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Heterogeneity in the Strehler-Mildvan

General Theory of Mortality and Aging

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Abstract

The objective of this study is to examine and further develop the Strehler-Mildvan (SM) general theory of mortality and aging published in 1960 in Science. We tested three predictions from the SM theory by examining the age dependence mortality patterns for 46 countries (including developed and developing countries) over the period of 1955-2003. By using descriptive analyses, finite mixture regression models, and random effects panel regression models, this study finds that: (1) the SM correlation exists but is not constant; (2) within the SM framework, the implied age of expected zero vitality appears to be variable over time; (3) longevity trajectories are not homogeneous among the countries; (4) Central American and South-East Asian countries have higher expected age of zero vitality than other countries in spite of relatively disadvantageous national environments; (5) within the group of Central America and South-East Asia countries, the relationship between the national environment parameter D and the rate of physiological decline parameter B is negative; and (6) larger shares of food industry in emissions of organic water pollutants, GDP per capita, and urbanization are very important factors of a country's environment which can promote survival. These findings indicate that the SM theory needs to be generalized to incorporate heterogeneity among human populations, and the puzzling inverse relationships suggested by the fourth and fifth findings require additional research.

INTRODUCTION

The objective of this research is to examine and further develop a general theory of mortality and aging published nearly 50 years ago by Bernard L. Strehler and Albert S. Mildvan in *Science*. This theory presents a very elegant mathematical model of the age dependence of human mortality that can be used to rank countries by the extent to which their social and physical environments promote survival. The theory also can be used to produce estimates of age of expected zero vitality.

However, the empirical estimates used by Strehler and Mildvan (1960) to test their theory were based on a cross-sectional sample of a limited number of countries (30 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 their 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.

This research is important in the context of aging societies because it contributes to the scientific understanding of aging patterns and causes of different aging patterns across countries and periods. In addition, this research has important implications for public policy. Understanding the relationships between socioeconomic and other environmental factors and aging is essential for developing better welfare and other relevant policies to enhance population health.

THE STREHLER-MILDVAN THEORY

The *Strehler-Mildvan (SM) theory* synthesized 1) *Gompertz's Law*—the exponential increase of human mortality with age, $R_t = R_0 e^{\alpha t}$, with 2) the *linear decline of a vitality index* $V_t = V_0 (1 - Bt)$ with increasing age, and 3) *parameters measuring environmental stresses* (a measure of the frequency of environmental variations K and their average magnitude $_\varepsilon D$), where the attrition coefficient B ($= b + f(D)$) is the fractional loss each year of original vitality $V_0,$ b is the attrition coefficient due to normal aging, and *f(D)* is the attrition coefficient due to environmental factors as a function of the *summary measure of relative environmental deleteriousness*, D. This theory has three important predictions:

(1) the intercept lnR_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 group 1 has lower initial mortality rate than group 2, it should have 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;

(2) the fractional loss of vitality *B*, where vitality is the capacity of an individual organism to stay alive, is constant regardless of different situations; in other words, it is dominated by the normal aging process *b* and independent of environmental stresses *f(D)*; and

(3) 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.

This model has stimulated many subsequent studies, some of which suggest the SM correlation pattern was stable for adult mortality patterns from the year 1900 to 1986 in the US (e.g., Riggs 1992) and other developed countries, including mortality trends in industrialized countries (e.g., Riggs and Millecchia 1992; Prieto et al. 1996), stroke mortality (e.g., Riggs and Myers 1994), colon cancer mortality (e.g., Riggs 1993), malignant brain tumor mortality (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 US and suggest further extensions of the SM theory (Yashin et al. 2000, 2001, 2002a, 2002b).

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 is found that mortality tends not to accelerate, but rather to decelerate, at the oldest ages. Moreover, gains of survival over age 80 and even over age 90 have been accelerating since 1960 in nine countries (Wilmoth 1997). The population heterogeneity hypothesis argues that the deceleration is a statistical effect of compositional change, which 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 at present it seems implausible that all of the observed deceleration of mortality at older ages is an artifact of heterogeneity. Rather, it has been argued that "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). In

addition, the maximum age at death for some national populations has risen and continues to rise in a steady, almost linear fashion; "if we were close to observing a biological maximum, this trend would show some sign of deceleration, although none is evident" (Wilmoth and Lundstrom 1996).

RESEARCH QUESTIONS

Given these conflicting findings, the objective of this study is to subject the SM theory to further empirical evaluation by examining the age dependence mortality patterns for 46 countries (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 46 countries and the time period 1955-2003?

(2) Is the fractional loss of vitality B constant, i.e., is it dominated by normal aging process b and independent of the environment $f(D)$?

(3) Does a constant, or relatively constant, age of expected zero vitality[1] ($1/B$) exist? Or do the SM estimated age of expected zero vitality change over time?

(4) If the estimated age of expected zero vitality is not constant over time, do all 46 countries have the same trajectory of changes?

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[1] 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*. Since the SM model is deterministic, the implied age of zero vitality, estimated by 1/B, should be regarded as an expected value. In empirical applications to human populations, stochastic variability around this age 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 the 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 age of zero vitality for the population as a whole does not imply that no particular individual can live beyond that age.

(5) If there are detectable differences of trajectories of change in age of expected zero vitality, what accounts for this? The SM model also uses overall D to rank countries by the extent to which their social and physical environments promote survival but does not specify what national sociodemographic and economic characteristics account for D. Can any specific characteristics be identified?

METHODS

Data

The country-period-age-specific mortality dataset analyzed herein was compiled from the *Demographic Yearbook* 1955-2003 published annually by Department of Economics and Social Affairs of the United Nations. In order to directly test the SM theory, this dataset was organized into a pooled country-period data design. Each country-period case has the following five estimated SM parameters: the logarithm of initial mortality rate LnR_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 environment D.

National socidemographic and economic data were complied from World Development Indicators (WDI) published by the World Bank Group. We collected indicators of physical, socioeconomic, demographic, and medical environment. The earliest time period of these indicators is 1960, different from 1955 in *Demographic Yearbook* mortality data.

Analytic Methods

This study examines the five research questions stated above 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 semiparametric group-based trajectory model or the latent class trajectory model (Jones et al. 2001; Nagin 1999; Land, McCall and Nagin 1996; 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 modeling strategy and is designed to identify relatively homogeneous clusters of trajectories of development or change over time (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 in the finite mixture models. 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. Jones et al. (2001) shows the likelihood of observing the data trajectory for subject *i*, given he belongs to group *k*, is

$$\Pr(Y_i = y_i \mid C_i = k, W_i = w_i) =$$

$$\prod_{y_{ij}=Min} \Phi(\frac{Min - u_{ijk}}{\sigma}) \prod_{Min<y_{ij}<Max} \frac{1}{\sigma}\varphi(\frac{y_{ij} - u_{ijk}}{\sigma}) \prod_{y_{ij}=Max} (1 - \Phi(\frac{Max - u_{ijk}}{\sigma})),$$

where,

$$u_{ijk} = \beta_{0k} + year_{ij}\beta_{1k} + year_{ij}^2 \beta_{2k} + ... + w_{ij}\delta_k$$

We estimated random effects panel regression models to address the fifth question, that is, to ascertain what, if any, national physical environment, economic characteristics, and socciodemographic factors account for variation in the overall environmental deleteriousness parameter *D*. Random effects panel regression models generally are more statistically efficient than pooled OLS and fixed effect models (Wooldridge 2002). As an initial step in exploring 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: one indicator of national location, *region*, one indicator of pollution of the physical environment, the *share of a country's biological water pollutants accounted for by the food industry*, one indicator of economic development, *Gross Domestic Product* (GDP), and on indicator of the *extent of urbanization* of the country.

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 apply 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:

$$D_{it} = \beta_0 + \beta_1 Year_t + \beta_2 \operatorname{Re} gion_i + \beta_3 Pollution_{it} + \beta_4 GDP_{it} + \beta_5 Urban_{it} + c_i + u_{it}$$

where c_i is unobserved in all periods but constant over time, and u_{it} is a time-varying idiosyncratic error. The composite error is defined as $v_{it} = c_i + u_{it}$.

RESULTS

(1) Are $\ln R_0$ and α negatively correlated?

Figure 1 shows trends in estimates of $\ln R_0$ and α for each country for each five-year period from 1955 to 2000 and then 2003. It can be seen that some trend up, others go down, and still others appear to be quadratic, cubic and even irregular. Four different types of trends are more specifically illustrated in Figure 2[2]: (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 over time irregularly (e.g., the United States and Portugal); and (4) both $\ln R_0$ and α decline over time (e.g., Argentina and Australia). One thing in common in these different types is that $\ln R_0$ and α are inversed to each other. Figure 3 also clearly indicates that at the aggregate level, $\ln R_0$ and α are negatively correlated among all 46 countries, 1955-2003.

(2) Is B constant, i.e., is B dominated by b and independent of the environment?

