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Long-Run Trends of Human Aging and Longevity

Holger Strulik and Sebastian Vollmer

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Abstract. Over the last 200 years humans experienced a huge increase of life-expectancy. These advances were largely driven by extrinsic improvements of their environment (for example, the available diet, disease prevalence, vaccination, and the state of hygiene and sanitation). In this paper we ask whether future improvements of life-expectancy will be bounded from above by human life-span. Life-span, in contrast to life-expectancy, is conceptualized as a biological measure of longevity driven by the intrinsic rate of bodily deterioration. In order to pursue our question we first present a modern theory of aging and show that immutable life-span would put an upper limit on life-expectancy. We then show for a sample of developed countries that human life-span thus defined was indeed constant until the 1950s but increased since then by about eight years in sync with life-expectancy. In other words, we find evidence for manufactured life-span.

Keywords: human life-span, life-expectancy, aging, compression of mortality, life-span extension.

[†]University of Hannover, Wirtschaftswissenschaftliche Fakultaet, 30167 Hannover, Germany; email: strulik@vwl.uni-hannover.de.
[**]University of Hannover, Institute of Macroeconomics & Harvard School of Public Health, Department of Global Health and Population. Email: svollmer@hsph.harvard.edu.

1. Introduction

For economists human-life span is a given constant. It is the upper bound "T" that we put on top of the integral or sum sign when we compute expected life-time utility. Life-span differs conceptually from life-expectancy. Although we frequently treat life-expectancy, a measure, which depends on the probability to survive from one period to the next, as a constant as well, this is an assumption made "for convenience", since survival depends certainly on the macro-economic environment (GDP per capita, doctors per square kilometer etc) and on individual economic decisions (nutrition, health expenditure etc).

While the majority of economic theory is based on the simplifying assumption that period survival is certain or a given constant (based on Yaari, 1965, and Blanchard, 1985) there exists also a by now rich literature that tries to endogenize survival and to incorporate empirically plausible survival probabilities into economic reasoning. To the best of our knowledge, however, there exists no research in economics on human life-span.[1]

With contrast to life-expectancy, which is population-specific and situation-specific, life-span is usually conceptualized as a *species-specific* characteristic (Arking, 2006, Gavrilov and Gavrilova, 1991). Life-expectancy of a population of mice, for example depends on the specific environment in the wild or in the laboratory. Life-span of mice, in contrast, is independent from such conditions but it differs from the life-span of fruitflies or elephants. Likewise, life-expectancy of a particular human population differs across countries (England vs. Uganda) and over time (England today vs. 200 years ago). Life-span of human beings, however, if it exists, should be invariant across populations. Probably all biologists agree to this notion of life-span. But how to measure life-span is – in contrast to life-expectancy – less easily agreed upon.

Defining human life-span as the maximum attainable age at death, as suggested in many general dictionaries and many older contributions in biology is certainly misleading (Wilmoth, 1999, Carey, 2003). Conceptually this idea is refuted by the insight that "however old we are, our probability to die within the next hour is never equal to one" (Jacquard, 1982). Empirically it has been refuted by the observation that maximum age at death has been continuously on the rise for at least 140 years (Wilmoth and Robine, 2003).

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[1]For economic theory with realistic survival probabilities see, for example, Bütler (2001), Boucekkine et al. (2002), French (2005) Bloom, Canning, and Moore (2007), Bullard and Feigenbaum (2007), Hansen and Imrohoroglu (2008), Heijdra and Romp (2008, 2009), and Kalemli-Ozcan and Weil (2010). A few economic papers, among them Ben Porath (1967), Sunde and Cervellati (2005), and Hazan (2009), could be interpreted as studies on the economic impact of increasing life-span, although they rarely call it so and ignore its biological foundation.

The simple fact that the "sample size" of people who ever lived on earth is continuously rising lets us expect that the maximum ever-observed life-length will rise as time proceeds. This is impressively shown by Finch and Pike (1996). They define life-span T as the estimated age at death of the last survivor of a population such that $S(x) = 1/N$ is the probability to be the last man standing out of N. Plugging $1/N$ into an empirically estimated Gompertzian survival function they obtain a life-span of 105 years for $N = 10^3$ and 114 for $N = 10^7$. In general, the dependence of maxima on sample size is a well-known fact of the statistics of extreme values (Gumbel, 1958).

From these observations it should be clear that life-span, with contrast to life-expectancy, cannot be defined as a mere statistical measure without biological foundation. We need some understanding about the biological mechanism of aging in order to infer an *intrinsic* mechanism that governs the pace at which our bodies deteriorate. Following standard reasoning in modern biology by taking aging and death as stochastic processes (Arking, 2006), we are interested in whether a regularity exists which is common to all humans independently from environmental and genetic characteristics. From this regularity, if it exists, we try to infer life-span not as an absolute maximum but as the characteristic length of life.

We begin our pursuit of human life-span in Section 2 by introducing two very strong empirical regularities, the Gompertz-Makeham law of mortality the compensation effect of mortality. We then introduce a modern theory of aging, and show how it explains these regularities and how it leads to a reasonable conceptualization of human life-span. Quantitatively, thus defined human life-span can be inferred from the compensation effect of mortality. In Section 3 we show how an immutable human life-span would lead to a rectangularization of the survival curve and how it would put an upper bound on future advances of human longevity.

We then re-examine the Gompertz-Makeham law and the compensation effect over the long-run and infer human life-span. We observe a time-invariant compensating effect until the 1950s for all countries in our sample. For the time after the 1950s we find evidence for a secular increase of human life-span. In other words we find that human ingenuity has effectively interfered with nature and has created "manufactured life-time" (Carnes and Olshansky, 2007). Finally we show that life-expectancy in the second half of the last century increased in sync with life-span. To a large and increasing extent we are living longer because technological progress has changed the way we age and the way our bodies decay.

2. Human Life-Span: Theory

2.1. **The Gompertz Makeham Law of Mortality.** All theories of human life-span are based on a very strong empirical regularity, the Gompertz-Makeham law of mortality. It originates from actuary Benjamin Gompertz (1825) who observed that there exists a long period of life, ranging from about 30 to 90 years of age, for which age and mortality are log-linearly related. Let $\mu(x)$ denote the *force of mortality*, that is the conditional probability to die at age x given survival up to age x. The Gompertz law is then formally stated as $\mu(x) = R \exp(\alpha x)$. Noting that not all causes of death are age related, Makeham (1860) added a constant, which provides the famous Gompertz-Makeham formula.

$$\mu(x) = A + Re^{\alpha x}. \tag{1}$$

Taking both simplicity and precision into account, the Gompertz-Makeham formula is to the present day the most appropriate, concise, and widely used formal description of aging (Olshansky and Carnes, 1997). Its parameters are estimated with great precision with correlation coefficients above 0.9 not only for humans but also for species as different as yeast, fruitflies, and horses. The estimated coefficients, of course, differ greatly, reflecting the large variation in life-span across species (Arking, 2006).

Using the Gompertz-Makeham law and solving $\dot{S}(x)/S(x) = -\mu(x)$ we obtain the unconditional probability to survive to age x. Given that $S(0) = 1$ we arrive at (2).

$$S(x) = \exp\left(-Ax - \frac{R}{\alpha}\left(\exp(\alpha x) - 1\right)\right). \tag{2}$$

From $S(x)$ we can infer life-expectancy (expected remaining years to live) at age x as $L(x) = \int_x^\infty S(a)\mathrm{d}a/S(a)$.

Over the last century human life expectancy at birth increased by more than 20 years in most of the fully developed countries (Riley, 2001). It is interesting to investigate how these huge improvements of human longevity are captured by the Gompertz-Makeham law. For this purpose it is helpful to isolate the Makeham-parameter A because it reflects age-unrelated forces of mortality, i.e. background mortality (Bongaarts, 2005) or extrinsic mortality (Carnes and Oshansky, 2007). We expect prevention, eradication, or cure of age-unrelated diseases to be

reflected in changes of A. In contrast, any progress with respect to the aging process itself would be reflected in a change of the age-dependent Gompertz-parameters R and α.

Over the last two centuries background mortality went down dramatically in the today fully developed countries (for Sweden, for example, from $5.5 \cdot 10^{-3}$ to $4.8 \dot{1} 0^{-4}$; Gavrilov and Gavrilova, 1991). With A being close to zero, future advances in life-expectancy will have to come from improvements of age-dependent mortality. In other words, if technological progress could affect only background mortality but not the intrinsic rate of bodily decay, the observed trend of improving life-expectancy at birth by about 3 month per year of birth (Oeppen and Vaupel, 2002) would not be sustainable in the future. Inspired by this fact some gerontologists have concluded that future life-expectancy at birth will unlikely exceed 85 years (Fries, 1980, Carnes and Olshansky, 2007). The problem with such a hypothesis is that – although the virtually zero background mortality leaves no scope for improvement – we cannot (yet) see any slowdown or convergence of life-expectancy in the data (Wilmoth, 1997, 1999). This means that recent advances of life-expectancy must have originated from a change of the Gompertz parameters.

2.2. **The Compensation Effect of Mortality.** The Gompertz-parameters α and R, which are estimated with high precision for a *given* population, differ actually across sexes, across countries, and over time. This means that, while all humans seem to age according to a common general law of mortality, the specific parameters governing this law depend on sex, provenance, and year of birth. Furthermore, R and α seem to depend also on geography and tend to fall (in the case of R) or rise (in the case of α) with economic development. Because of their instability the Gompertz-parameters as such are thus not suitable to identify human-life span.

The Gompertz parameters, however, are not changing independently from each other but, strikingly, in a specific way such that they preserve an inverse association between α and the log of R. This fact is known as the Strehler-Mildvan correlation or the compensation effect of mortality (Strehler and Mildvan, 1960, Gavrilov and Gavrilova, 1991). It is exemplarily shown for Sweden in Figure 1 and 2. Figure 1 shows the long-run trends for α and R. Over time, the slope parameter α tends to rise and the level parameter R tends to fall. This means that over time Swedish women tend to age at a faster speed but start out at lower initial mortality. Below we show the generality of this phenomenon across countries and how it can be explained by increasing initial redundancy in the reliability theory of aging.

FIGURE 1. Aging Trends for Swedish Females 1751 - 2005
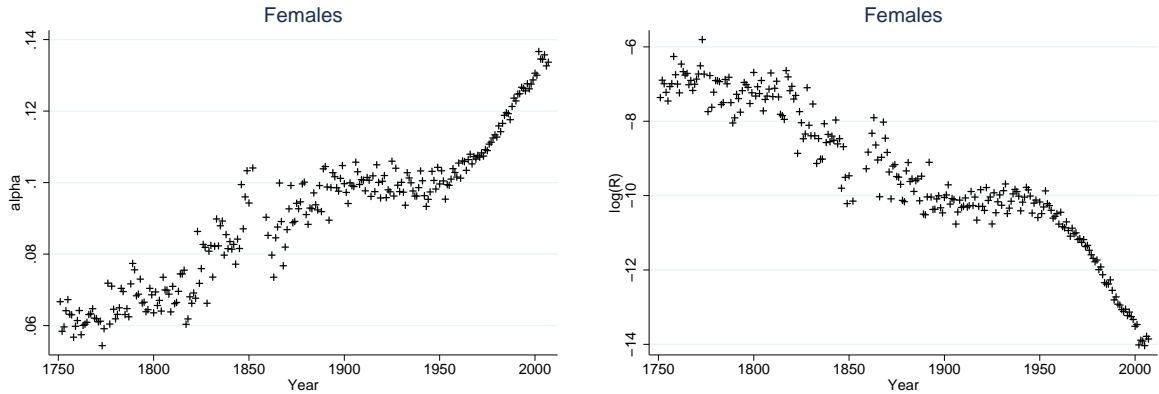
Left panel: Development of α over time. Right panel: Development of R over time.

Formally, the compensation effect of mortality states that

$$\log R_{it} = \log M - \alpha_{it} \cdot T \tag{3}$$

in which R_{it} and α_{it} are the population- and year-specific parameters of the Gompertz law and M and T are invariant parameters of the compensation effect of mortality. Figure 2 shows the correlation for Swedish females and males.

To see how the compensation effect inspired a general theory of human aging insert (4) into country- and sex-specific age-dependent mortality $\mu_i^x \equiv \mu(x) - A = R_i \exp(\alpha_i x)$ to obtain

$$\mu^x = M \mathrm{e}^{\alpha(x-T)}. \tag{4}$$

Observe that age-dependent mortality exhibits a fixed point. Equation (4) predicts that all men (and likewise all women) of a country's population share a common force of mortality M at age T independently from the year or century of birth. In other words, any improvement in the initial force of mortality μ^0 is compensated by a faster increase of μ^x with age. The final step in the derivation of human life-span is to conclude that T is approximately constant across countries. This has been found by Gavrilov and Gavrilova (1991) for 209 human life-tables. They thus interpret the focal point (T, M) as a *species-specific* constant. For humans the point estimate was $T = 95$ years, identified by Gavrilov and Gavrilova as "the life-span of human beings".

