemiological transition, wherein big increases in the age of zero vitality have been substantially influenced by imported modern medical technology made available through bilateral or international cooperation (Omran 1982) while their national ecological system improves slowly.

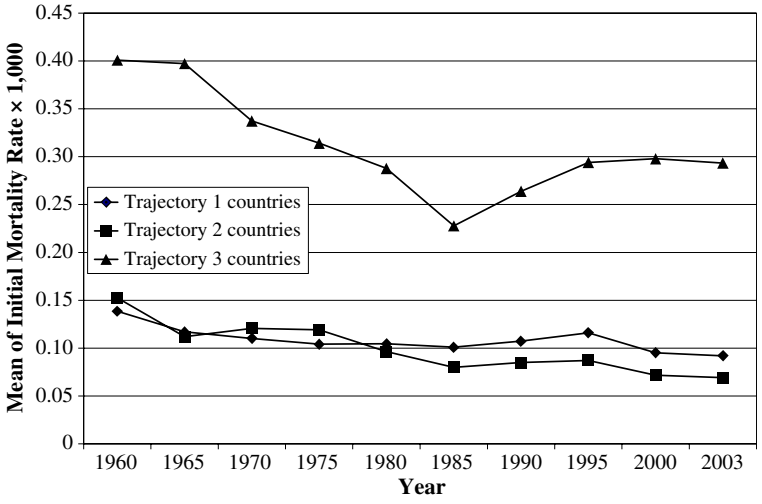

Fig. 9 Means of initial mortality rates $R_0 \times 1{,}000$ among the three trajectory groups, 1960–2003

This may explain the negative relationship between D and B in the trajectory 3 countries. While these explanations can solve the second question, they cannot address the first one: why Central American and Southeast Asian countries have a relatively unfavorable national ecological system but higher estimated ages of expected zero vitality than other countries.

A fourth possible explanation is the *basic ecological demography physical environment variable, regional location itself, and the impact this variable has on the health, mortality, and longevity of adults in the middle and older ages*. The impact of regional location can occur through various mechanisms. One that appears to be quite important is that Central American and Southeast Asian countries are located in the tropics with year-round sun exposure, which helps generate vitamin D inside human bodies (Grant et al. 2003; John et al. 2005; Tuohimaa et al. 2007). Vitamin D can reduce disease risk through reduced incidence of cancers and cardiovascular disease and through stronger bones, which reduce the chances of falls and bone fractures among the elderly (Broe et al. 2007; Garland et al. 2006, 2007; Gorham et al. 2005; Grant and Holick 2005; Gross 2005). Faloon (2008) recently reviewed 175 scientific articles and concluded that vitamin D can prevent millions of needless deaths by suppressing chronic inflammation, which is an underlying factor in the development of numerous age-related diseases, such as rheumatoid arthritis, chronic muscle pain, metabolic syndrome, congestive heart failure, and stroke. It is estimated that 40%–100% of the elderly in the United States and Europe have insufficient or deficient levels of vitamin D (Holick 2007). Compared with the United States and Sweden, Costa Rican nonagenarians have exceptionally high life expectancy, and this advantage comes mostly from a lower prevalence of cardiovascular diseases in the older ages (Rosero-Bixby 2008). This explanation sheds light on the question of why Central American and Southeast Asian countries have relatively unfavorable national ecological systems but higher estimated ages of expected zero vitality than other countries. But this explanation cannot solve the second question.

Related to this is a final possible explanation: *gene structures and expressions*. That is, Central Americans and Southeast Asians may have similar longevity genes and/or genes that are activated by interactions with their physical and social environments through such factors as sun exposure, activity patterns, social relationships, and diets that enable them to have higher ages of expected zero vitality despite relatively disadvantageous ecological systems. This explanation also can answer the first but not the second question.

In brief, a full explanation for the smaller values of the vitality attrition coefficients B and the correspondingly higher ages of expected zero vitality $1/B$ for countries located in Central America and Southeast Asia in the presence of higher coefficients of environmental deleteriousness coefficients D likely involves a combination of these and possibly other theories, explanations, and mechanisms. That is, the higher levels of environmental deleteriousness may indeed work through heterogeneous frailty and survival tradeoff effects to increase the initial mortality rate $\ln R_0$. Individuals who survive to ages 30 and beyond accordingly may have increased adaptive capacity and may be less frail. They also may benefit, during their middle and older adult ages, from the high levels of sun exposure in these regions and the beneficial effects of vitamin D production on longevity gene expressions and reductions in disease risk associated therewith. Piecing together and assaying the

relative impacts of these and other possible mechanisms to address the two questions stated above is a major question for future research.