When the aggregate ($\ln R_0$, α) inverse relationship is decomposed by countries and periods, the instability of the slope ($-1/B$) becomes evident. Figure 4 shows the trajectories of $1/B$ for the 46 countries from 1955 to 2003. As can be seen, some trajectories remain relatively constant, some increase, some decrease, and others follow quadratic and even cubic trends over time. The predicted $1/B$ also clearly represents different types of trajectories over the years for the 46 countries as shown in Figure 5. In Figure 6, the plots of $\ln R_0$ against α (where the slope is $-1/B$) also sheds light on variations in the SM correlation across populations: (1) some patterns have

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[2] The different trajectories for countries in Figure 1 and Figure 2 result from the different scales used in these two figures. In addition, Figure 1 portrays the trend of alpha × 100, while Figure 2 portrays the trend of alpha.

nearly constant slope over the last fifty years (e.g., The Netherlands and Greece); (2) some patterns have typical "hooks" corresponding to recent 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; (3) some patterns show the negative relationship between $\ln R_0$ and α, but the Y-intercept is decreasing 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); and (4) 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 B is not constant either over years or across countries. The next question is: Why is B not constant? Is it because the normal aging process parameter b is changing, or is it due to changes in the environmental relative deleteriousness parameter D? Since it is nearly impossible to capture changes in the aging process b by using this aggregate demographic dataset, we focus on how the environment D may affect the attrition coefficient B.

Figure 7 suggests that B is not independent of relative environmental deleteriousness D. That is, these two variables have a nearly linear relationship. But, surprisingly, when a national environment becomes worse (the D value increases further from 100), the rate of physiological decline B also decreases, which somewhat supports the "survival trade off" theory (Yashin et al. 2002b; Kirkwood 1990, 1996) 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" (Yashin et al. 2002b). In this case the value of D increases, but B decreases. However, this is not the only pattern displayed by these 46 countries. As shown in Figure 8, in some developed countries, such as U.S., Australia, Canada etc., the relationship between D and B is positive, that is, when environment D becomes better (i.e., the value of D

becomes smaller), the fractional loss of vitality becomes smaller. We further examined the correlation between b and D separately for developed and developing countries and found that it clearly negative in the developing countries, but essentially null in the developed countries, as shown in Figure 9.

(3) Does a constant, or relatively constant, age of expected zero vitality (*1/B*) exist?

Figure 10 plots the SM estimates of age of expected zero vitality *1/B* for each time period. This figure shows that the centers of these period-specific frequency distributions tend to increase slightly over time. As *B* decreases and vitality V_t increases, a population's age of expected zero vitality increases. Figure 11 shows the trajectory of the mean of *1/B* among these 46 countries from 1955 to 2003. As shown in Figure 11, *1/B* increases from 1955 to 1960 and then decreases until early 1970s and afterwards continually increases. This trend is consistent with the theory of third and fourth stages of the epidemiological transition (Omran 1971, 1982; Olshansky and Ault 1986). In the third stage, the *age of degenerative and man-made diseases*, the mortality level was stable at some low level and the life expectancy approached the "limits" (Omran 1971). But, since the late 1960s and early 1970s, the U.S. and other developed countries have experienced rapid declines in mortality rates for the major degenerative diseases (e.g., heart disease, cancer and stroke) and rapid increases in life expectancy. These are characteristics of the fourth stage of epidemiological transition, termed the *age of delayed degenerative diseases*) (Olshansky and Ault 1986).

(4) Do all 42 countries have the same trajectory of changes of SM estimates of age of expected zero vitality? If not, how do variations in national environment account for the different trajectories[3]?

Figure 4 and 5 remind us that the longevity trajectories are not homogeneous among these countries. A more accurate identification of different clusters of longevity trajectories shed more light on this pattern. By estimation of the semiparametric group-based trajectory model, we found three distinct longevity trajectories as shown in Figure 12. All three trajectories are quadratic in shape (BIC=-1138.08)[4]. Of the 42 countries, 64.6% and 18.8% belong to quadratic trajectories 1 and 2, respectively, wherein age of expected zero vitality shows declines during the 1960's and then steadily increases since 1970. The difference among these two trajectories is that the first trajectory has a higher starting age of expected zero vitality than the second one. Nearly 16.6% countries belong to trajectory 3, where levels of age of expected zero vitality are higher than those of the other two and it decreases after 1960 and then increases again since 1980. More specifically, trajectory 1 contains countries like Norway, US, 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 countries like Argentina, Egypt, Ireland, Netherlands, Mauritius, Singapore, Malta and UK; trajectory 3 contains countries like Costa Rica, Mexico, Panama, Puerto Rico, Ecuador, Philippines and Thailand.

The three trajectories provide an intriguing classification of the countries, since the countries belonging to trajectory 3 are concentrated in Central America and South-East Asia. We

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[3] To run SAS TRAJ analysis, we did not use four countries that have very incomplete mortality data.
[4] We dropped 1955 mortality data to match with national sociodemographic and economic data available from the World Bank Group. Three quadratic trajectories are consistent with the trajectory of the mean of 1/b from 1960-2003 as shown in Figure 10.

re-estimated the finite mixture modeling by adding time-constant risk factors (in this case, the risk factor is region) and found that the region variable has a strongly significant effect on membership belongings. As shown in Table 1, region (Central American and South-East Asia countries = 1, others = 0) significantly distinguishes trajectory 3 from trajectory 1 (which is the reference group).

This raises the question: Why does region have such a strong effect on trajectory membership? Is it because Central American and South-East Asia countries have similar advantageous physical, sociodemographic, and economic environments that can promote survival? Or is it because they have gene structures or expressions that may result in similar normal aging process? Recalling the formula for B, which is determined by the normal aging process parameter b and the relative environmental deleteriousness parameter D. Since it is nearly impossible to estimate variations in the aging process from this demographic dataset, we next examine whether the Central American and South-East Asia countries have similar advantageous environments with respect to the deleteriousness parameter D.

(5) Do any national physical environment, economic, and sociodemographic characteristics account for variations in D?

The SM model uses the overall relative environmental deleteriousness parameter D to rank countries by the extent to which their social and physical environments promote survival, but does not specify what national sociodemographic and economic characteristics account for D. Therefore, we next address this limitation by using random effects panel regression models for the merged panel data[5].

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[5] The country-period mortality data and national socidemographic and economic data are merged. We applied the Hausman test to determine if it is statistically justifiable to use random effects instead of fixed effects regression

Table 2 presents the results for factors that have significant effects on D. As shown in model 1, the estimated coefficient for year is a statistically significant -0.14. This implies that the D value decreases over time, or the average national environment across the 46 countries became less deleterious over the period 1960-2003. Larger shares of 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, which means larger shares of food industry in BOD emissions are good for national environment. GDP per capita makes for a less deleterious national environment. When GDP per capita is included in the regression, year becomes non-significant, which means overall increasing GDP per capita explains why the national environment became less deleterious over the years 1960 to 2003. Urbanization is also an important beneficial national environment factor which can promote survival.

The most interesting finding from Table 2 is that Central American and South-East Asia countries actually have relatively unfavorable national environments that cannot be explained by the physical environment, economic, and sociodemographic indicators examined in Table 2[6]. While these countries have relatively unfavorable national environments, they have higher age of expected zero vitality than other countries and smaller estimated fractional losses of vitality. These exceptional countries make B and D negatively correlated in Figure 7 although they are positively or not related in some other developed countries as shown in Figure 8. Figure 13 further demonstrates the unusual negative relationship between B and D in the trajectory 3 countries compared to other countries belonging to trajectory 1 or 2.

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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 = 0.31); therefore, the random effects for the country-specific intercepts can be assumed to be independent of the regressors and to use the coefficient estimates from the random effects panel model.

[6] The effect of region is highly significant throughout all models.

The remaining question is: Why do Central American and South-East Asia countries have relatively unfavorable national environments but higher estimated age of expected zero vitality than other countries? Another related but distinct question is: Why is the correlation between B and D is negative *within* these countries? One possible explanation, as mentioned above, is "survival trade off" theory. This theory suggests 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; Kirkwood 1990, 1996). This explanation can simultaneously solve the above two questions. However, if this explanation is applicable, the question remains: Why do the beneficial national environments of other countries reduce the fractional loss of vitality and thereby promote survival? Another possible explanation is population composition. A more deleterious environment will kill more frail individuals and leave more strong persons. As shown in Figure 14, when D becomes bigger (i.e., environment becomes worse), initial mortality rate will increase[7]. Therefore, it is possible that deleterious environment kills more young frail Central Americans and South-East Asians and leave more strong adults than other countries, and our mortality data just include people age 35 and over, which may generate artificial higher age of expected zero vitality than other countries. Although this explanation can solve the first question, it cannot address the second question. A third possible explanation is gene structures and expressions, that is, Central Americans and South-East Asians may have similar longevity genes and/or genes that are activated by interactions with their environments through such factors as diets that enable them to have higher age of expected zero vitality despite relatively disadvantageous environments. This explanation can solve the first question and potentially

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[7] We also can get the positive relationship between D and LnR_0 by deducting the equations for B and D. D is the standardized B/α. To simplify, $D=B/\alpha$. Since $B=-\alpha/LnR_0$, therefore, $D=-1/Ln\ R_0$, or $LnR_0=-1/D$. Thus, as D becomes bigger, LnR_0 also becomes bigger.

solve the second question, but more interdisciplinary research needs to be conducted to solve these puzzles.

CONCLUSION

In sum, this research provides evidence that (1) the SM correlation exists but is not constant over time; (2) within the SM framework, the implied age of expected zero vitality appear to be variable over time; (3) trajectories of the implied age of expected zero vitality are not homogeneous among the countries; (4) Central American and South-East Asian countries have higher age of expected zero vitality than other countries in spite of relatively disadvantageous national environment; (5) the relationship between the national environment parameter D and the rate of physiological decline parameter B is negative *within* Central America and South-East Asia countries; and (6) larger shares of food industry in emissions of organic water pollutants, GDP per capita, and urbanization are important beneficial national environment factors which can promote survival.