FIGURE 2. The Compensation Effect of Mortality in Sweden 1751 - 2005
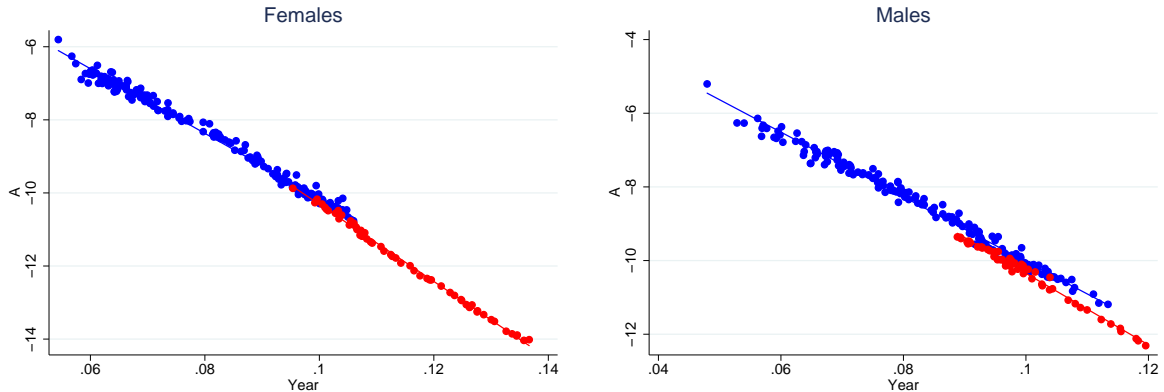
Correlation between $\log(R)$ and α for males (right) and females (left). Observations before 1950 are marked blue, and observations after 1950 are marked red.

The difference between the Strehler-Mildvan correlation and the compensation effect is that Strehler and Mildvan (1960) in their original approach inferred the correlation from the Gompertz law, without the Makeham amendment. In order to infer life-span properly, however, the Gompertz parameter have to be derived from the Gompertz-Makeham law, with explicit consideration of background mortality A. Otherwise, results would be biased and changes in the extrinsic rate of mortality would be absorbed by the parameters governing the intrinsic rate of mortality. This has been impressively demonstrated by Gavrilov and Gavrilova (1991) who called the unbiased correlation the "compensation effect of mortality". Controlling for A is of particular importance when background mortality is high, that is in the analysis of historical data and of contemporaneous less developed countries.

2.3. **Reliability Theory.** The strong regularities identified by Gompertz law and the compensation effect suggest that all humans share a common mechanism of aging, a common stochastic process according to which individual bodies lose function over time and bodily failures and impairments are accumulated. The pursuit to rationalize these phenomena has inspired an application of reliability theory (Barlow and Proschan, 1975) and produced the modern theory of aging. Its main idea is based on redundancy within the human body which is depleted over time. This notion of aging as accelerated loss of organ reserve is in line with the mainstream view in the medical science. For example, initially, as a young adult, the functional capacity of human organs is estimated to be tenfold higher than needed for survival. (Fries, 1980). It accords also well with the modern view in biology, which conceptualizes aging and death as

driven by accidental stochastic shocks on the molecular level (Arking, 2006). Here we sketch a basic theory by Gavrilov and Gavrilova (1991, 2001) and refer to their work as well as to Novoltsev (2006), Finkelstein (2008) and Milne (2008) for extensions.

A common characteristic of all reliability-based models is that organisms are conceptualized as complex systems consisting of essential parts (e.g. organs, tissue) connected in series, which are in turn built of smaller entities connected in parallel. Parallel connectivity means that every reliability theory is built upon the idea of redundancy. Another common theme is a stochastic failure rate for the basic entities. The notion of aging as driven by a "natural" stochastic process helps to explain the "unfair" nature of human fate, i.e. why we actually observe large differences of aging on the individual level. Reliability theory can explain why individuals raised under equal conditions and/or built from the same genes (monozygotic twins) can age and eventually die in very different ways. At the same time the model provides a toehold to explain how population- (e.g. country-) specific characteristics and the environment early in life have a bearing on aggregate aging behavior of entire populations and/or sub-populations.

Suppose an organism consists of m irreplaceable blocks, i.e. blocks are connected in series such that the organism dies if one block fails. Each block consists of n elements, connected in parallel with age-independent failure rate λ. The probability for a block to expire before age x is thus given by $F(x) = [1 - \exp(-\lambda x)]^n$. Suppose that many elements are initially defect. The probability of an initially functioning element is given by q. It can then be shown that the failure rate of the organism (i.e. the mortality rate) is approximately given by (5).

$$\mu(x) \approx R e^{\alpha \cdot x} \quad \text{where} \begin{cases} R & \equiv mc\lambda k \exp(-k) \\ \alpha & \equiv k\lambda. \end{cases} \tag{5}$$

in which $k \equiv nq$ is the mean number of initially functioning elements and c is a constant to provide a unitless result. The model thus explains Gompertz' law. Taking log's of R we get $\log R = \log(cmk\lambda) - k$ and inserting $\alpha = k\lambda$ we arrive at $\log R = \log M - \alpha T$, with $M \equiv \alpha mc$ and $T \equiv 1/\lambda$. The model thus explains as well the compensation effect of mortality.

For an interpretation of the result note that T is uniquely pinned down by λ, the age-independent failure rate of an element. If λ is a species-specific constant, then the model supports a unique focal point T, i.e. a species-specific life-span. Across species, T depends inversely on the robustness of its non-aging elements. Let's reasonably assume that m, the number

of irreplaceable blocks, is also a species-dependent constant. Then all variation within a species results from variation of $k = nq$, the mean number of initially functioning elements. Suppose that available nutrition (for mother and child) and disease exposure early in life have shaped k. Taking the historic improvement in nutrition and health into account, the model then predicts that with ongoing economic development people start out much better at young age but are aging faster. Consequently, survival prospects and life-expectancy have improved at any age up to age T.

In other words, a time-invariant compensation effect of mortality would imply that better health care and nutrition or, more generally, improvements of the economic environment through technological progress have increased life-expectancy "only" through improving somatic redundancy (for example, more healthy body cells; Fogel. 1994; Fogel and Costa, 1997). In order to improve life-span T technological progress needs to have a bearing on λ, that is on the intrinsic rate of bodily decay.

3. Implications of Time-Invariant Human Life-Span

3.1. **Compression of Morbidity.** The notion of a constant life-span T is sometimes expressed as "rectangularization" or as "compression of morbidity" (Fries, 1980). These concepts are illustrated in Figure 3. The left panel shows an improvement of the age-dependent force of aging according to the Strehler-Mildvan correlation. The slope parameter α increases from 0.08 (blue line) to 0.11 (red line). This is about the actually observed average increase for women during 20th century in our sample of countries. The level parameter R is assumed to adjusts in a way that supports a fixed point at $T = 83$.

The middle panel shows the implied unconditional survival rate $S(x)$ and conveys the idea of rectangularization. Compared to the beginning of the century a higher share of women reaches an old age of, say 70 years, and expires then more quickly during their last years before death. Visually, the survival curve becomes closer to rectangular over time. This implies that gains in life-expectancy, for example brought forward by technological progress in medicine, decrease with age as well, as demonstrated in the panel on the right hand side of Figure 3. Improvements in health at earlier stages of life lead to a faster deterioration in old age, a compression of morbidity, such that women reaching an age of T share the same force of mortality at the beginning and at the end of the century.

8

FIGURE 3. Aging under Compensation of Mortality
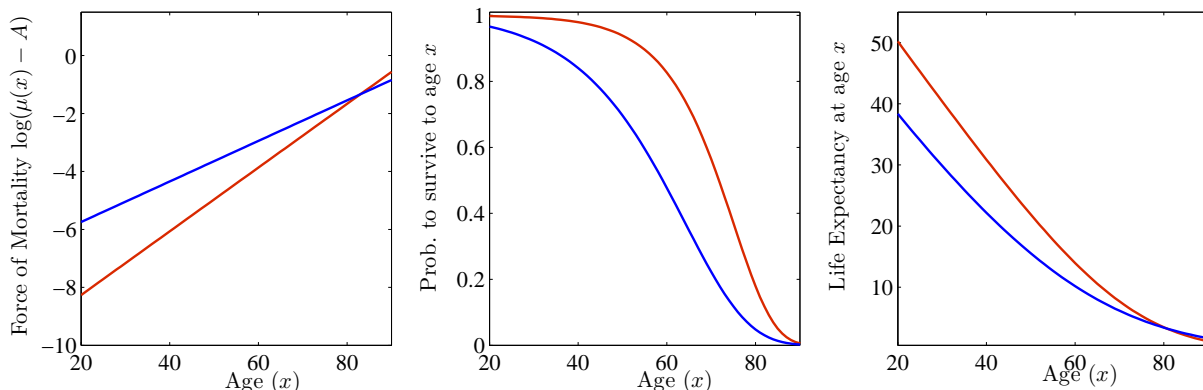
$A = 0.002$, $\alpha = 0.08$ (blue lines) and $\alpha = 0.11$ (red lines), $M = \exp(-0.07)$, $R = M\exp(-\alpha \cdot T)$, $T = 83$.

For comparison, Figure 4 shows the effect on survival and life-expectancy caused by an improving level parameter R without compensation effect. The slope parameter α is hold constant. In this case the curves shift to the right at all ages, indicating that aging as such has been postponed at all ages and providing a visible increase of life-expectancy also for the old (see also Vaupel, 2010). In terms of theory, the inherent failure rate of the elements of which humans are constructed λ has been manipulated. Human life-span T increased.

FIGURE 4. Manufactured Life-Span
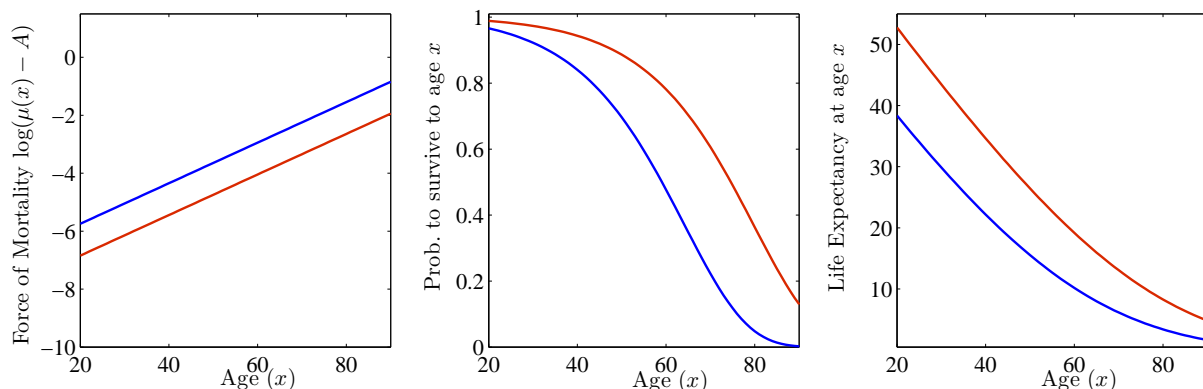
$A = 0.002$, $\alpha = 0.011$, $R = 2.7 \cdot 10^{-5}$ (blue lines) and $R = 0.9 \cdot 10^{-5}$ (red lines).

3.2. **The Future of Longevity.** In this section we show that if the compensation effect of mortality is time-invariant and M and T are species-specific constants, then future gains of life-expectancy are bounded from above. This means that the notion of "broken limits to life-expectancy" (Oeppen and Vaupel, 2002) inferred from extrapolation of past trends would be ill-considered. Actually there would exist a certain limit to life-expectancy. In order to

9

show this, we insert the compensation effect of mortality, $R = M \exp(-\alpha T)$, into into (1). Assuming that A is zero (which is reasonable approximation for contemporaneous fully developed countries) we get the force of mortality $\mu = M \exp[\alpha(x - T)]$. The implied survival at age x is $S(x) = \exp(y) \exp\left[-M/\alpha \exp(\alpha(x - T))\right]$ with $y \equiv M/\alpha \exp(-\alpha T)$. For analytical simplicity we focus on life expectancy at birth, which is given by (6).

$$L(0) = \exp(y) \int_0^\infty \exp\left[-M/\alpha \exp(\alpha(x - T))\right] \mathrm{d}x. \tag{6}$$

Substituting $u = M/\alpha \exp[\alpha(x - T)]$ into (6) provides the simplification (7).

$$L(0) = \exp(y)\frac{1}{\alpha} \int_y^\infty \exp(-u)\frac{1}{u}\mathrm{d}u = \exp(y)\frac{1}{\alpha}\Gamma(0, y) \tag{7}$$

in which Γ is the incomplete gamma function. Applying the mean value theorem of integral calculus we know that there exists a ξ with $y < \xi < \infty$ such that

$$\int_y^\infty \exp(-u)\frac{1}{u}\mathrm{d}u = \frac{1}{\xi} \int_y^\infty \exp(-u)\mathrm{d}u = \frac{1}{\xi} \exp(-y).$$

Thus, $L(0) = 1/(\alpha\xi)$, implying, since $y < \xi < \infty$ that $L(0) < 1/(\alpha y)$. This constraint turns out to be very useful for the "curve discussion" of life expectancy. Taking the derivative of life-expectancy with respect to life-span we obtain

$$\frac{\partial L(0)}{\partial T} = \left[L(0) + \frac{\exp(y)}{\alpha}\frac{\partial \Gamma}{\partial y}\right]\frac{\partial y}{\partial T} = \left[L(0) - \frac{1}{\alpha y}\right] \cdot (-\alpha y) > 0 \tag{8}$$

because $L(0) < 1/(\alpha y)$. That is, life-expectancy and life-span are positively correlated. Analogously we obtain $\partial L(0)/\partial M < 0$.