Can Unreliable Age-Specific Mortality Data Account for the Findings?

An alternative explanation for the hidden heterogeneity in trajectories of SM implied ages of zero vitality that we have found, as illustrated in Fig. 6, and the related independent effects of regional location on the environmental deleteriousness parameter D is that these findings are due to unreliable data on the age-specific mortality rates used in the analyses. In particular, the exceptionally high SM ages of expected zero vitality that we have estimated for the Central American and Southeast Asian countries in trajectory 3 of Fig. 6 can be called into question and ascribed to data inaccuracies and unreliability, as demographers would expect to find at least some developed nations (e.g., Japan, Sweden, or France) in this trajectory.

Although the possible effects of data unreliability on findings from cross-national demographic studies—especially those that employ data from developing countries—can never be absolutely ruled out, there are two important considerations that indicate that these effects do not fully account for our findings. First, the highest age group used in these mortality analyses and estimates of the SM parameters is the open-ended interval of ages 85 and older. In particular, the analyses are not based on age-specific mortality rates for the extreme old ages of 90 and above, wherein the accuracy of age specificity due to age exaggeration or misreporting can deteriorate even for data from developed countries.

Second, independent of the present study, some demographic research has found relatively higher life expectancy, better health, and/or lower mortality for the elderly in some trajectory 3 countries. Specifically, using ages (at death or any time of observation) computed from birthdates in the Costa Rican birth registration ledgers rather than those reported by respondents, Rosero-Bixby (2008) found that mortality at age 90 in Costa Rica is at least 14% lower than 13 high-income countries (including the Unites States and Sweden), and this advantage comes mostly from lower prevalence of cardiovascular diseases in the older ages. Sorlie et al. (1993) found that middle-aged (45 to 64) and older (65 and older) Hispanic Americans (e.g., Mexican Americans and Puerto Rican Americans) have lower mortality from all causes than non-Hispanic Americans (also see Rosenwaike 1987). Their advantages were even greater after adjusting for lower annual family income than non-Hispanic Americans. Furthermore, foreign-born Mexican Americans have lower mortality risk compared with native-born persons (Sorlie et al. 1993). Although these results may be partly due to "healthy migrant" effects, several studies have found that incidence and death rates from coronary heart disease and cancer are lower in Puerto Rico than in the United States (Garcia-Palmieri et al. 1965; Gordan et al. 1974; Martinez et al. 1975). Rosenwaike and Hempstead (1990) found that, while Puerto Rico residents have higher mortality rates than Puerto Rico–born residents of the mainland United States (which suggests "healthy migrant effect"), they have advantages on all causes of death (including heart disease and cancer) over U.S. whites. There is relatively less research literature on longevity patterns in Thailand and the Philippines, but the U.N. Department of International Economic and Social Affairs (1982) found that age-specific mortality patterns in these two countries are similar to those in Latin American countries.

In sum, although inaccurate and unreliable mortality data in trajectory 3 countries, located in Central America and Southeast Asia, might be a problem for some analyses, we believe that we have minimized its effects and that it is not large enough to invalidate the main findings reported herein.

Conclusions

Our findings reveal considerable hidden heterogeneity in the parameters of the Strehler-Mildvan general theory of mortality and aging. They also show the fruitful results of extending SM theory by synthesizing it with ecological demography theory. Specifically, evidence has been provided that (1) the SM correlation exists but is not constant over time; (2) within the SM framework, the implied ages of expected zero vitality appear to be variable over time; (3) trajectories of the implied ages of expected zero vitality are not homogeneous among the countries; (4) Central American and Southeast Asian countries have higher ages of expected zero vitality than other countries in spite of relatively disadvantageous national ecological systems; (5) within the group of Central American and Southeast Asian countries, a more disadvantageous national ecological system is associated with a smaller rate of physiological decline (parameter B) and a higher age of expected zero vitality ($1/B$); and (6) larger agricultural and food productivities, higher labor participation rates, higher percentage of population living in urban areas, and larger GDP per capita and GDP per unit of energy use are important beneficial national ecological system factors that can promote survival.

The first three findings indicate the fruitfulness of generalizing Strehler-Mildvan theory to incorporate heterogeneity among human populations. The last three findings demonstrate the utility of integrating Strehler-Mildvan theory with ecological demography theory and suggest three further research directions: (1) identify other population, organizational, environmental, and technological characteristics that affect national environmental deleteriousness D; (2) study why Central American and Southeast Asian countries have higher ages of expected zero vitality than other countries in spite of relatively disadvantageous national ecological systems; and (3) explain the inverse relationship between the vitality attrition coefficient B and environmental deleteriousness D *within* Central American and Southeast Asian countries.

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