The first three findings indicate that the Strehler-Mildvan theory needs to be generalized to incorporate heterogeneity among human populations. The last three findings suggest three further research directions: (1) study what other sociodemographic and economic characteristics account for national environment *D*; (2) study why Central American and South-East Asian countries have higher age of expected zero vitality than other countries in spite of relatively disadvantageous national environment; (3) analyze the puzzling inverse relationship between vitality declining rate *B* and national environment *D within* Central American and South-East Asia countries.

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Figure 1. Trajectories of LnR_0 and α for each country, 1955-2003.
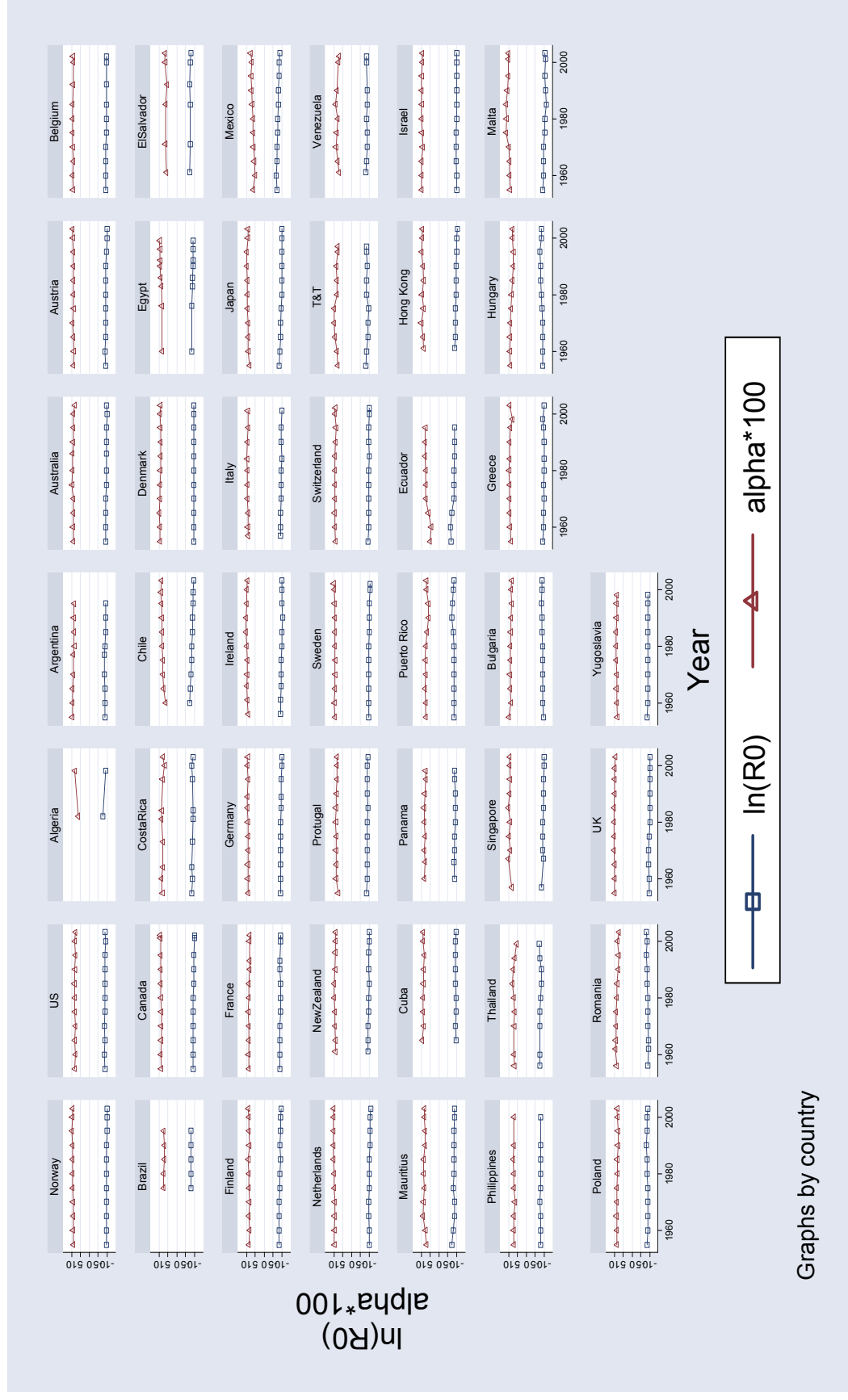
Graphs by country

Figure 2. Trajectories of LnR_0 and α for Netherlands, Denmark, US and Argentina, 1955-2003.
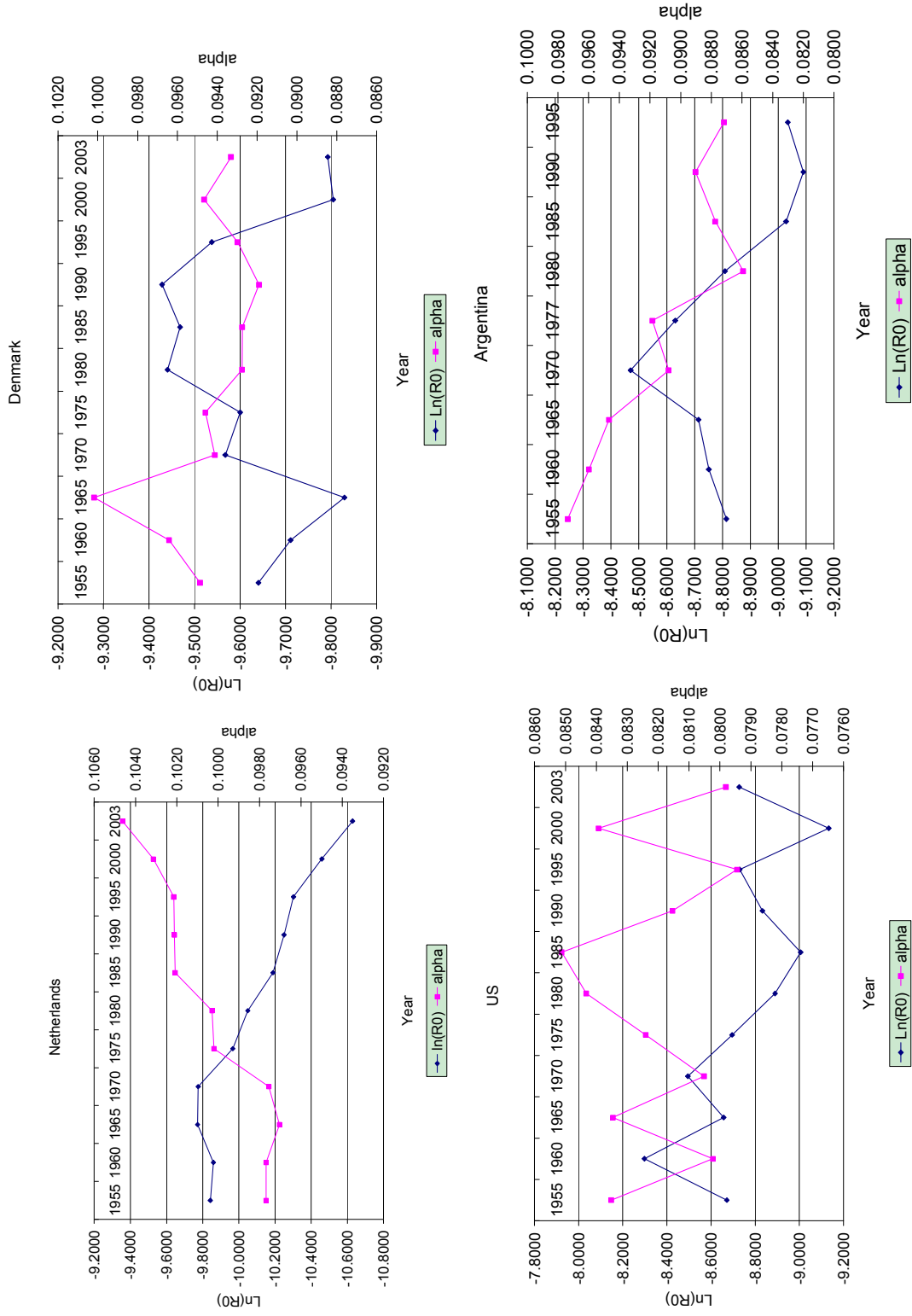
Figure 3. The Inverse Relationship between LnR_0 and α for 46 countries, 1955-2003.