If the compensation effect of mortality is time-invariant and M and T are constants, the only way to improve life-expectancy is through α. In order to obtain the effect of α on $L(0)$ we compute

$$\frac{\partial L(0)}{\partial \alpha} = -\frac{L(0)}{\alpha} + \left[L(0) - \frac{1}{\alpha y}\right]\frac{\partial y}{\partial \alpha} = -\frac{L(0)}{\alpha} - \left(\frac{1}{\alpha} + T\right)yL + \frac{1}{\alpha}\left(\frac{1}{\alpha} + T\right).$$

The sign of the derivative is ambiguous and there exists an extremum for $\alpha = \alpha^*$ where $\partial L/\partial \alpha = 0$, that is at life-expectancy

$$L(0, \alpha^*) = \frac{\frac{1}{\alpha} + T}{1 + y + \alpha yT}. \tag{9}$$

After some algebra, the second derivative is obtained as

$$\frac{\partial^2 L}{\partial \alpha^2}_{|\alpha^*} = \frac{1}{\alpha^3} \cdot \frac{1}{1+y+\alpha yT} \cdot \left[y\alpha T(1+\alpha T)^2 - 1 \right].$$

From this we conclude that the extremum is a maximum if

$$(1+\alpha T)^2 \leq \frac{1}{\alpha yT} = \frac{1}{MT}e^{\alpha T} \quad \Rightarrow \quad eMT \cdot (1+\alpha T)^2 < e^{1+\alpha T}.$$

Since the slope of the exponential function is generally steeper than the slope of the quadratic, we conclude that for sufficiently large α there exists a maximum life expectancy. This in turn means that life expectancy is effectively bounded from above by life-span. Since y converges very rapidly to zero with rising α, we expect maximum life-expectancy to be about $T + 1/\alpha^*$.

FIGURE 5. Life-Expectancy is Bounded by Life-Span
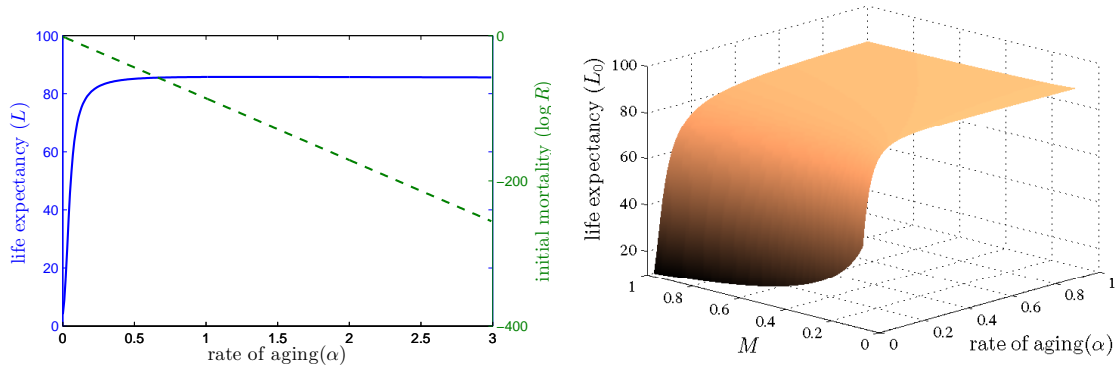
Left panel: Life expectancy (blue solid line) and initial mortality $\log R$ under the compensation effect of mortality (3) (green dashed line) for alternative α and $M = 0.26$. The – almost indiscernible – maximum life-span is assumed for $\alpha = 1.27$. Right panel: life-expectancy for alternative values of α and M. Both panels: lifespan $T = 85$.

Figure 5 visualizes these results for life-span $T = 85$. As the force of mortality α increases, the logarithm of initial mortality $\log R$ declines according to the compensation effect (3). This is shown by the dashed green line in Figure 5, assuming that $M = 0.26$. Accordingly life-expectancy at birth increases steeply for small α and reaches an almost indiscernible maximum of 85.78 years at 1.27. Afterwards life-expectancy converges towards T from above (blue solid line). The panel on the right hand side demonstrates robustness of this result by – hypothetically – considering alternative survival rates M at age T. Life-expectancy is effectively bounded by life-span, implying that "broken limits to life-expectancy" are inconsistent with a time-invariant

11

compensation effect. In order to allow for "unlimited" life-expectancy, life-span of humans must be modifiable.

4. Empirical Evidence

In this section we use the established methodology and investigate whether human life-span is constant or modifiable. For that purpose we use data on 1×1 period death rates from the Human Mortality Database. 1×1 means that the data come for single years of age in one year time intervals. The Human Mortality Database contains detailed population and mortality data for 37 countries (there are multiple data series for France, Germany, New Zealand and the United Kingdom). The period of data coverage differs from country to country. For Sweden, for example, data is available way back to 1751, while for Chile data coverage starts only in 1992. We confine our analysis to countries that have full data coverage from 1900 through 1999 (12 countries) or 1950 through (26 countries). This way we ensure a consistent sample and that our results are not due to changes in the sample composition.

TABLE 1. Slope Parameter α across Countries and Time

Country	1900-24 male	1900-24 female	1925-49 male	1925-49 female	1950-74 male	1950-74 female	1975-1999 male	1975-1999 female
Australia					0.077	0.096	0.090	0.111
Austria					0.086	0.101	0.095	0.118
Belgium	0.086	0.090	0.089	0.093	0.088	0.101	0.089	0.116
Bulgaria					0.075	0.083	0.087	0.100
Canada					0.084	0.099	0.088	0.109
Czech Republic					0.085	0.099	0.084	0.106
Denmark	0.098	0.093	0.094	0.094	0.093	0.104	0.087	0.111
England & Wales	0.078	0.081	0.085	0.091	0.082	0.098	0.086	0.106
Finland	0.090	0.094	0.077	0.089	0.084	0.098	0.086	0.113
France	0.089	0.088	0.088	0.096	0.089	0.106	0.099	0.125
Germany					0.087	0.102	0.091	0.114
Hungary					0.091	0.098	0.084	0.103
Ireland					0.088	0.093	0.084	0.102
Italy	0.084	0.075	0.095	0.095	0.093	0.101	0.094	0.119
Japan					0.086	0.099	0.101	0.124
Netherlands	0.088	0.088	0.093	0.093	0.094	0.104	0.087	0.118
New Zealand	0.077	0.084	0.089	0.092	0.082	0.100	0.086	0.108
Northern Ireland					0.083	0.091	0.083	0.103
Norway	0.100	0.100	0.100	0.102	0.095	0.106	0.092	0.119
Portugal					0.088	0.098	0.092	0.111
Scotland	0.078	0.081	0.085	0.090	0.082	0.094	0.081	0.099
Slovakia					0.094	0.101	0.084	0.106
Spain					0.086	0.097	0.096	0.120
Sweden	0.101	0.100	0.099	0.099	0.096	0.104	0.098	0.120
Switzerland	0.084	0.082	0.086	0.090	0.092	0.105	0.100	0.127
United States					0.079	0.097	0.086	0.105
Average	**0.088**	**0.088**	**0.090**	**0.094**	**0.087**	**0.099**	**0.090**	**0.112**

4.1. **The Gompertz Parameters.** We begin with estimating the parameters of the Gompertz-Makeham equation (1) by gender for all countries and years with the method of non-linear least squares. It is well known from the literature that the Gompertz-Makeham equation does not hold for young and very old people. We thus restrict the sample to ages 30 through 90. Both Gompertz parameters are estimated with very high precision. The R^2 is around 0.99 or higher in all regressions. Figure A.1 in the appendix shows the distribution of the estimated parameters divided by their respective standard errors. There is not a single case of an insignificant α parameter. In the 1950-1999 sample, we lose one country-year observation due to an insignificant estimate of the R parameter for Finland. In the 1900-1999 sample, we lose 18 country-year observations due to insignificant estimates of the R parameter for Finland and New Zealand.

TABLE 2. Level Parameter $\log(R)$ across Countries and Time

	1900-24		1925-49		1950-74		1975-1999	
Country	male	female	male	female	male	female	male	female
Australia					-8.24	-10.15	-9.58	-11.71
Austria					-8.88	-10.34	-9.79	-12.06
Belgium	-8.71	-9.16	-9.06	-9.53	-9.12	-10.41	-9.35	-11.99
Bulgaria					-8.18	-8.90	-9.02	-10.27
Canada					-9.00	-10.47	-9.44	-11.66
Czech Republic					-8.71	-10.06	-8.68	-10.85
Denmark	-9.89	-9.58	-9.62	-9.67	-9.67	-10.74	-9.21	-11.64
England & Wales	-8.11	-8.54	-8.68	-9.47	-8.55	-10.27	-9.10	-11.19
Finland	-9.04	-9.47	-8.06	-9.18	-8.62	-9.96	-9.07	-11.73
France	-8.93	-9.03	-8.92	-9.73	-9.22	-10.95	-10.30	-12.97
Germany					-8.98	-10.39	-9.50	-11.74
Hungary					-9.23	-10.03	-8.72	-10.63
Ireland					-9.07	-9.75	-8.87	-10.72
Italy	-8.54	-7.85	-9.52	-9.59	-9.54	-10.44	-9.81	-12.28
Japan					-8.82	-10.20	-10.50	-12.84
Netherlands	-9.03	-9.08	-9.50	-9.60	-9.77	-10.73	-9.24	-12.30
New Zealand	-8.29	-8.94	-9.20	-9.64	-8.67	-10.46	-9.21	-11.42
Northern Ireland					-8.64	-9.59	-8.81	-10.83
Norway	-10.21	-10.35	-10.27	-10.52	-9.88	-10.93	-9.71	-12.35
Portugal					-9.03	-10.09	-9.62	-11.46
Scotland	-8.14	-8.53	-8.65	-9.30	-8.50	-9.80	-8.56	-10.49
Slovakia					-9.58	-10.33	-8.77	-10.89
Spain					-9.01	-10.14	-10.11	-12.44
Sweden	-10.22	-10.17	-10.03	-10.14	-9.88	-10.72	-10.19	-12.45
Switzerland	-8.46	-8.42	-8.77	-9.26	-9.47	-10.71	-10.37	-13.08
United States					-8.51	-10.34	-9.30	-11.29
Average	**-8.963**	**-9.095**	**-9.190**	**-9.635**	**-9.029**	**-10.265**	**-9.416**	**-11.665**

The parameter estimates for α and $\log(R)$ are reported in Tables 1 and 2. For better visualization we break the data down into 25-year periods and report period averages. The overall trend shown in Section 2 for Sweden is clearly visible across all countries: α increases and R

decreases over time. There are also interesting gender differences: α is higher for females than for males, whereas R is higher for males than for females. On average, α increased for females from 0.088 in 1900-24 to 0.112 in 1975-99. For males the increase is more modest, starting from the same level in 1900-24 and reaching 0.090 in 1975-99. For females R decreased by more than one order of magnitude from $\exp(-9.95) = 1.1 \cdot 10^{-4}$ in 1900-24 to $\exp(-11.66) = 8.5 \cdot 10^{-6}$ in 1975-99. Again, the decrease is somewhat more modest for males. Summarizing, across all countries over the last century the trend shows that people become more healthy in young age but are aging faster as they get older.[2]

4.2. **Survival Probability.** We next use the average parameters of the Gompertz equation to compute the unconditional probability to survive to age x.

$$S(x) = \exp\left(-\frac{R}{\alpha}\left(\exp(\alpha x) - 1\right)\right). \tag{10}$$

Figure 6 shows the survival functions for average females and males in 1900-24 (solid lines) and 1975-99 (dashed lines). Two salient phenomena can be observed. First, a rectangularization or compression of morbidity is clearly visible. In 1900-24 the decline of the survival probability starts already around age 20 whereas in 1975-99 survival probability stays close to unity until around age 40 and then declines more rapidly than in the first quarter of the century. Second, in addition to the rectangularization of the survival function, we observe also that the survival function shifts to the right from period 1900-24 to 1975-99. The right shift is not uniform across all ages, but clearly very old people are benefitting from improved survival probabilities as well. As explained in Section 2 this finding is inconsistent with a time-invariant compensation effect of mortality. It is a first indication that human life span might be modifiable.

In Figure A.2 in the appendix we redo the analysis country by country. We take the period averages of α and R for the first available period and compare the obtained survival function to the survival function in 1975-99 for each country. It turns out that the pattern that we have

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[2]In a related study, Bongaarts (2005) estimates the parameters of the Gompertz law for 14 countries in the period 1950-2000 and finds a "nearly constant" slope parameter (α in our notation) and a secularly falling level parameter (R in our notation). It is tempting to conclude from Bongaarts' finding of decreasing R that human life-span has risen over the last half of the 20th century. On a closer look, however, this evidence is insufficient for a conclusion on human life span. Firstly, α and R are not species-specific parameters, they vary across countries and sexes. Secondly, it is misleading to compare α and R at the same (absolute) scale. Inspection of (1) shows that a unit change of α contributes as much to the force of mortality as a unit change of the logarithm of R.

FIGURE 6. Survival Function 1900-24 and 1975-99
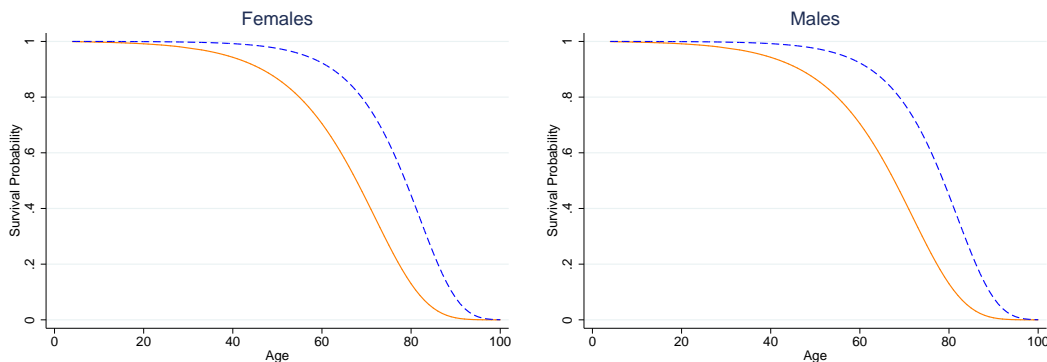
Dashed blue line: Survival function 1975-1999. Solid orange line: Survival function 1900-25.

illustrated for averages – rectangularization cum right shift of the survival function – is universal across all countries.[3]

4.3. **Human Life-Span.** The observations from the previous section suggest that life span T according to the definition from the compensation effect of mortality is not constant but changing over time. We substantiate this finding by estimating the compensation effect. In the model

$$\log(R_{it}) = \beta_0 + \beta_1 \cdot \alpha_{it} + \epsilon_{it}. \tag{11}$$

β_1 represents the negative life span $-T$ and β_0 represents $\log(M)$, the mortality rate shared by all humans of age T. Exploratory data analysis in Figure A.3 in the appendix suggests that the slope coefficient T indeed changes over time. Pooling the observations over a too long period would lead to misleading results. We thus focus on the 26 countries, for which we have a full data from 1950-1999, and estimate equation (11) with pooled OLS for 1950-74 and 1975-1999 respectively. The result is visualized in Figure 7. Red circles identify data points in the 1950-74 period and blue circles reflect the 1975-99 period. It is clearly visible that in the later period an improvement of $\log(R)$ was associated with a somewhat smaller increase of α than in the earlier period, implying that the slope – representing human life span – increased in absolute value during the 20th century.

