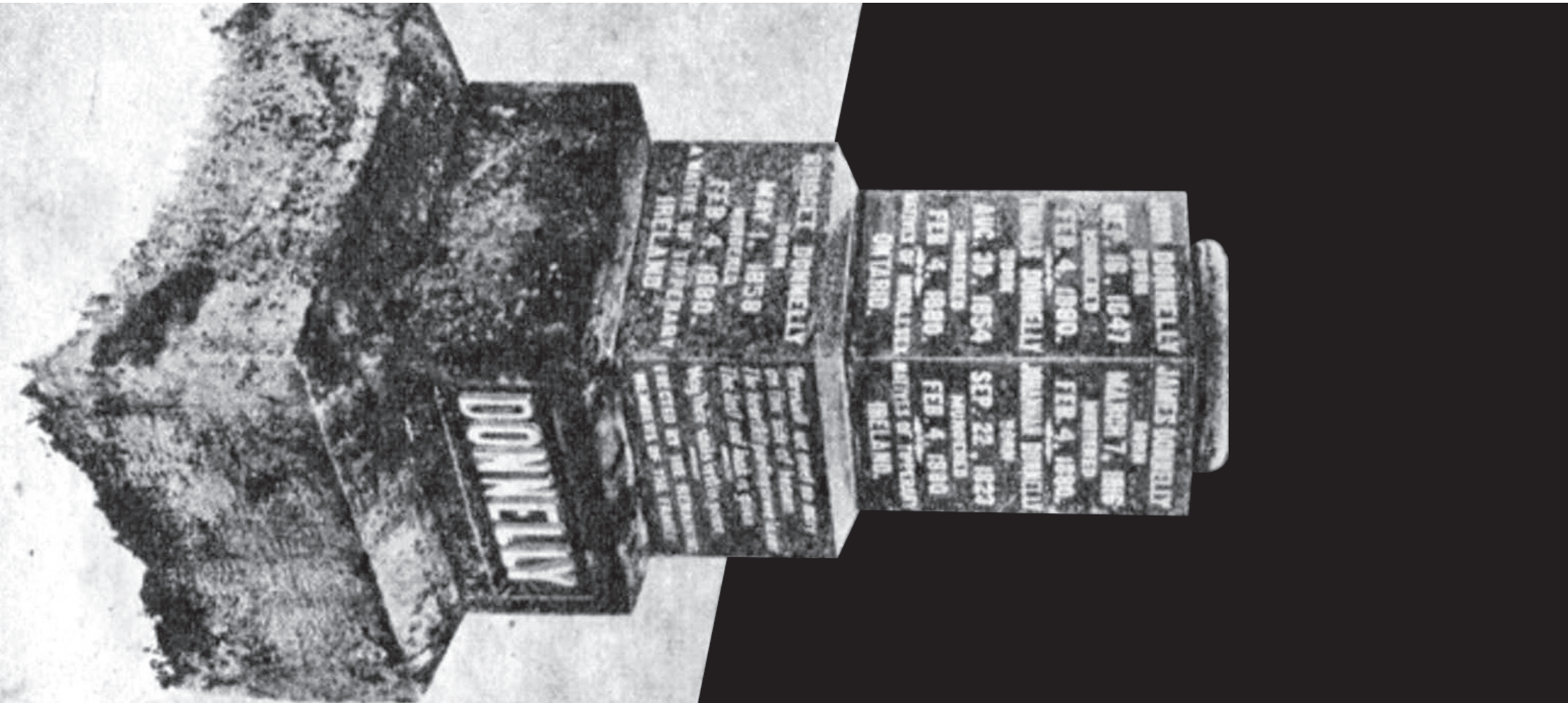
LIFESPAN VARIATION
METHODS, TRENDS AND THE ROLE OF SOCIOECONOMIC INEQUALITY

ALYSON VAN RAALTE



Lifespan Variation

Methods, trends and the role of socioeconomic inequality

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Introduction

CHAPTER 1

The life expectancy revolution

Pre-industrial Europe was characterized by centuries of high mortality, with life expectancy vacillating between 25 and 40 years.[1 2] Malthus[3] famously described the population as being kept down by the mortality checks of his time, namely pandemics and war, but adding (p. 61) "should success be still incomplete, gigantic inevitable famine stalks in the rear, and with one mighty blow levels the population with the food of the world."