lnr0 against alpha, 1955-2003
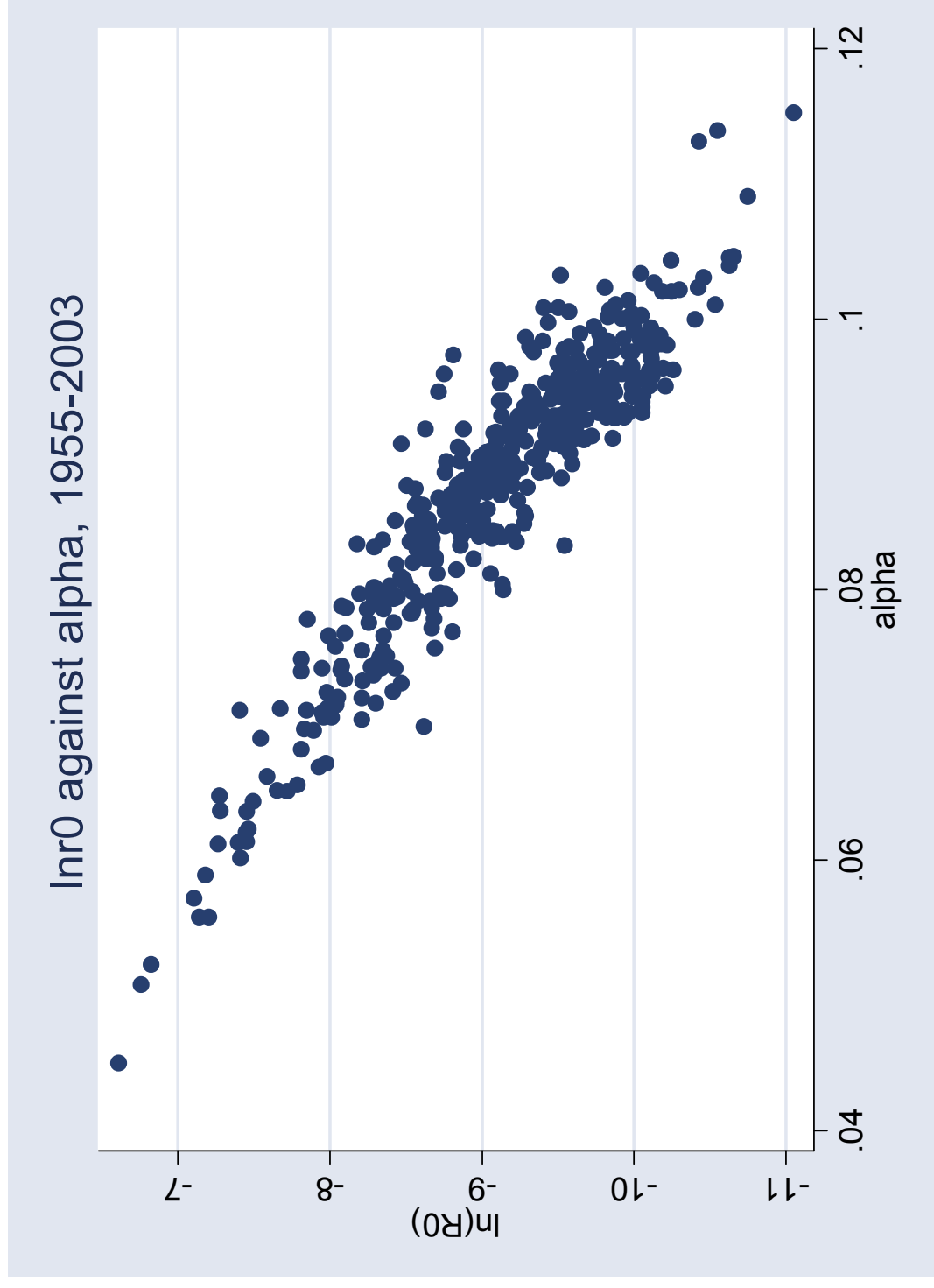
Figure 4. Trajectories of 1/B for each country, 1955-2003.

Graphs by country
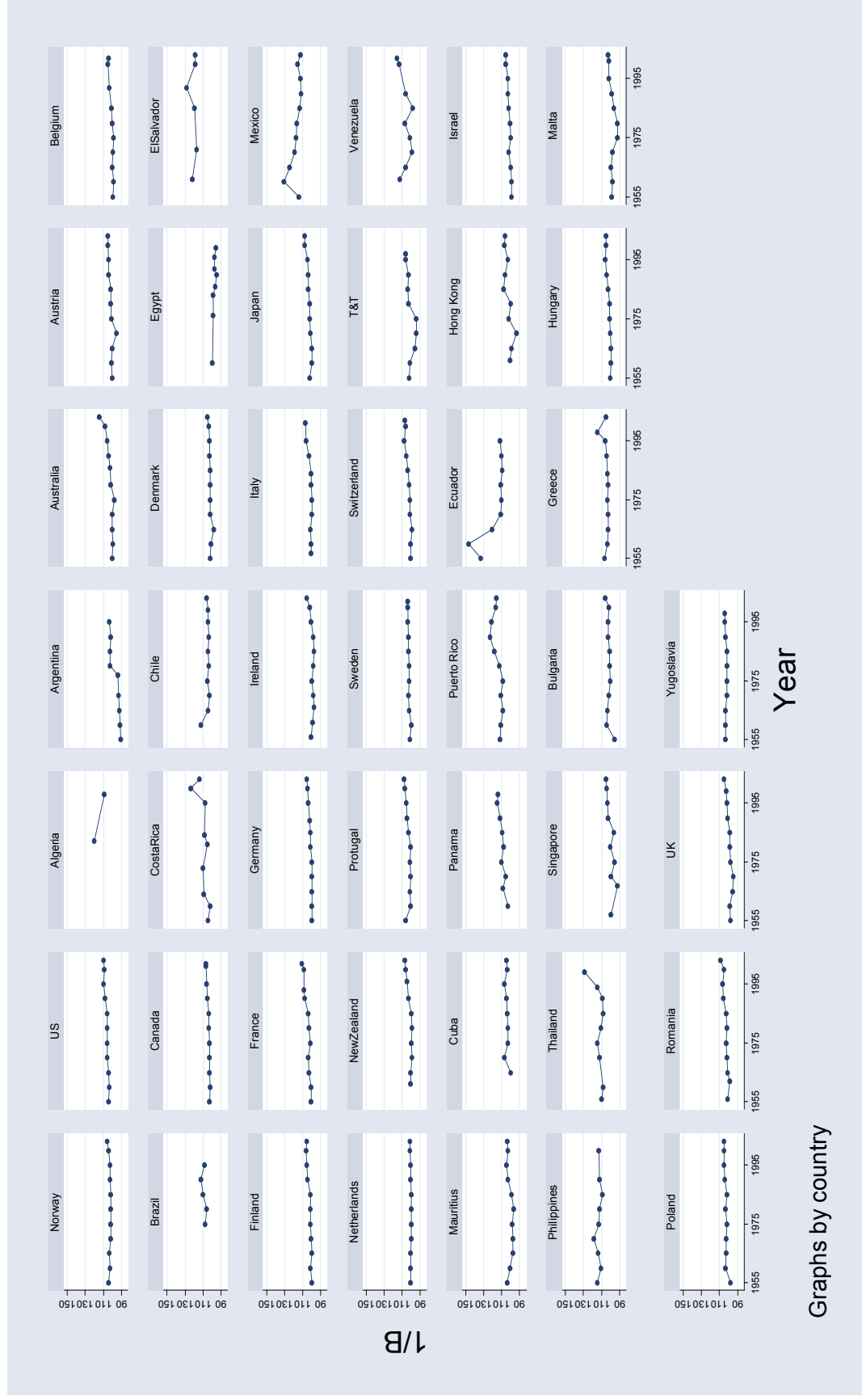
Figure 5. Predicted Trajectories of 1/B for each Country and For All 46 Countries Pooled Together.

Predicted 1/B for each country and for 46 countries

Predicted 1/B ——— Predicted 1/B for each country ——●——— Fitted 1/B for 46 countries