Table 3 reports the estimation results. During the second half of the 20th century female life span increased from 89 years to about 96 years and male life span increased from 83.5 years

[3]Yashin et al. (2001) arrive at a similar conclusion using period and cohort data for a smaller sample of countries (France, Japan, Sweden and the US).

FIGURE 7. Strehler-Mildvan Correlation 1950-1999
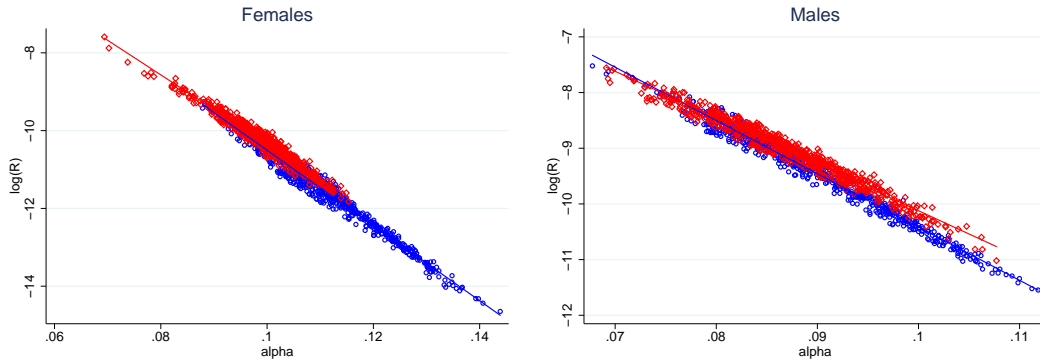
Data points 1975-99 (blue circles) and 1950-74 (red diamonds). The solid lines represent the pooled OLS fit for each period. The countries in the sample are: Australia, Austria, Belgium, Bulgaria, Canada, Czech Republic, Denmark, England & Wales, Finland, France, Hungary, Iceland, Ireland, Italy, Japan, Netherlands, New Zealand, Northern Ireland, Norway, Portugal, Scotland, Slovakia, Spain, Sweden, Switzerland and United States.

to almost 96 years. These absolute number should be taken with some caution. Pooling the data over (arbitrarily chosen) 25 year periods clearly has some unwanted side effects if the slope coefficient changes over time. The main point that we want to make here is that the slope coefficient, that is human life span, was not time-invariant.[4]

TABLE 3. Human Life Span 1950-1999

| | 1950-74 | | 1975-1999 | |
	male	female	male	female
T	83.5	88.9	95.7	96.7
	(0.62)	(0.77)	(0.54)	(0.64)
log(M)	-1.78	-1.45	-0.85	-0.83
	(0.05)	(0.08)	(0.05)	(0.07)
R^2	0.97	0.95	0.98	0.97

Standard errors in parentheses. The countries in the sample are: Australia, Austria, Belgium, Bulgaria, Canada, Czech Republic, Denmark, England & Wales, Finland, France, Germany, Hungary, Ireland, Italy, Japan, Netherlands, New Zealand, Northern Ireland, Norway, Portugal, Scotland, Slovakia, Spain, Sweden, Switzerland and United States.

Expanding life-span, however is a fairly recent phenomenon. Extending the sample period over the the whole last century provides supporting evidence for the notion of invariant life-span

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[4]Recently, Zheng et al. (2011) arrived at a similar result. Their estimates, however were unfortunately based on the Gompertz equation, without the Makeham amendment. Since their sample contained many less developed countries, the bias incurred from ignoring background mortality was potentially large (see Gavrilov and Gavrilova, 1991). This potential bias may explain why they arrive at a much higher estimate of life-span (referred to as the age of zero vitality according to the Strehler-Mildvan methodology) for a sample containing many developing countries than we do for a sample of fully developed countries.

during the first half of the century. As shown in Table 4, we estimate with great precision a life-span of about 88 years for both man and women in the 1900-1924 period as well as in the 1925-49 period. Then, in the 1950-74 period, female life-span took off and male life span followed in the 1975-99 period. Remarkably, male and female life-span are estimated to be about the same in the earlier periods, as theory predicts. We interpret these results as follows. During the first half of the 20th century (and presumably also earlier in human history) the data supports the notion of an invariant human life-span of about 88 years. Observable improvements of life-expectancy during that period originated from declining background mortality (sanitation, vaccination) and from a reduction of R in association of movement along the mortality compensation line, indicating better (initial) physiological conditions (better nutrition, e.g. Fogel and Costa, 1997). Then, in the later 20th century, "something" happened. According to reliability theory these events effectively manipulated the failure rate, that is the intrinsic rate of bodily decay.

TABLE 4. Human Life Span 1900-1999

| | 1900-24 | | 1925-49 | | 1950-74 | | 1975-99 | |
	male	female	male	female	male	female	male	female
T	87.1	89.5	87.4	86.8	89.3	95.6	94.0	93.6
	(0.75)	(0.88)	(0.87)	(1.18)	(0.83)	(1.08)	(0.48)	(0.57)
log(M)	-1.31	-1.22	-1.33	-1.50	-1.28	-0.77	-1.02	-1.22
	(0.07)	(0.08)	(0.08)	(0.11)	(0.07)	(0.11)	(0.04)	(0.07)
R^2	0.98	0.97	0.97	0.95	0.97	0.96	0.99	0.99

Standard errors in parentheses. The countries in the sample are: Belgium, Denmark, England & Wales, Finland, France, Italy, Netherlands, New Zealand, Norway, Scotland, Sweden and Switzerland.

4.4. **Life Span and Life Expectancy.** Finally we would like to explore the link between life-span and life-expectancy empirically. For that purpose we estimate (11) annually from 1950 to 1999 for the sample of 26 countries for which we have consistent data and compare it with the "frontier life expectancy" calculated by Oeppen and Vaupel (2002). We visualize the combined data in Figure 8 in a simple scatter plot and add a spline fit with a 95 percent confidence interval to it.

Interestingly, life span and frontier life expectancy are virtually uncorrelated during about the first 20 years of the observation period, which further corroborates the idea of improving life-expectancy through lower background mortality or through better health and nutrition in line with an invariant compensation effect. Then, from about the 1970's onwards we observe

a strong positive correlation between the two variables, indicating that recent improvements in life-expectancy were indeed driven at least partly by expanding human life-span.

FIGURE 8. Life Span and "Frontier" Life Expectancy
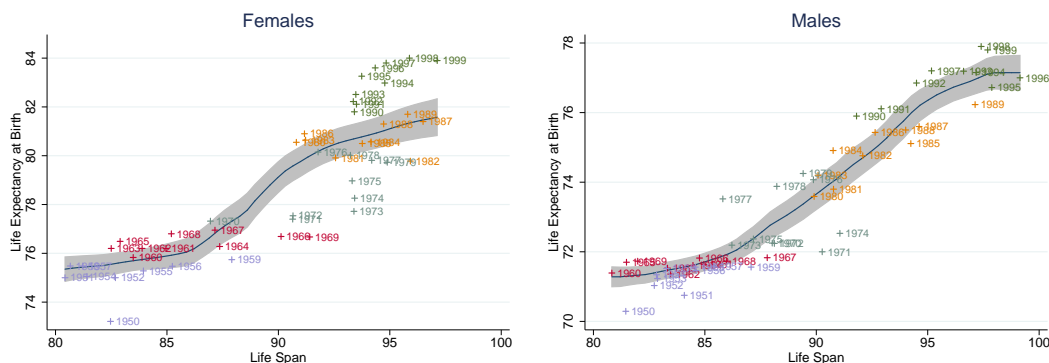
Life span for a given year is obtained by a simple linear cross-country regression of $\log(R)$ on α. The frontier life expectancy data is taken from Oeppen and Vaupel (2002). The solid line is a fitted spline with a 95 percent confidence interval around it (shaded in gray). Countries as for Table 3.

The notion of an expanding human life-span is helpful to rationalize why other researchers have not (yet) observed any convergence of increasing life-expectancy (Wilmoth, 1997, Vaupel, 2010). Predictions of a certain limit to life expectancy – for example, that it should not exceed 35 years at age of 50 (Olshansky et al, 1990) – are presumably made under the wrong perception of an invariant human life-span, in which case they would have been indeed fully justified and understandable as the analysis in Section 3.2 has shown. Unlike other species, however, humans seem to be able to modify life-span such that there is no limit of life-expectancy visible in the data.

5. CONCLUSIONS

In this paper we have introduced to the economics science a theoretical foundation of human life-span based on two strong empirical regularities, the Gompertz-Makeham law and the compensation effect of mortality. We have estimated the parameters of the Gompertz-Makeham model and found a remarkable long-run trend. Contemporaneous humans start out as young adults much more healthy than their forefathers a century ago but they are also aging faster. For a long time of our history this trend was consistent with the observation of rising life-expectancy under an invariant life-span.

If life-span were indeed immutable, as it is presumably for other animals, future improvements in life-expectancy would soon end. Life expectancy, as we have shown in this paper, is bounded from above by life-span. For the second half of the 20th century, however, we were able to present evidence for expanding life-span. It increased quite significantly by about 8 years. For the last quarter of the 20th century we find furthermore that life expectancy increased in sync with life expectancy, a phenomenon that explains why other researchers have observed "broken limits to life-expectancy" (Oeppen and Vaupel, 2002).

It is beyond the scope of the present paper to identify the cause of expanding life-span. Yet theory suggests that the potential candidates have to be fairly recent events which manipulated the failure rate of human body parts. The most natural candidates are probably regenerative medicine and replacement surgery. To be more concrete, heart bypass operation and dialysis machines are two prominent examples of medical innovations of the second half of the 20th century which prolonged human lifes by replacing the functional performance of failed organs.

The notion of modifiable life-span is important for economists, policymakers as well as theorists. Taking the "capital T" as constant may lead to severe misjudgements about the future of population aging and the financial stability, meaningfulness, and efficiency of social security and public health systems. Also, on the individual level, the awareness of modifiable and potentially further rising life-span beyond the "natural" improvements of life-expectancy, may have quite dramatic effects on life-cycle decisions like schooling, savings, and retirement. Re-considering our conventional life-cycle theory and integrating the notion of expanding life-span is a challenging task for future research.

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Figure A.1: Distribution of Estimated Gompertz Parameters / Standard Errors
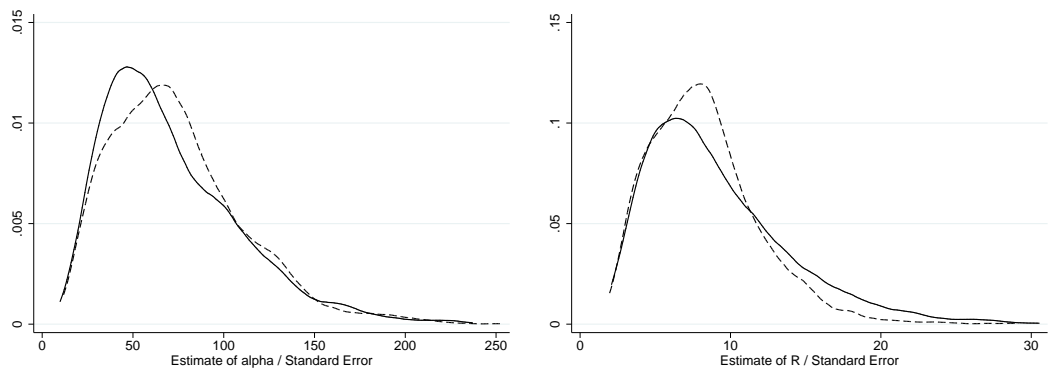
Figure A.2: Survival Probability in Australia, Austria, Belgium and Bulgaria
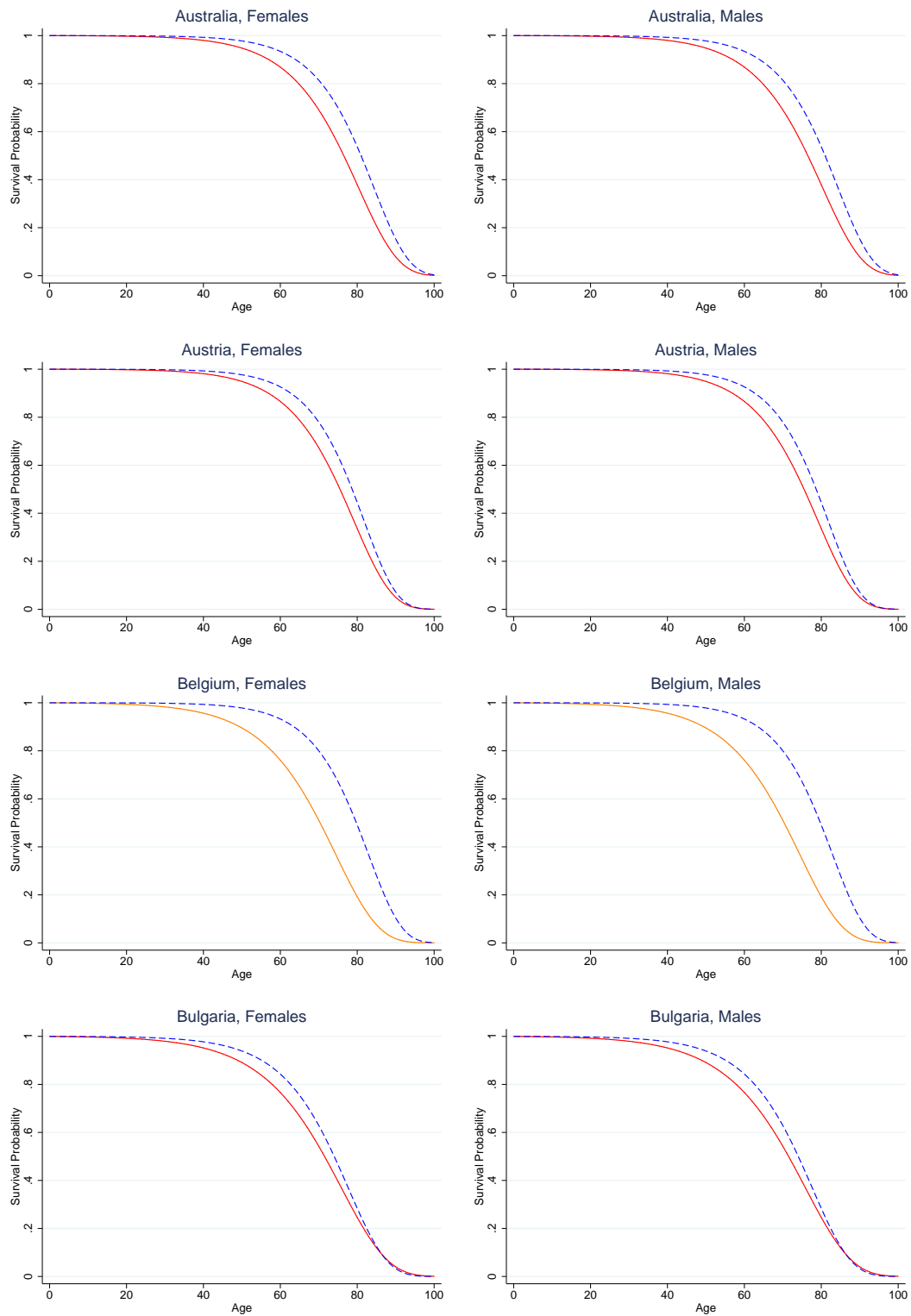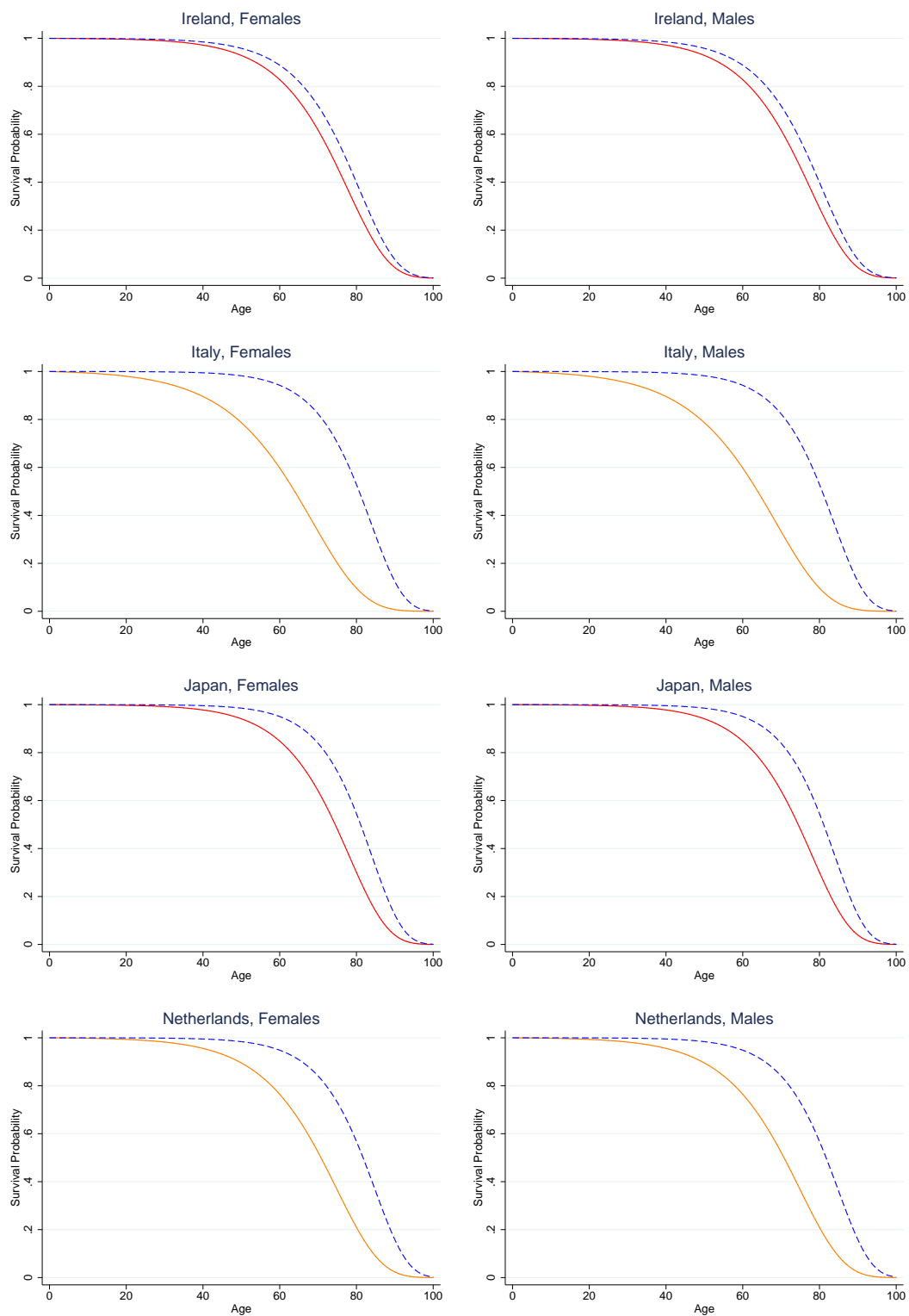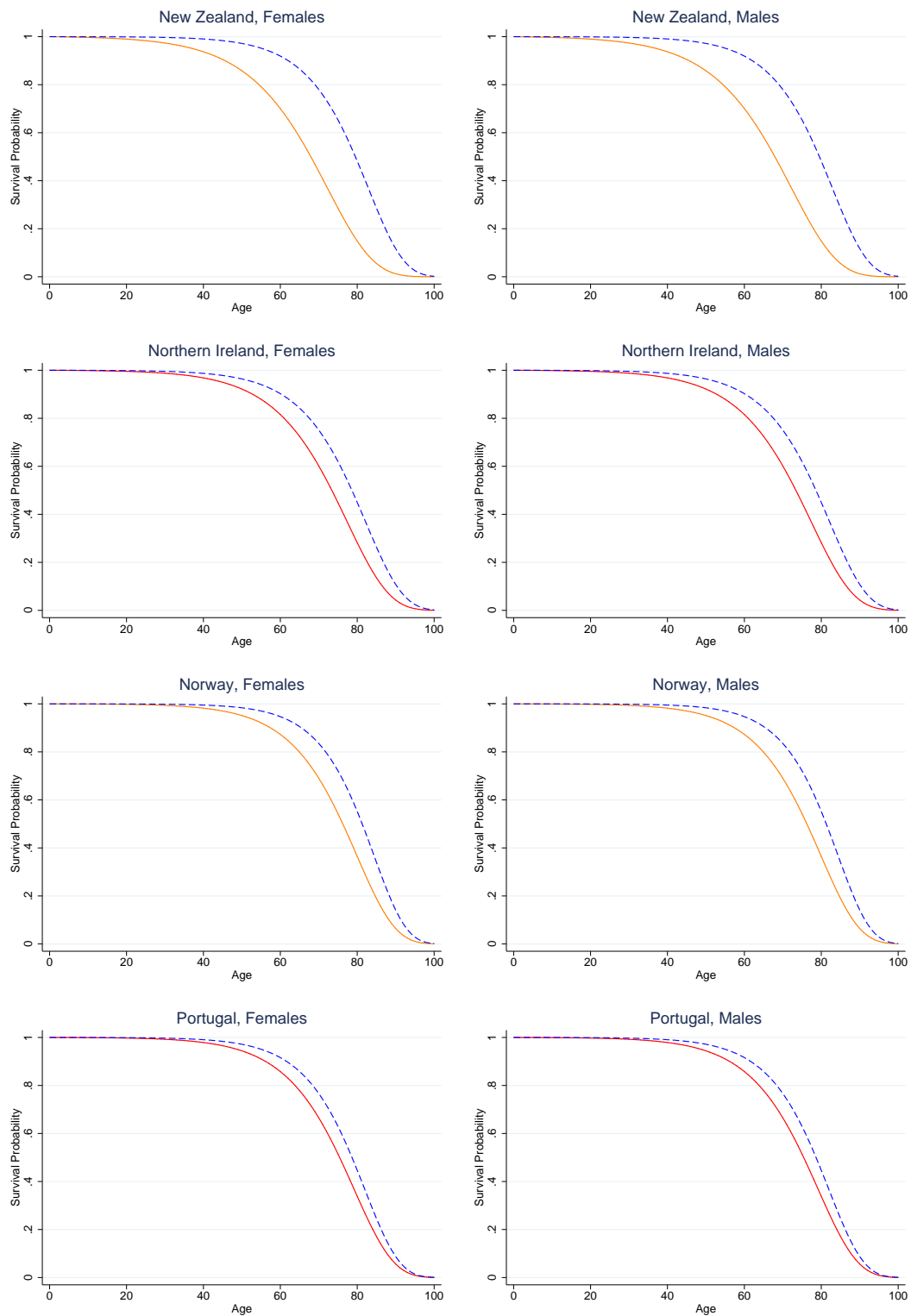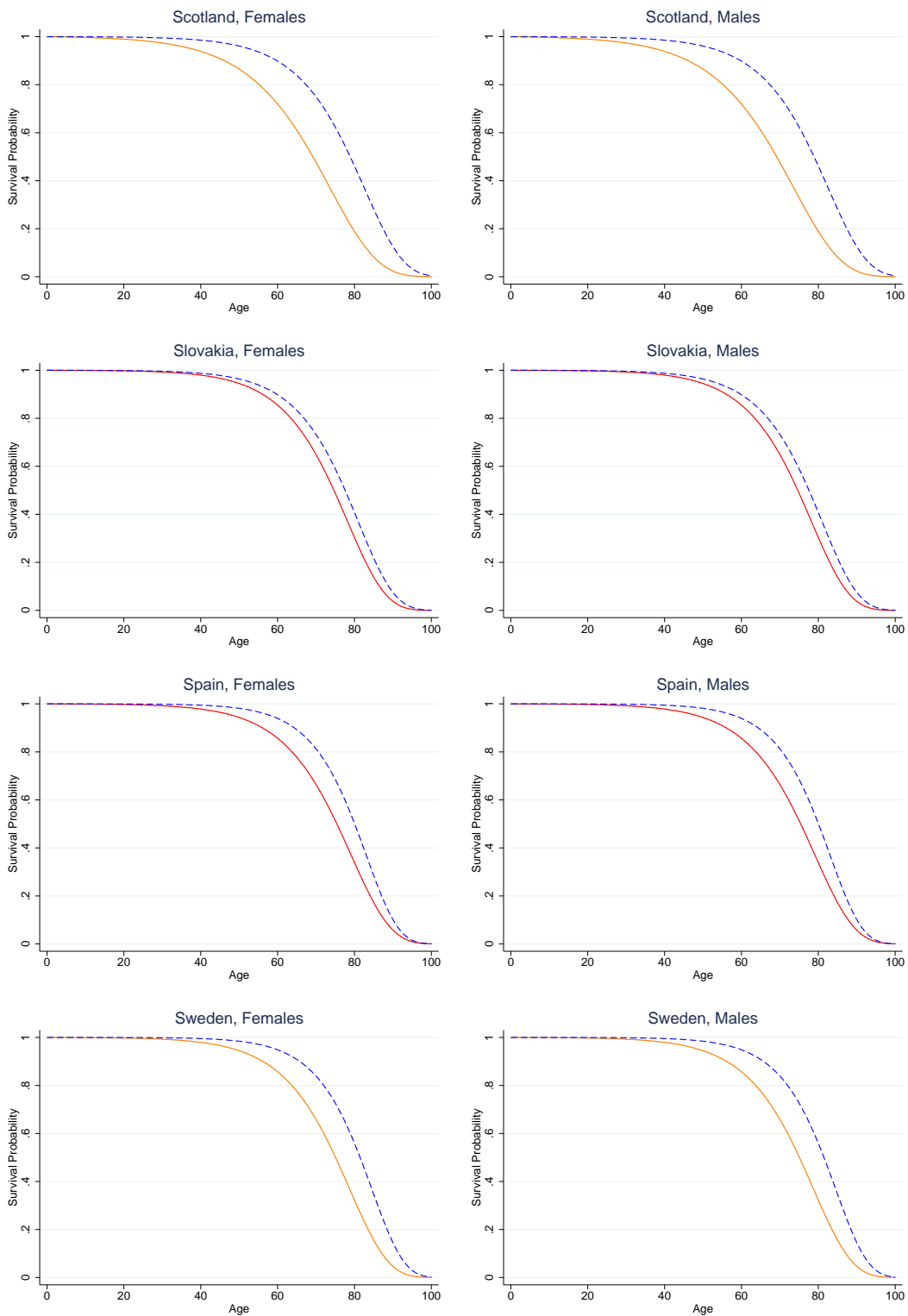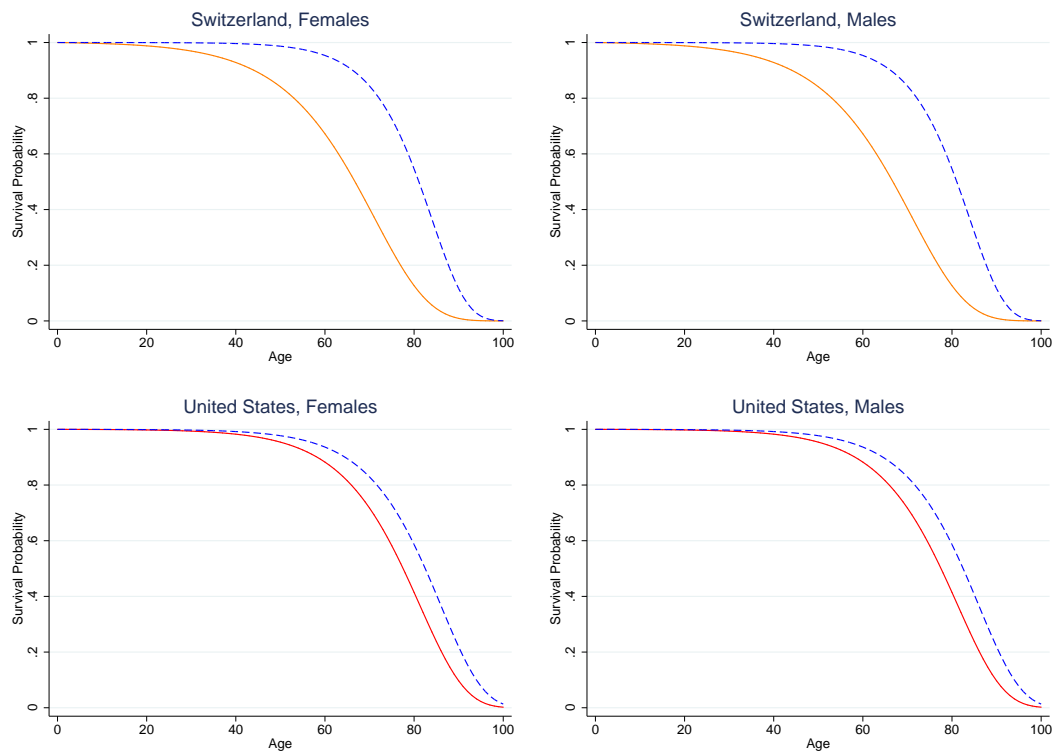