Year
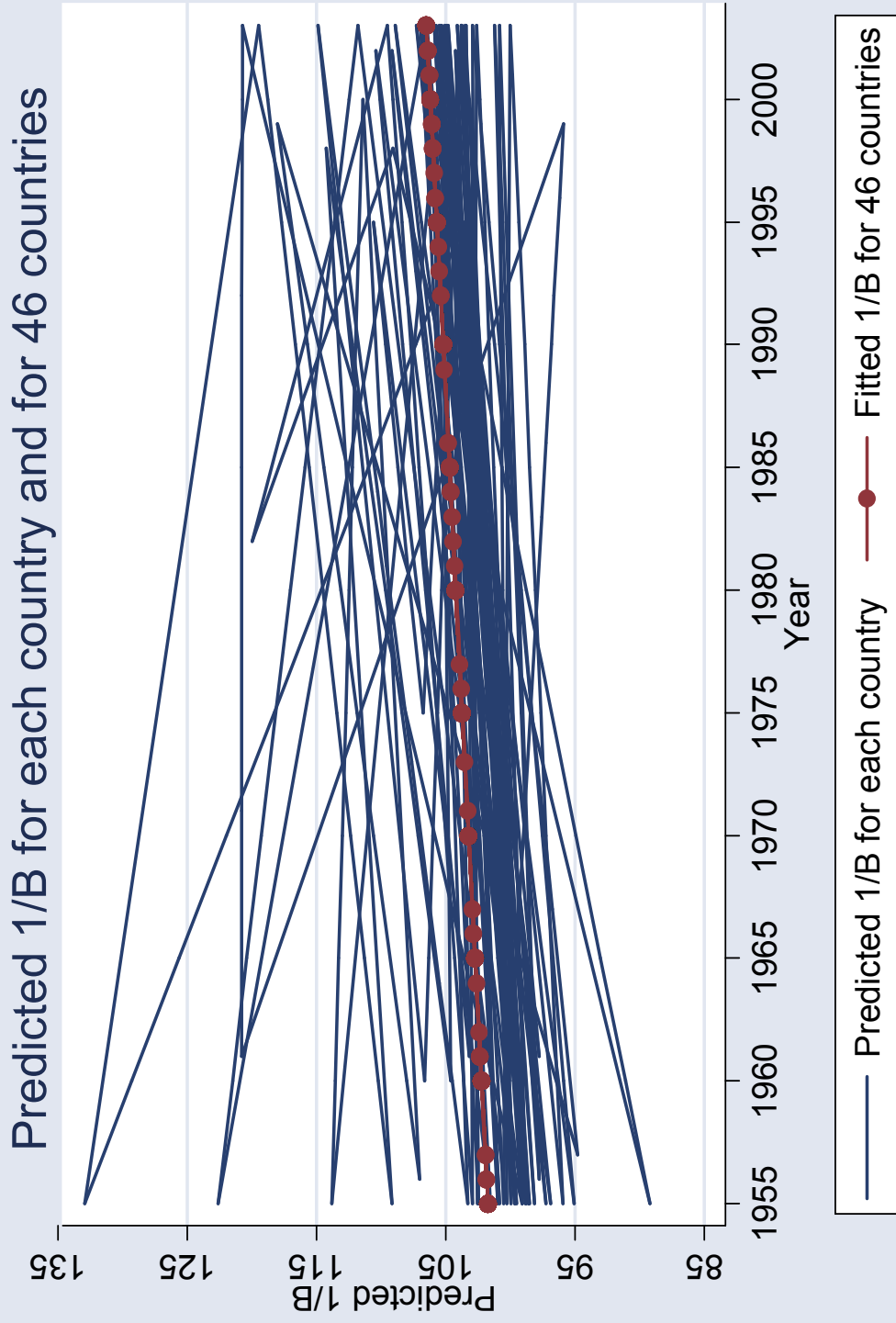
Figure 6. Plots of $\ln R_0$ against α for Netherlands, Japan, US and Australia, 1955-2003.
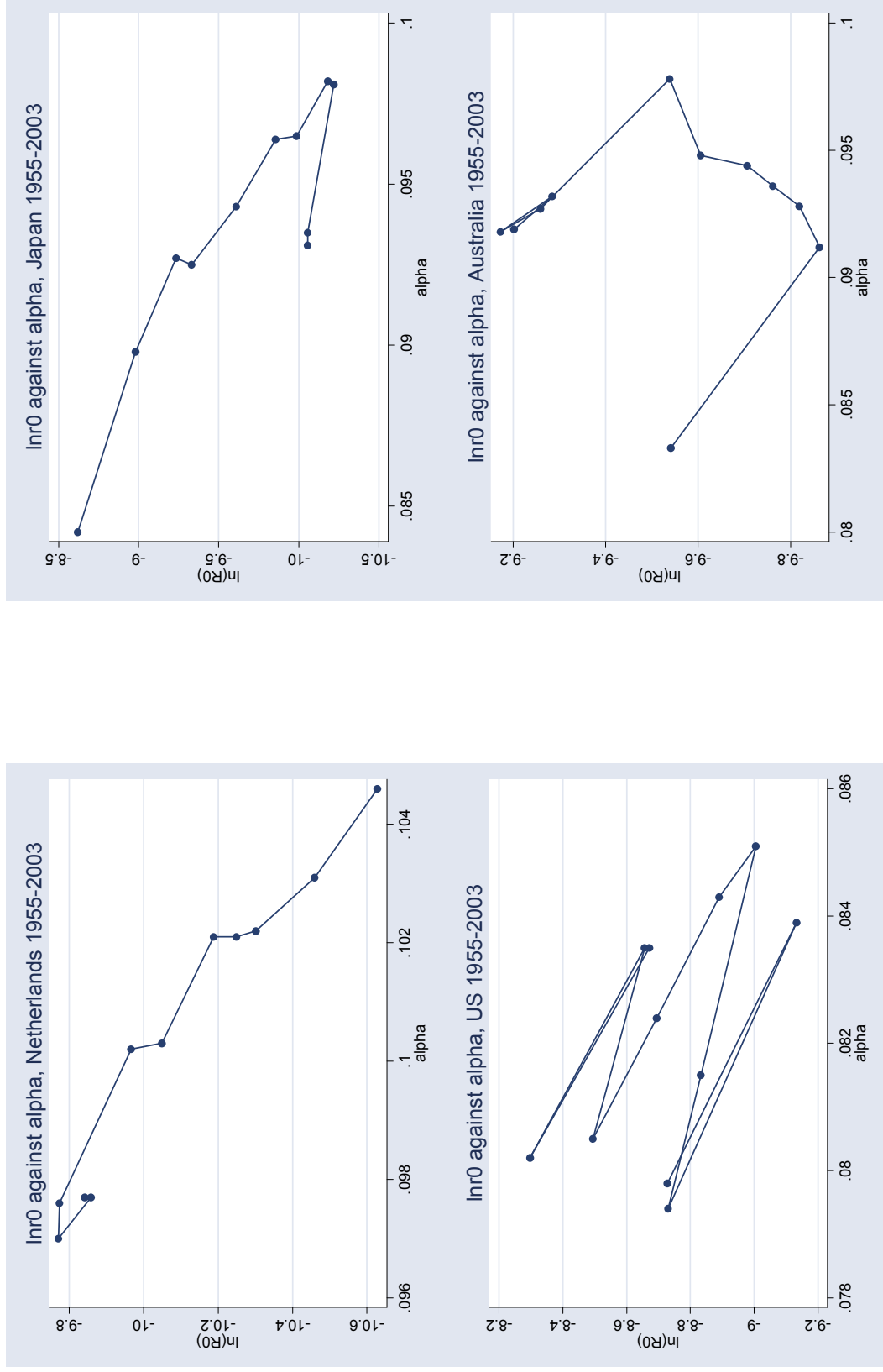
Figure 7. The Correlation of the Attrition Coefficient B and the National Environment Parameter D.
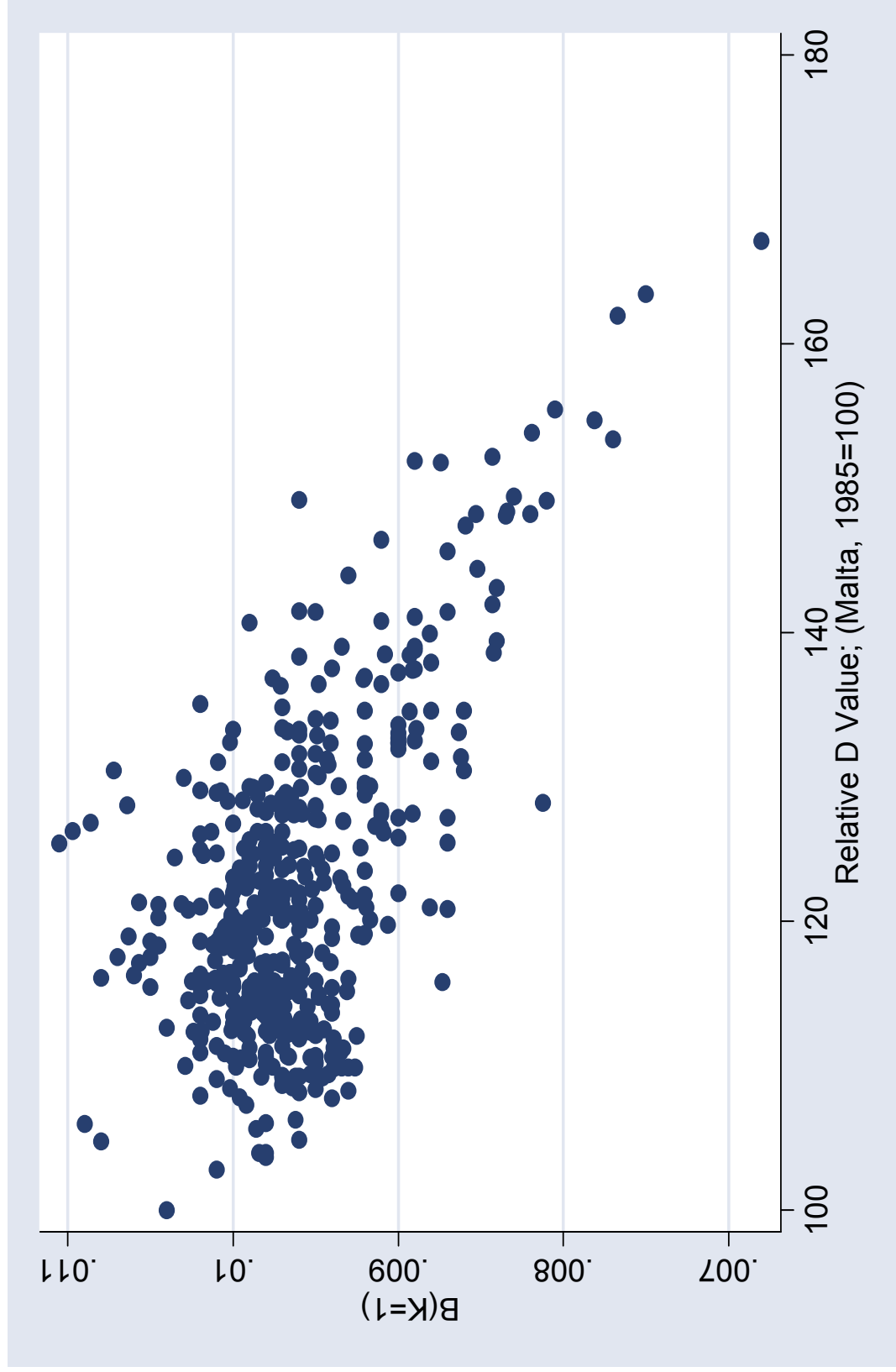
Figure 8. The Correlation of the Attrition Coefficient B and the National Environment Parameter D for Each Country.
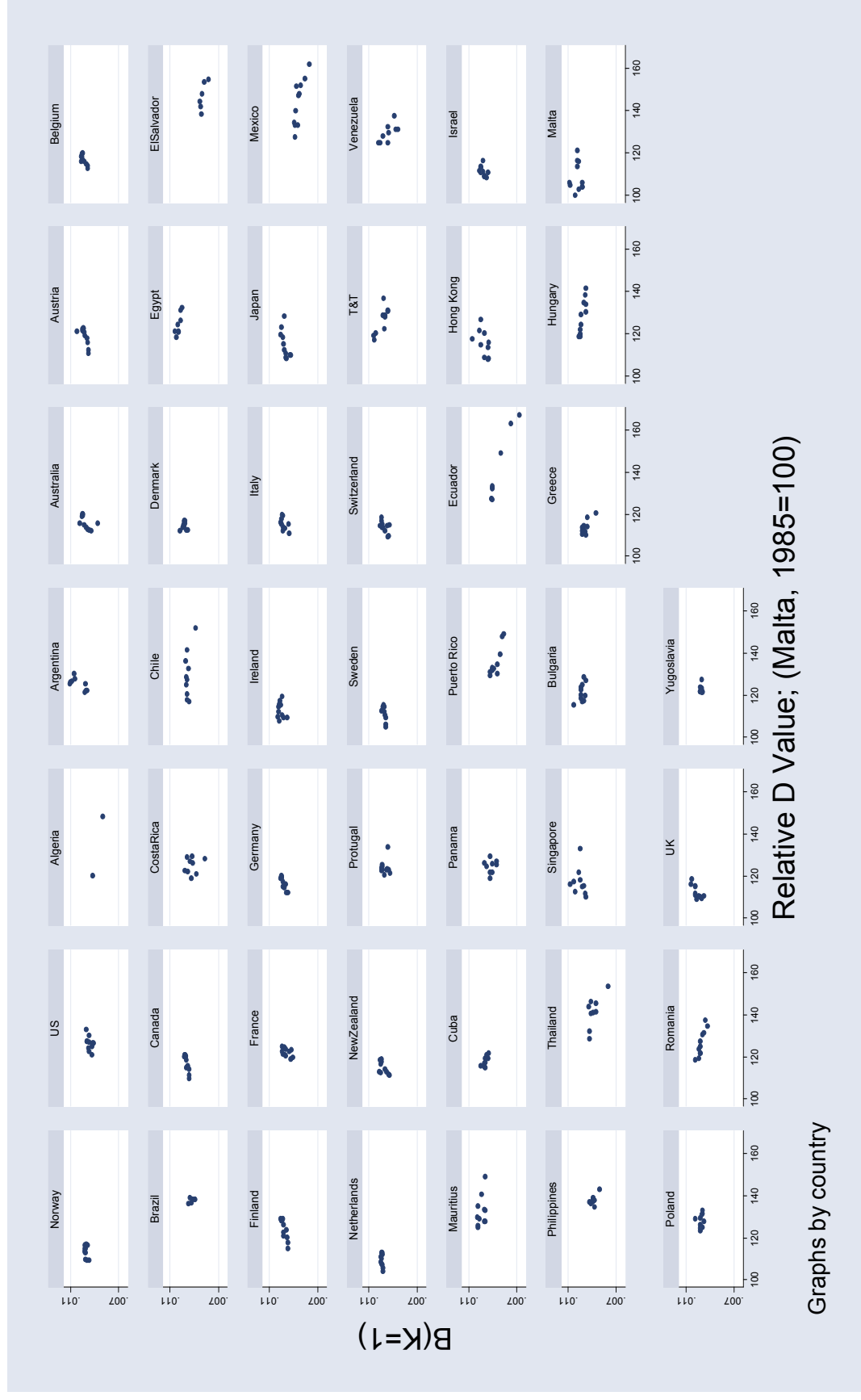
Graphs by country