Dashed blue line: Survival function 1975-1999. Solid red line: Survival function 1950-74. Solid orange line: Survival function 1900-24.

Figure A.2 (cont.): Survival Probability in Canada, the Czech Republic, Denmark and England
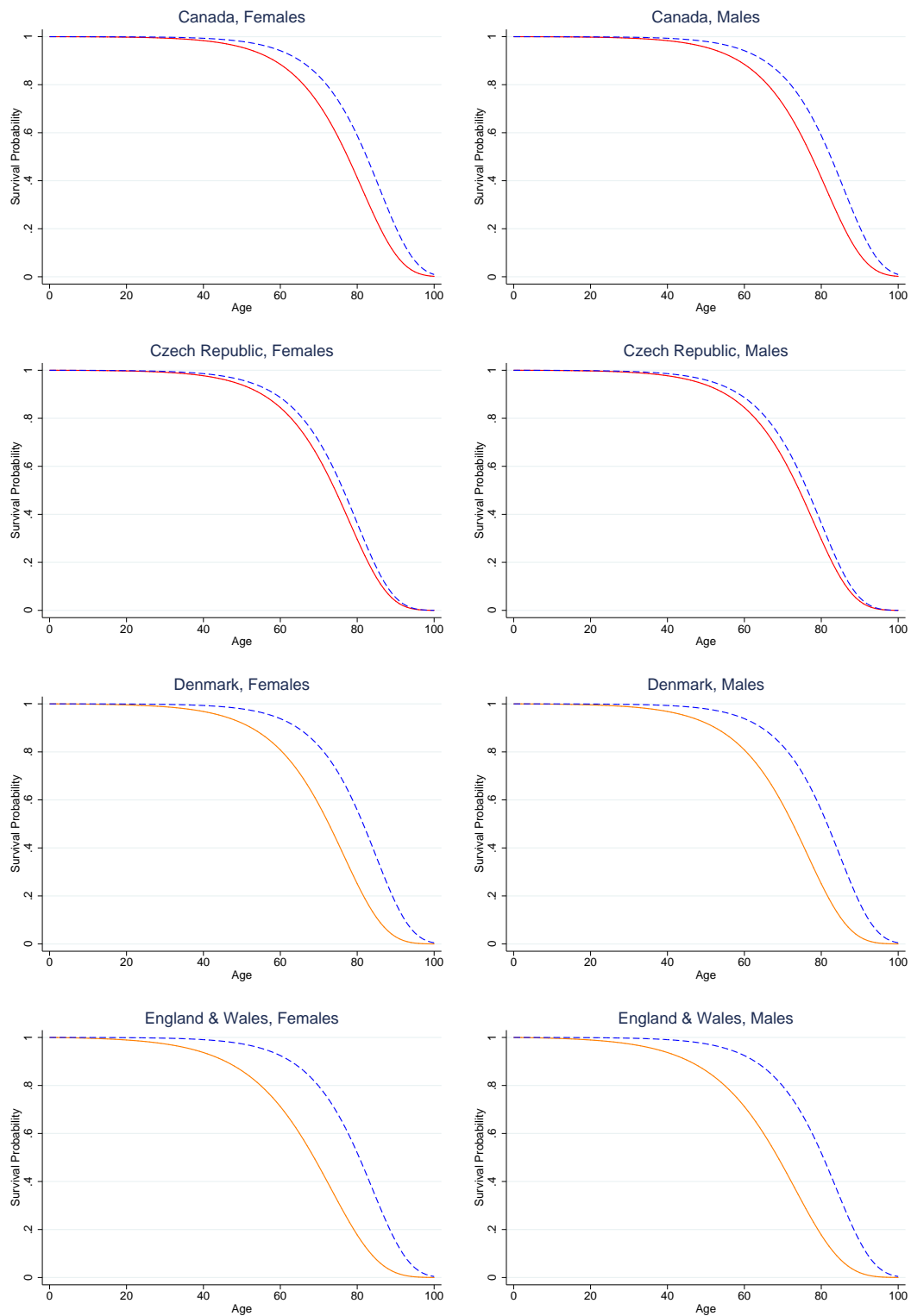
Dashed blue line: Survival function 1975-1999. Solid red line: Survival function 1950-74. Solid orange line: Survival function 1900-24.

Figure A.2 (cont.): Survival Probability in Finland, France, Germany and Hungary
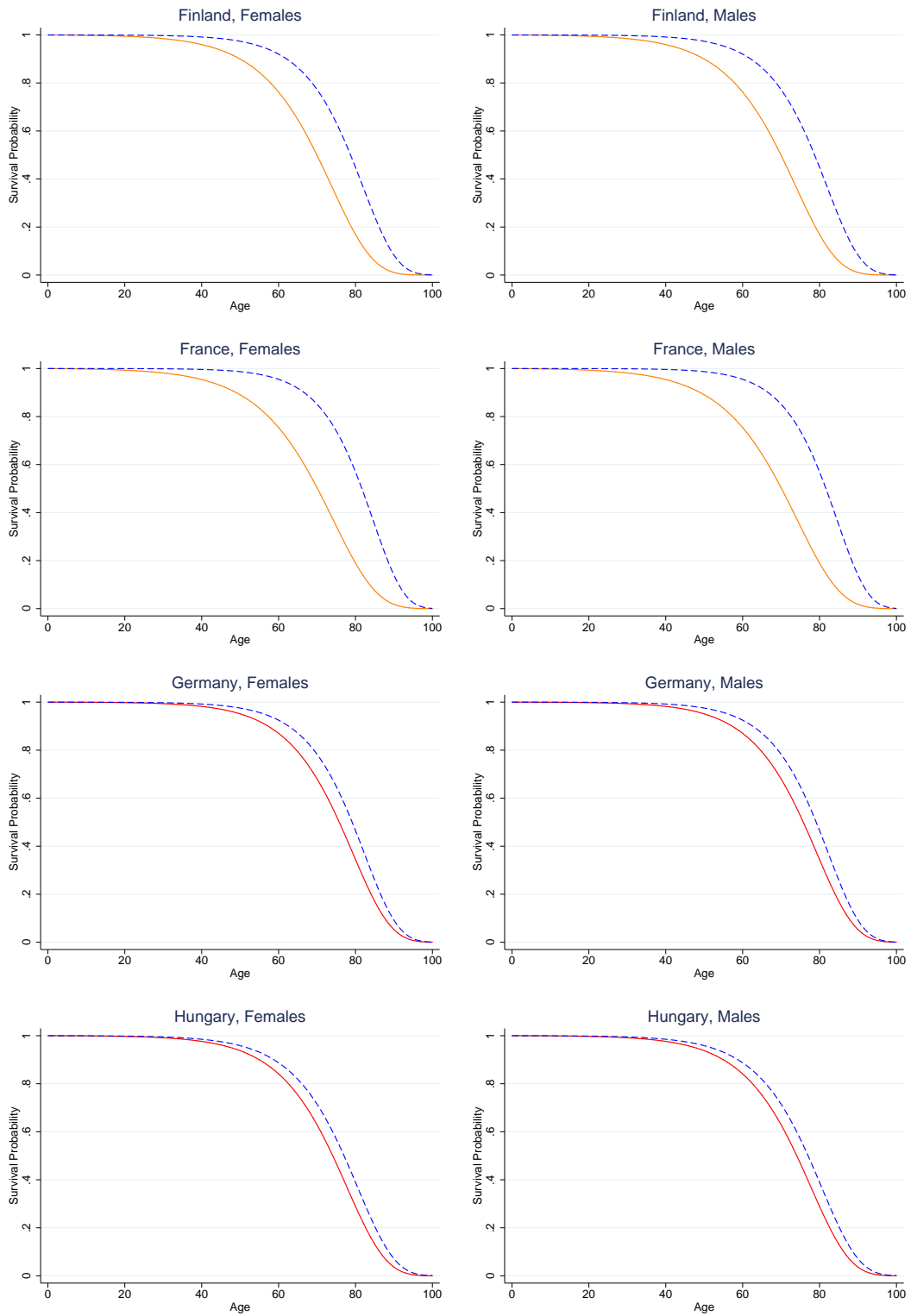
Dashed blue line: Survival function 1975-1999. Solid red line: Survival function 1950-74. Solid orange line: Survival function 1900-24.

Figure A.2 (cont.): Survival Probability in Ireland, Italy, Japan and the Netherlands

Dashed blue line: Survival function 1975-1999. Solid red line: Survival function 1950-74. Solid orange line: Survival function 1900-24.

Figure A.2 (cont.): Survival Probability in New Zealand, N. Ireland, Norway and Portugal

Dashed blue line: Survival function 1975-1999. Solid red line: Survival function 1950-74. Solid orange line: Survival function 1900-24.

Figure A.2 (cont.): Survival Probability in Scotland, Slovakia, Spain and Sweden

Dashed blue line: Survival function 1975-1999. Solid red line: Survival function 1950-74. Solid orange line: Survival function 1900-24.

Figure A.2 (cont.): Survival Probability in Switzerland and the United States

Dashed blue line: Survival function 1975-1999. Solid red line: Survival function 1950-74. Solid orange line: Survival function 1900-24.