Relative D Value; (Malta, 1985=100)

B(K=1)

Figure 9. The Correlation of the Attrition Coefficient B and the National Environment Parameter D for Developed and Developing Countries.
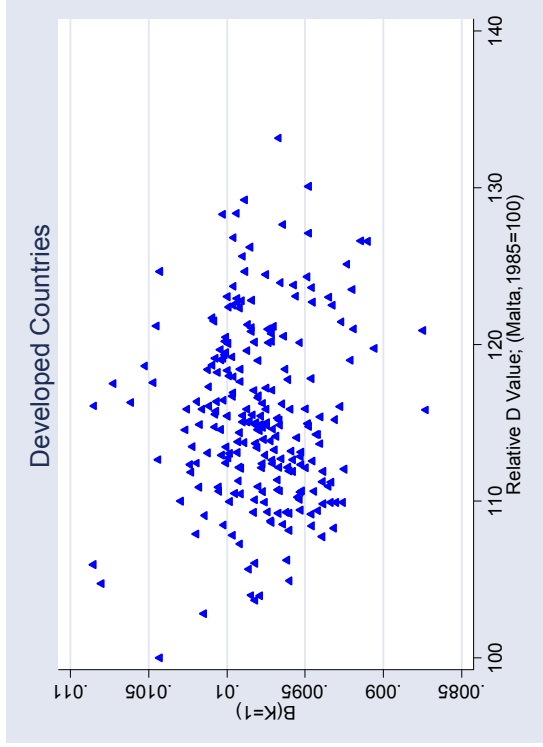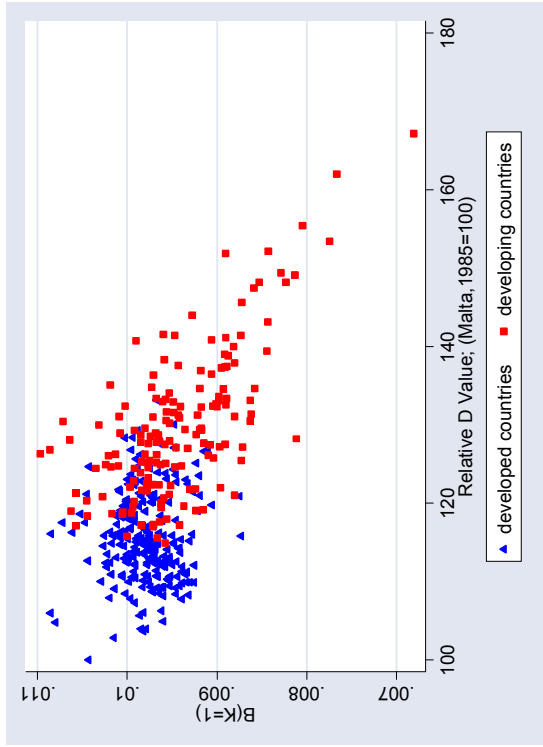
Figure 10. S-M Estimates of Age of Expected Zero Vitality 1/B, 1955-2003.
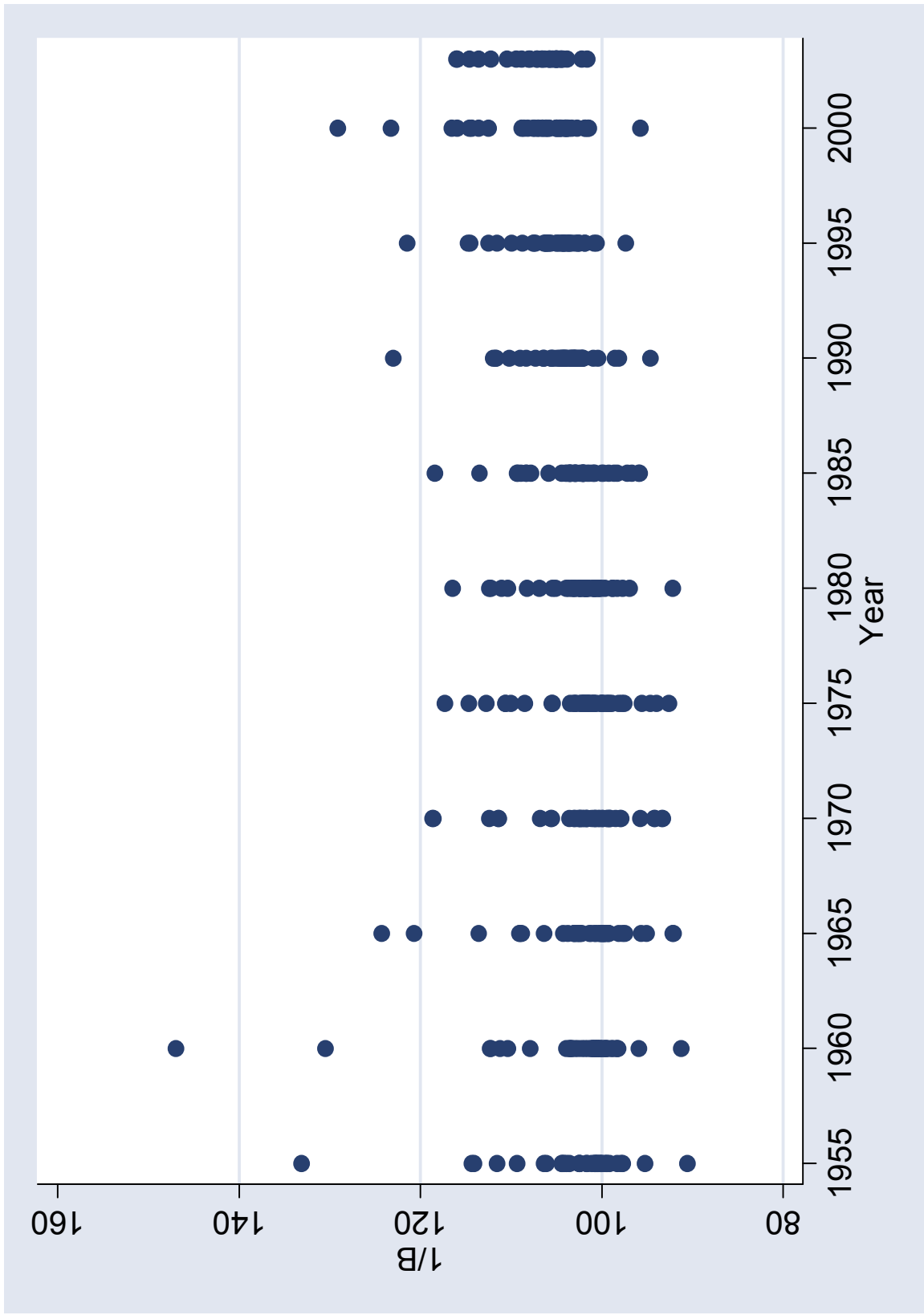
Figure 11. The Trajectory of the Mean 1/B among 46 Countries, 1955-2003.
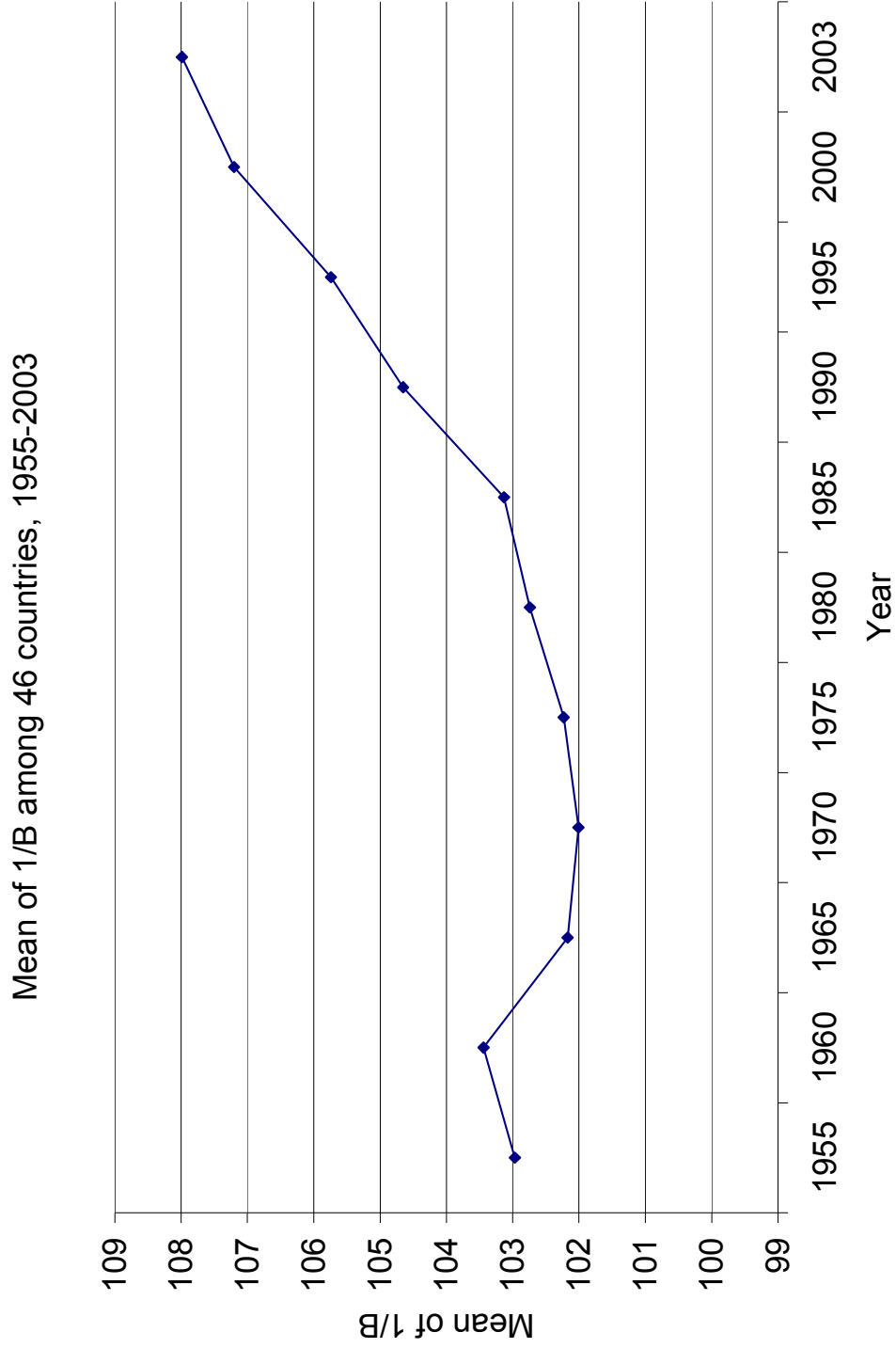
Mean of 1/B among 46 countries, 1955-2003

Figure 12. Three Distinct Longevity Trajectories Estimated by the Semiparametric Group-Based Trajectory Model Expected (Dashed Lines) and Observed (Solid Lines).
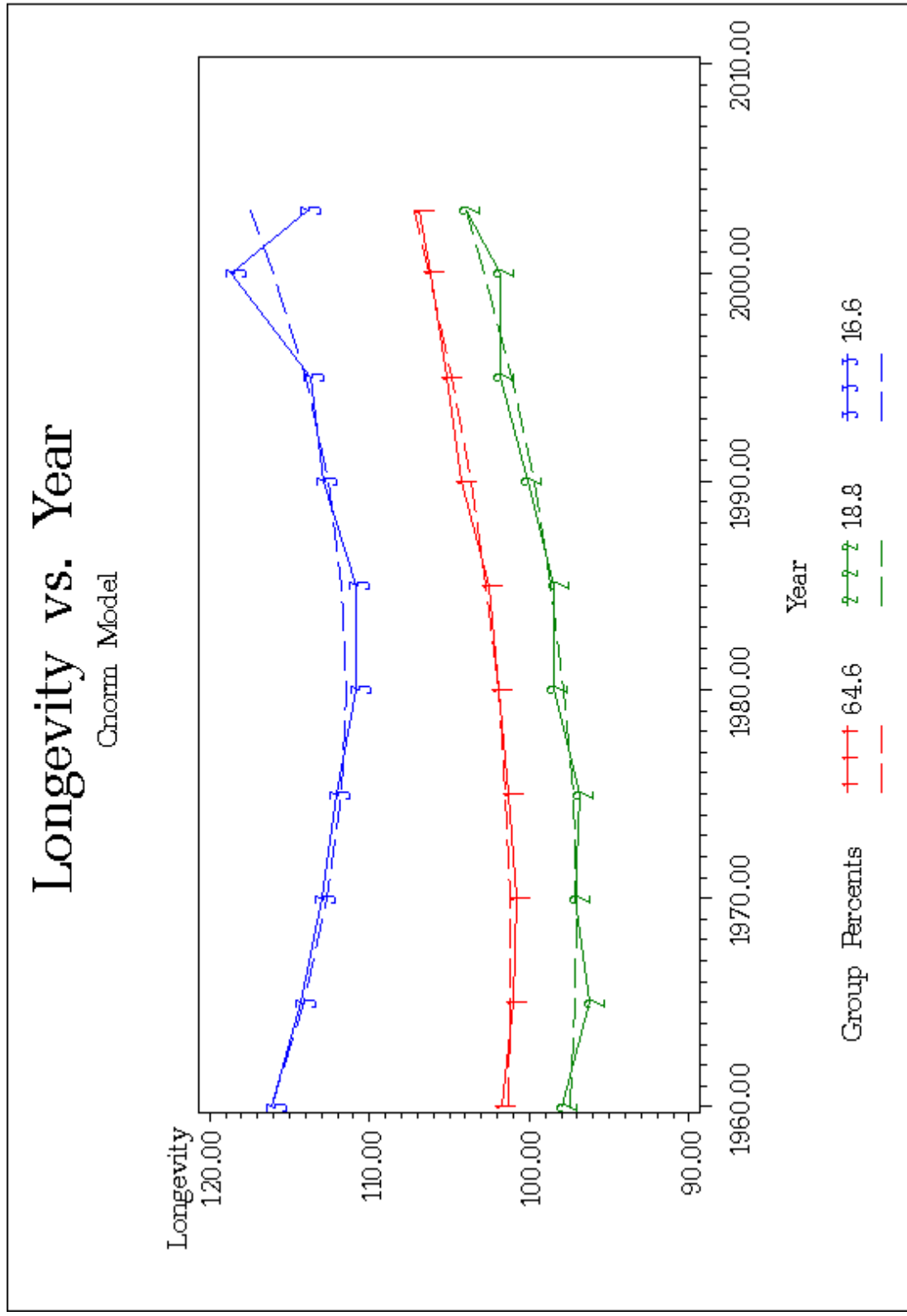

Longevity vs. Year

Cnorm Model

Year

Group Percents

+++ 64.6   ≥≥≥ 18.8   ϡϡϡ 16.6

Figure 13. The Correlation of the Attrition Coefficient B and the National Environment Parameter D for Three Trajectories
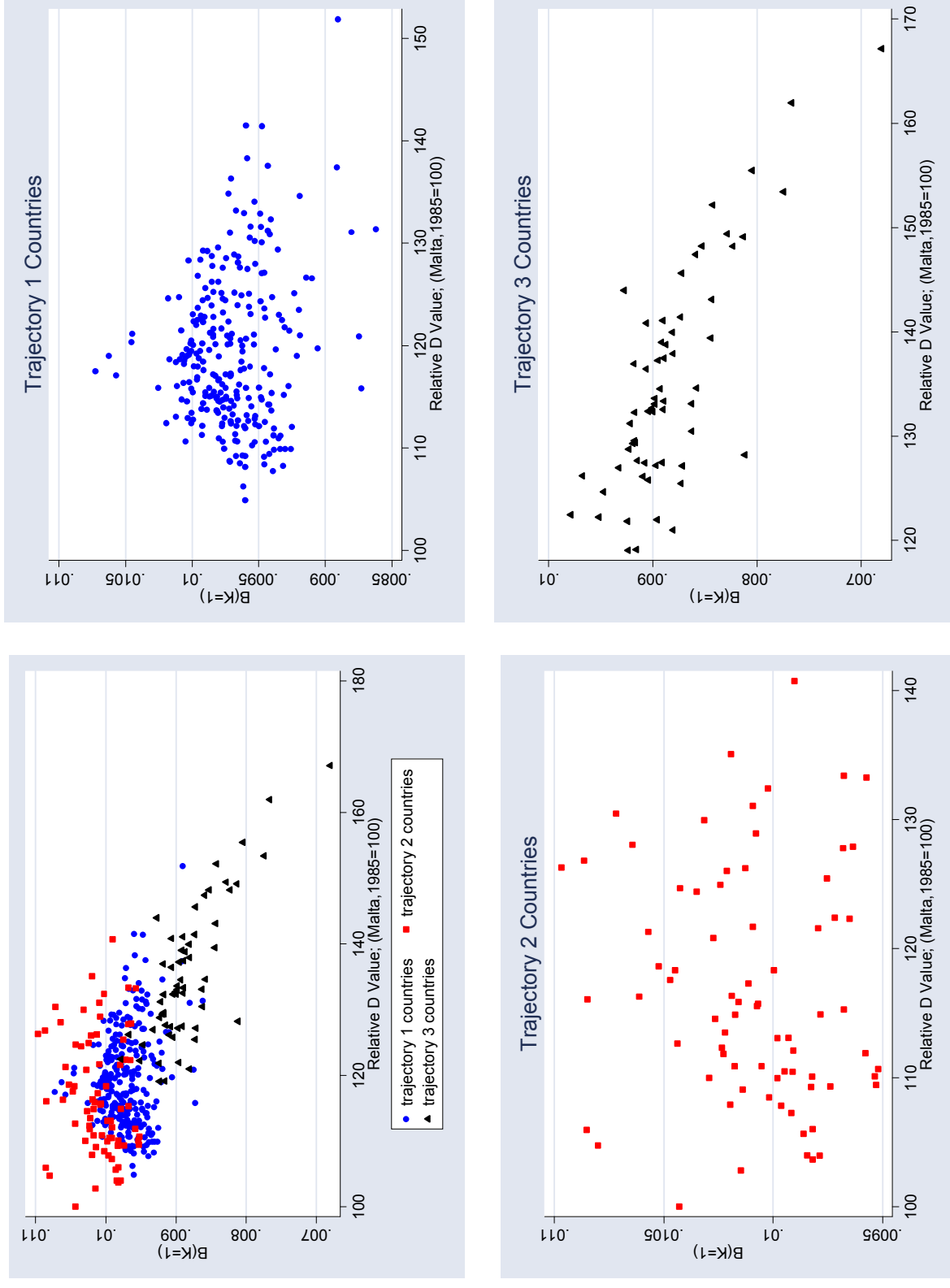
Figure 14. The Correlation of the Logarithm of Initial Mortality Rate LnR_0 and the National Environment Parameter D.
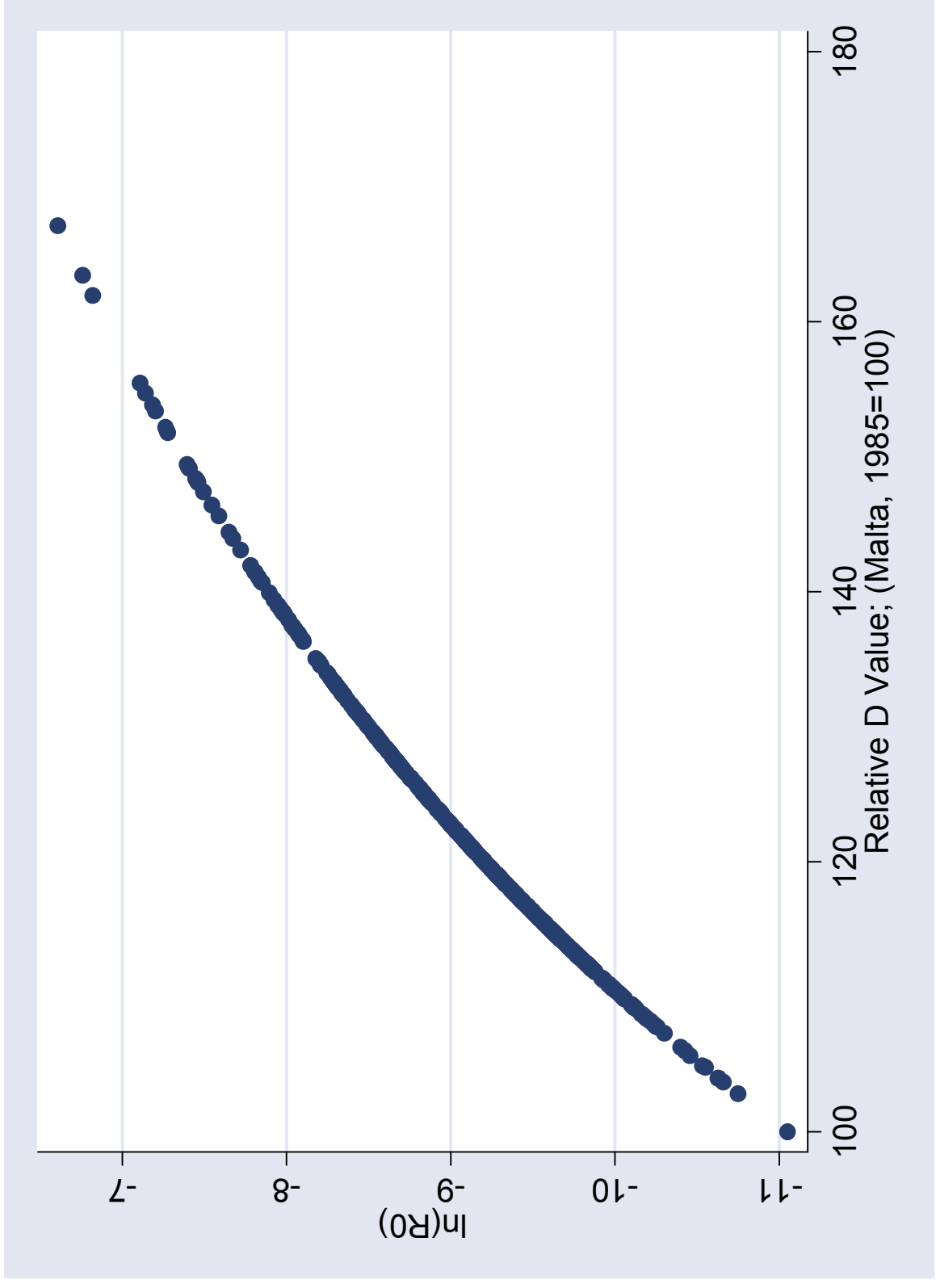
Table 1: Risk Factor Parameter Estimates, Errors, Tests, and p Values.

Trajectory	Parameter	Estimate	Error	Test	p Value
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

Table 2. Random Effects Model: The Determinants of *D*.

Determinants	Model				
	1	2	3	4	5
Year	-.139 *** (.017)	-.139 *** (.017)	-.078 ** (.031)	.051 (.041)	.036 (.045)
Region (Central America and South-East Asia Countries=1, others=0)		11.91 *** (2.394)	12.165 *** (2.772)	8.818 *** (2.632)	10.862 *** (2.665)
Water pollution, food industry (% of total BOD emissions)			-.116 ** (.038)	-.115 ** (.037)	-.134 ** (.046)
GDP per capita (constant 2000 US$)				-.0004 *** (.000)	-.0004 *** (.000)
Population in the largest city (% of urban population)					-.115 * (.047)
N	401	401	189	184	163
Wald chi2	64.07	89.77	34.38	59.31	66.36

Standard errors are in the parentheses.
*** p<.001, ** p<.01, * p<.05