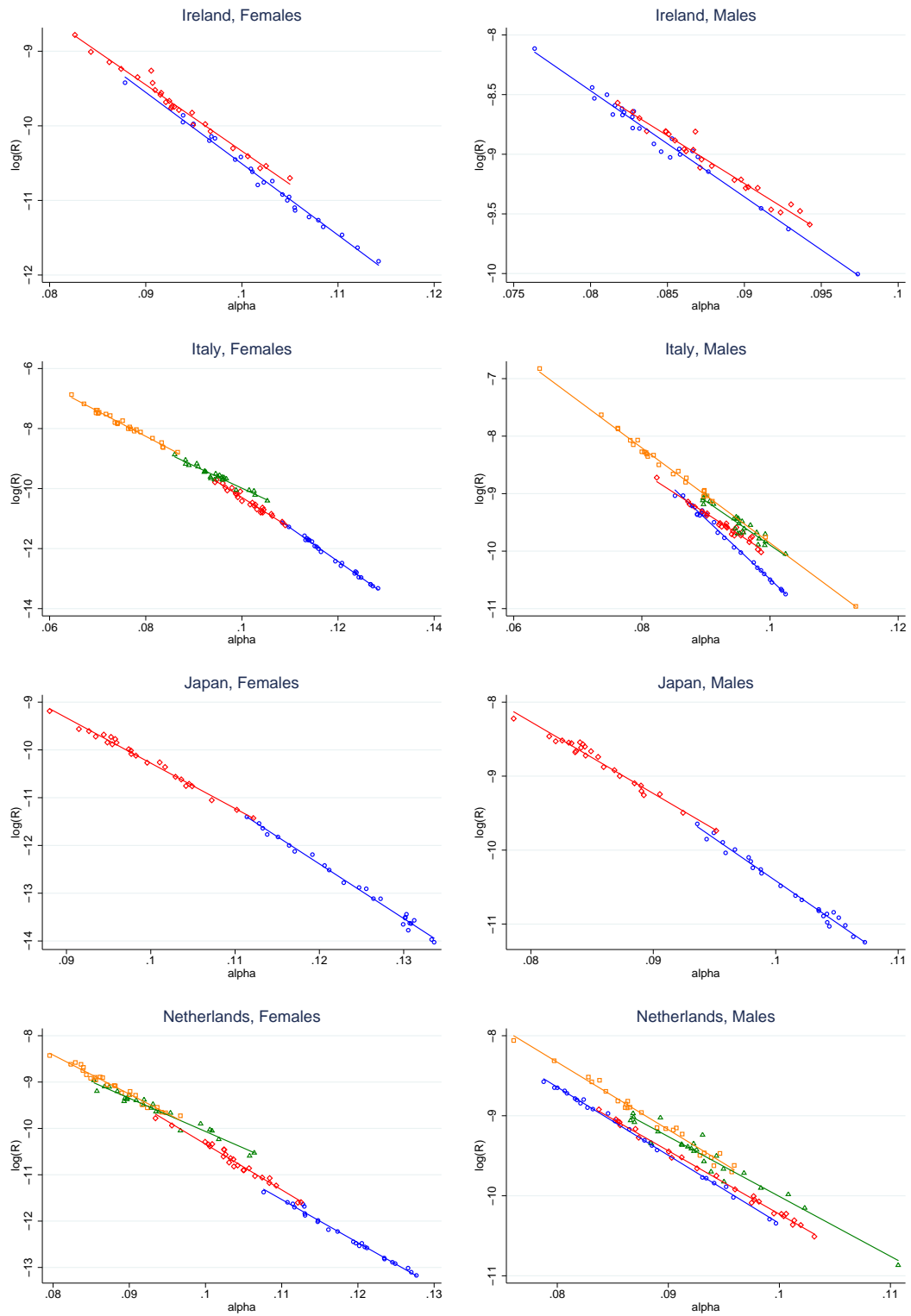Figure A.3: Strehler-Mildvan Correlation in Australia, Austria, Belgium and Bulgaria
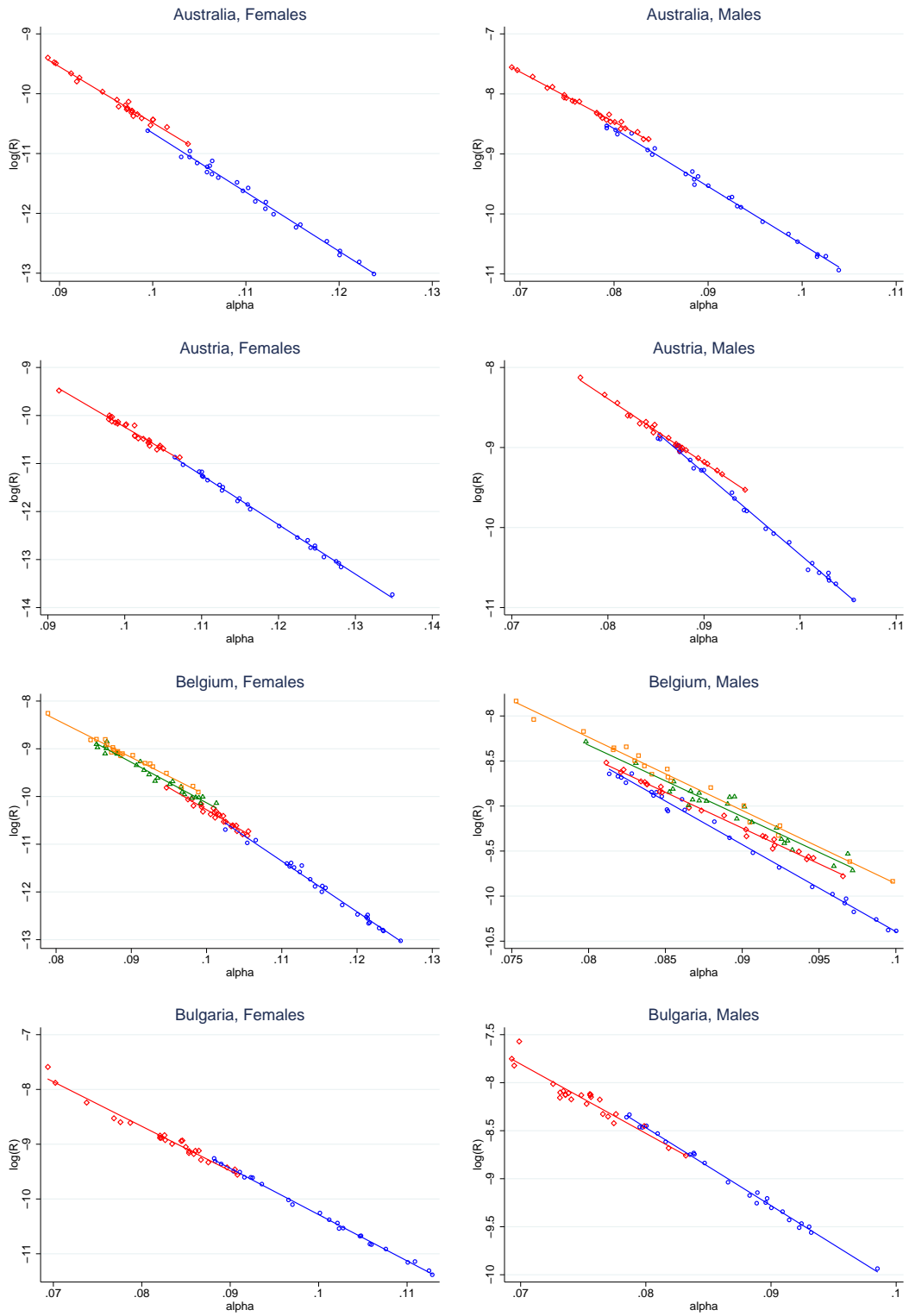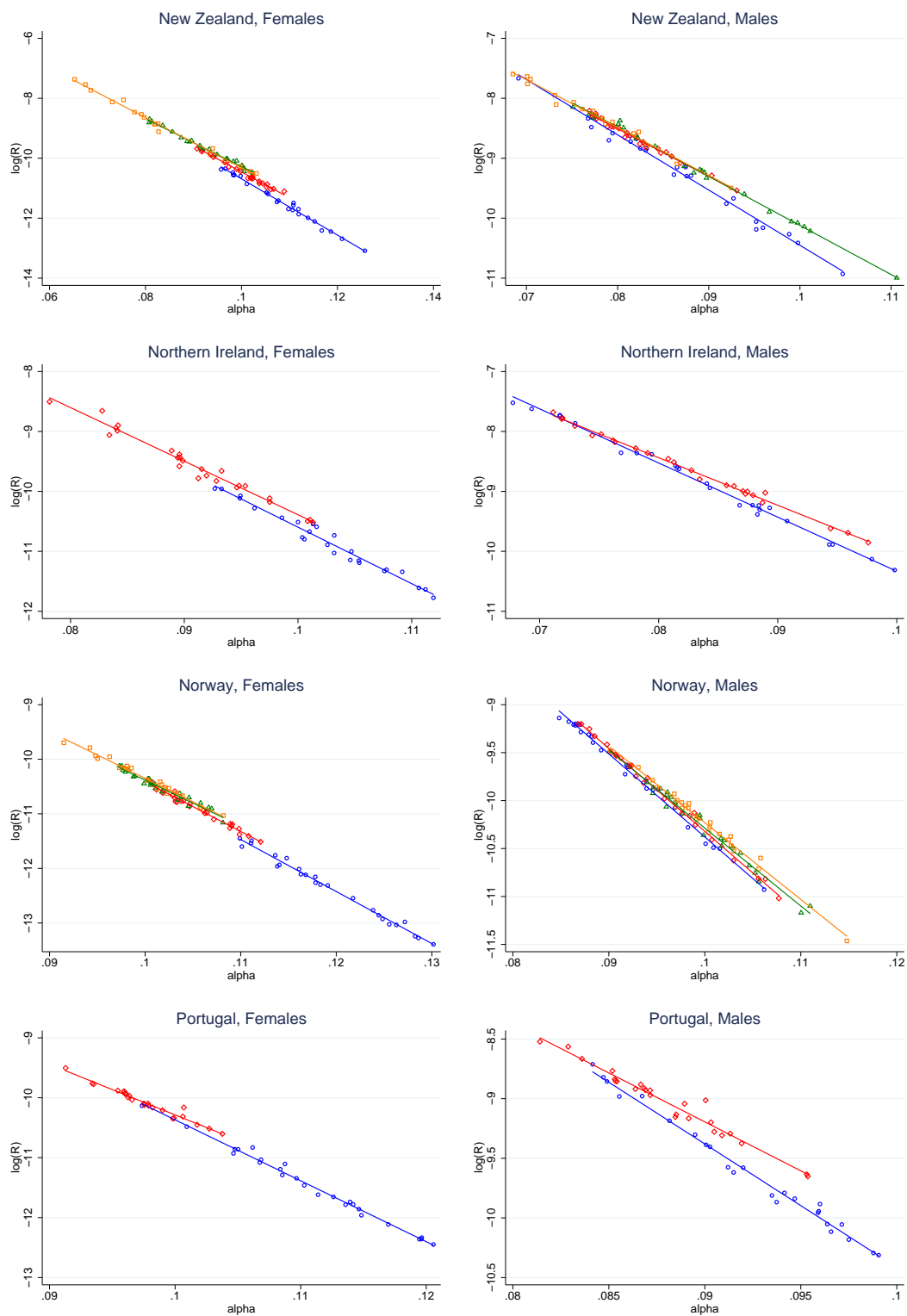
Data points 1975-99 (blue circles), 1950-74 (red diamonds), 1925-49 (green triangles) and 1900-24 (orange rectangles). The solid lines represent the OLS fit for each period.

Figure A.3 (cont.): Strehler-Mildvan Correlation in Canada, the Czech Republic, Denmark and England
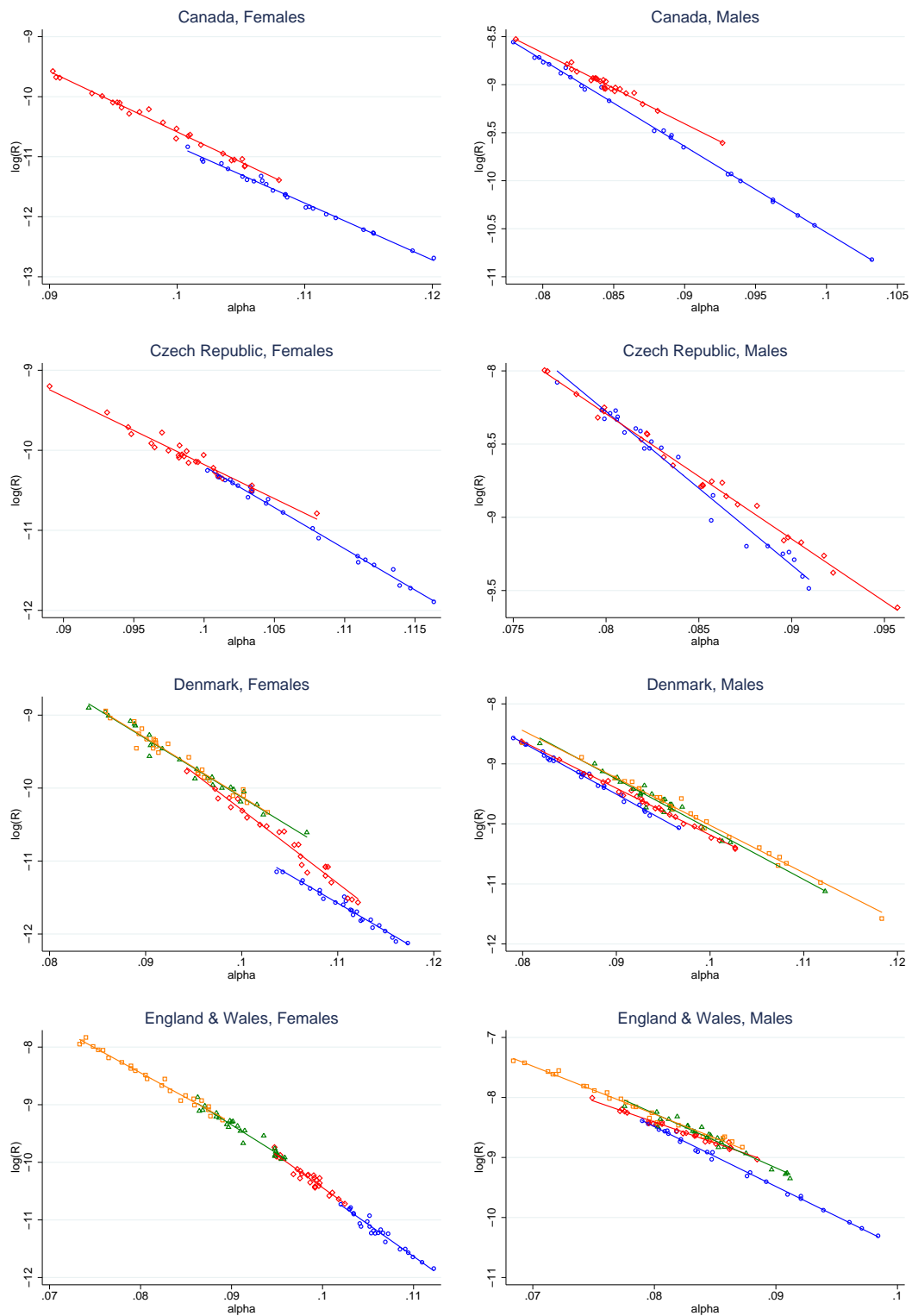
Data points 1975-99 (blue circles), 1950-74 (red diamonds), 1925-49 (green triangles) and 1900-24 (orange rectangles). The solid lines represent the OLS fit for each period.

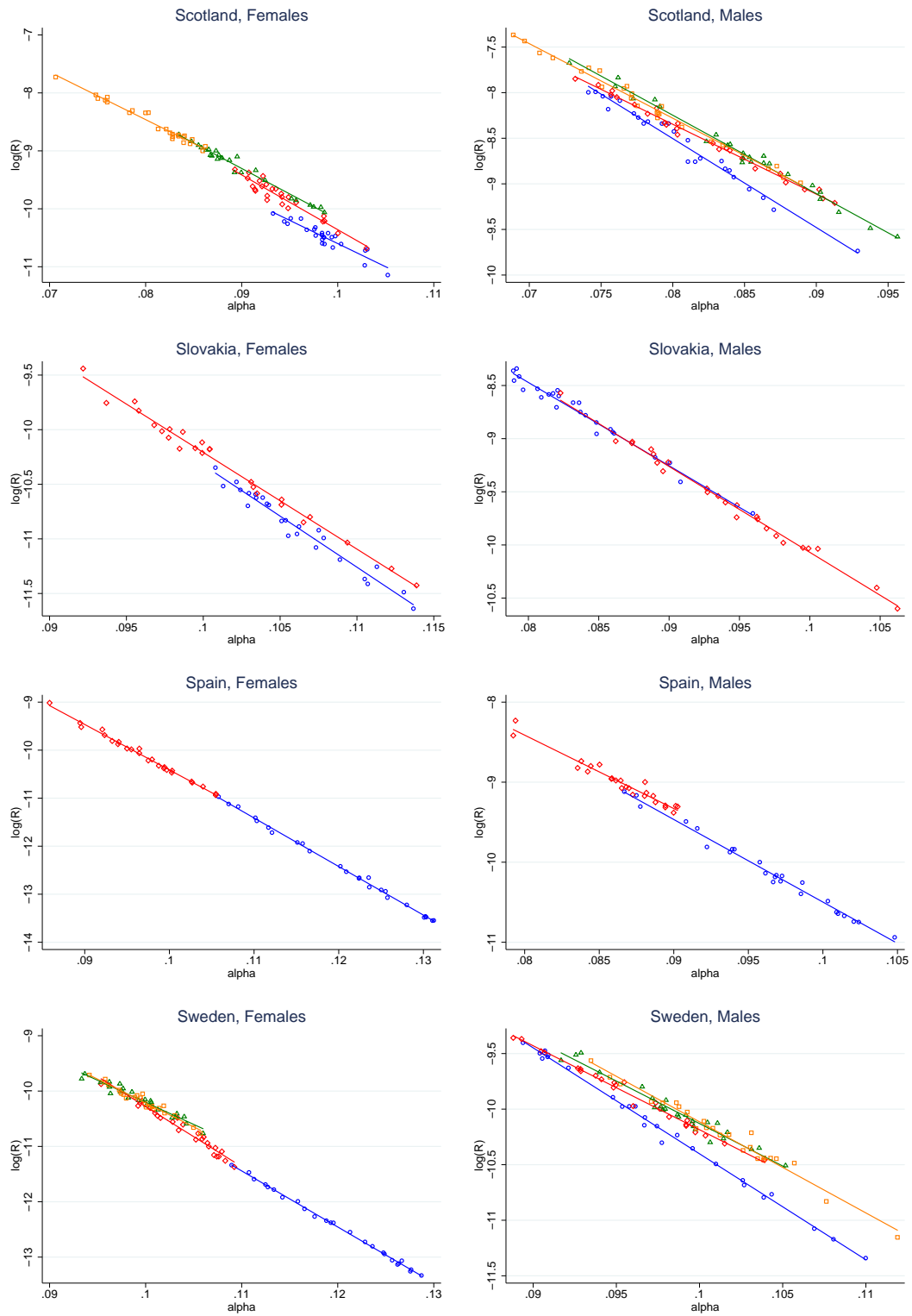Figure A.3 (cont.): Strehler-Mildvan Correlation in Finland, France, Germany and Hungary
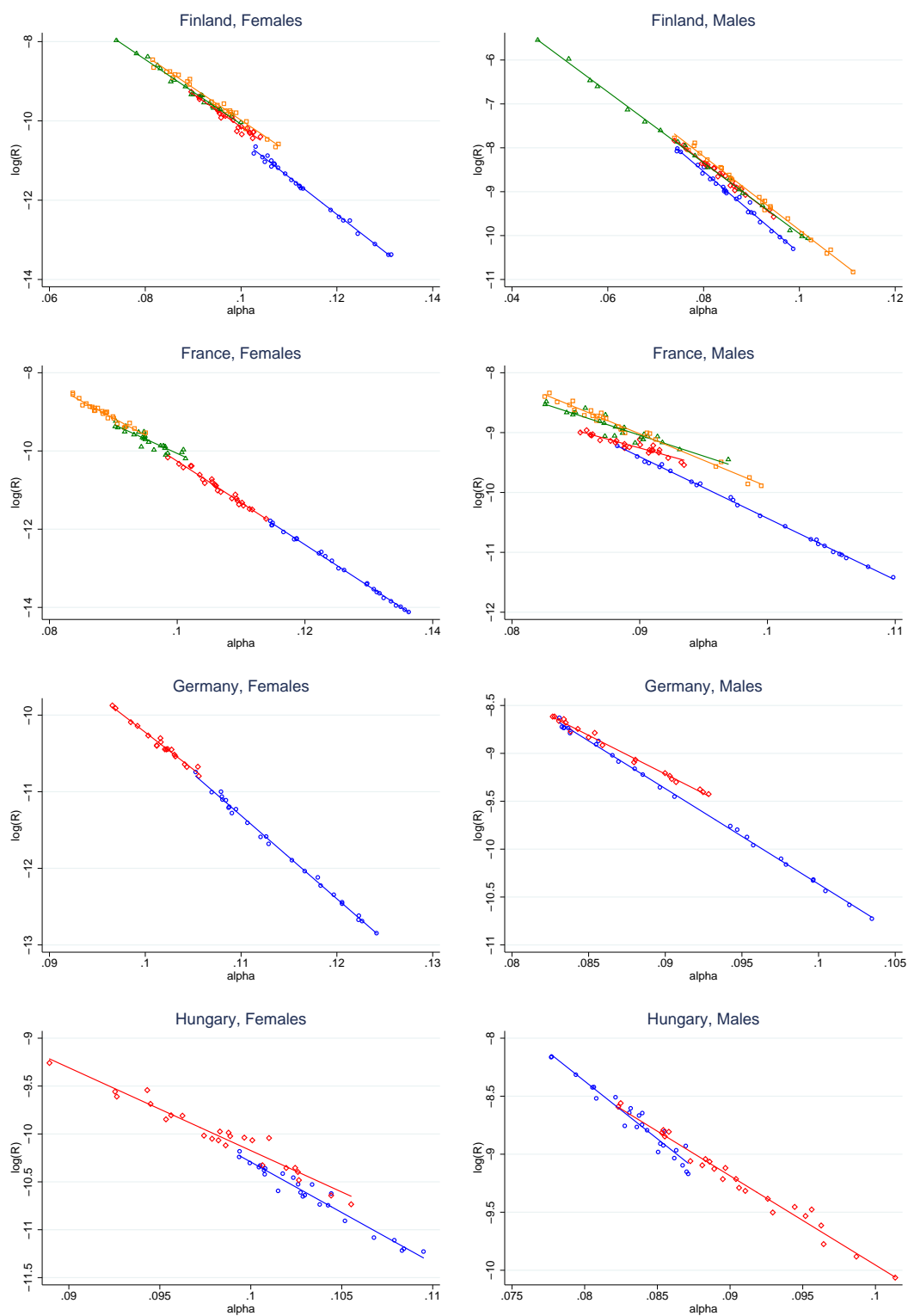
Data points 1975-99 (blue circles), 1950-74 (red diamonds), 1925-49 (green triangles) and 1900-24 (orange rectangles). The solid lines represent the OLS fit for each period.

Figure A.3 (cont.): Strehler-Mildvan Correlation in Ireland, Italy, Japan and the Netherlands
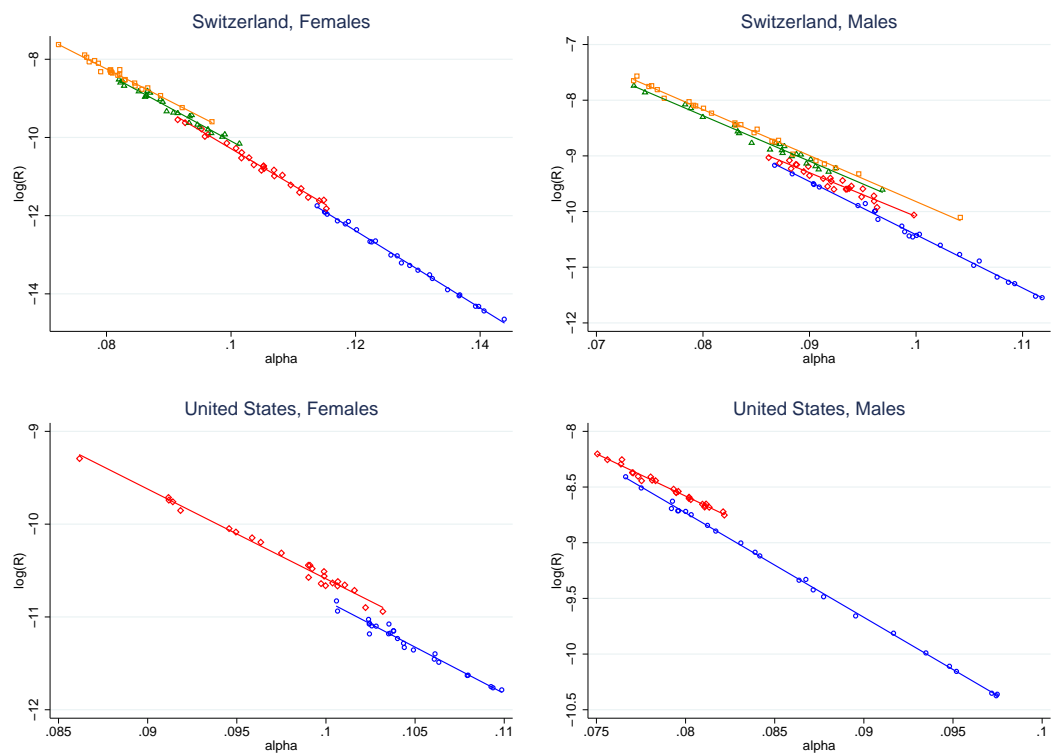

Data points 1975-99 (blue circles), 1950-74 (red diamonds), 1925-49 (green triangles) and 1900-24 (orange rectangles). The solid lines represent the OLS fit for each period.

Figure A.3 (cont.): Strehler-Mildvan Correlation in New Zealand, N. Ireland, Norway and Portugal

Data points 1975-99 (blue circles), 1950-74 (red diamonds), 1925-49 (green triangles) and 1900-24 (orange rectangles). The solid lines represent the OLS fit for each period.

Figure A.3 (cont.): Strehler-Mildvan Correlation in Scotland, Slovakia, Spain and Sweden
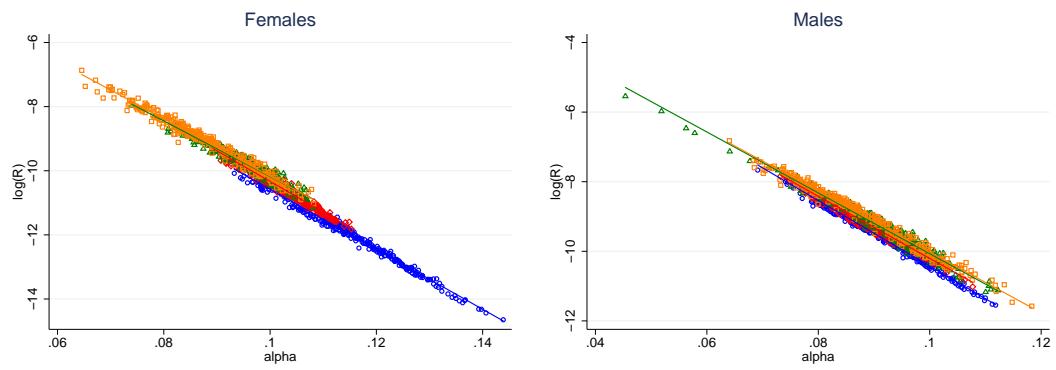
Data points 1975-99 (blue circles), 1950-74 (red diamonds), 1925-49 (green triangles) and 1900-24 (orange rectangles). The solid lines represent the OLS fit for each period.

Figure A.3 (cont.): Strehler-Mildvan Correlation in Switzerland and the United States

Data points 1975-99 (blue circles), 1950-74 (red diamonds), 1925-49 (green triangles) and 1900-24 (orange rectangles). The solid lines represent the OLS fit for each period.

Figure A.4: Strehler-Mildvan Correlation 1900-1999

Data points 1975-99 (blue circles), 1950-74 (red diamonds), 1925-49 (green triangles) and 1900-24 (orange rectangles). The solid lines represent the pooled OLS fit for each period. The countries in the sample are: Belgium, Denmark, England & Wales, Finland, France, Iceland, Italy, Netherlands, New Zealand, Norway, Scotland, Sweden and Switzerland.