Unfortunately for Malthus, he failed to observe that major changes to this stable system were already underway in his country, and by around the mid 19th Century had taken root in many parts of Western Europe. Mortality was declining, ushering in a rapid and unprecedented ascent in life expectancy.[2 4 5]

The reasons behind the onset of mortality decline have been intensely debated. For years most research assumed that the expansion of medical services, public health practices, and improved sanitation were responsible for the bulk of the decline. In 1976, McKeown[6] published an iconoclastic book that instead attributed a greater role to overall economic expansion, in combination with lower grain prices, which led to better nutrition. McKeown's thesis was later refined by Fogel[7], who argued that it was the increased 'nutritional status' (the balance of the intake of nutrients with the claims against it), particularly in infancy and childhood, which led to reductions in mortality. Criticism to McKeown's hypothesis was levelled on many fronts, most fiercely for his lack of compelling positive evidence and for his overreliance on English data.[8] Clean water and sewage improvements, for instance, were found to have been responsible for much of the mortality decline in French urban centres[9] and American cities.[10]

Although some dispute remains over the relative importance of public intervention versus economic factors, both factions are in agreement that it was ultimately the controlling of infectious diseases that initially brought down mortality, particularly over infancy and childhood. Cardiovascular disease and cancer were attributed to less than 6 percent of all deaths in pre-industrial London, according to Graunt's Bills of Mortality of 1662.[11] By comparison, they together accounted for almost two-thirds of all deaths in much of Western Europe and North America in the year 2000.[12]

In his highly influential epidemiologic transition theory, Omran[13] described these transformations in the age and disease profile of mortality. He divided society up into three distinct periods: (1) the age of pestilence and famine, (2) the age of receding pandemics, and (3) the age of degenerative and man-made diseases. This

transition can best be understood by examining the changes to the age-at-death profile, as pictured for Swedish males in Figure 1. The initial period up to the mid 19th Century showed no sustained shifts in the age pattern of mortality. Pandemic, war and famine years are also clearly demarked by the vertical lines. After about 1850 mortality began to decline. The reduction in infectious disease led to a gradual reduction of deaths through infancy and childhood, while the receding pandemics led to less yearly fluctuation in mortality. Shortly after the clearly visible 1918 flu pandemic and war year, a much greater concentration of deaths around the adult modal age becomes visible, consistent with a transition from early onset infectious diseases to degenerative diseases manifesting themselves at later ages.

Omran's theory was published in 1971, at a time when progress against cardiovascular disease was unforeseen. Since then some authors, including Omran himself, have called for a fourth age to be added, to account for progress against degenerative diseases.[16-19] Omran's theory has been criticized for its deterministic nature.[20] Nevertheless, the overriding change in the age and cause-of-death patterns he described have certainly revolutionized society.

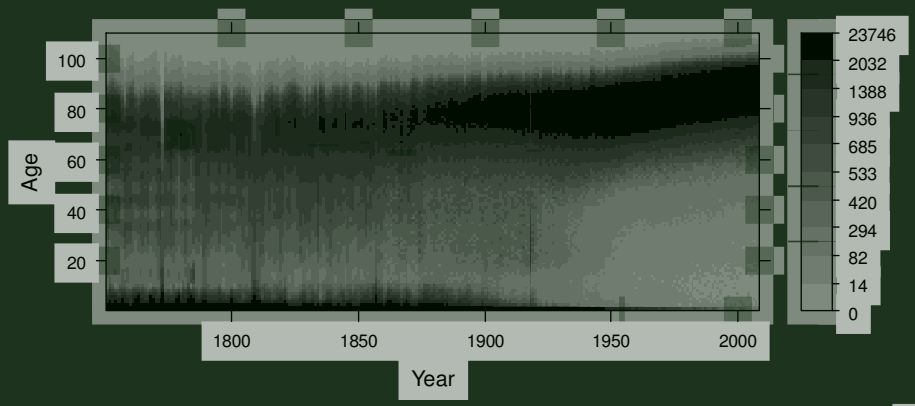

Figure 1: The change in the density of period life table deaths for each age and year, out of a life table radix of 100 000, Swedish males, 1751 to 2005. The cut points refer to death deciles, to more clearly delineate differences. Data is from the HMD.[14] Observations prior to 1802 are less reliable, but following a series of reforms, are be considered to be of a high quality since 1860.[15]

Remarkably, despite these differences in the age pattern of mortality, life expectancy itself has increased in an extraordinarily linear manner. Oeppen and Vaupel found that record female life expectancy had followed a linear trend line of 2.5 years per decade since 1840.[4] After refining the dataset and extending it back in time, Vallin and Meslé showed a segmented trend in record female life expectancy: flat and variable over the 1750-90 period, then increasing by 1.2 years (1790-1885), 3.2 years (1885-1960) and 2.3 years (1960-2005) per decade over the respective time periods.[5] Whereas in early years most of the gains to life expectancy came from mortality declines in infancy and childhood, recent gains have come about from declining mortality over middle and older adult ages.

Although life expectancy has risen in a mostly steady, linear fashion since the mid 19th Century, gains to other summary measures of longevity have been less pronounced.[21] The record median age at death has run almost parallel to the life expectancy, albeit at a level about 10 years higher, with the two lines slowly converging as infant mortality is becoming increasingly rare. The record adult modal age at death (to distinguish it from the sometimes larger infant mode) changed relatively little during the 1840-1940 period, hovering at around 80 years, until the mid-1940s for females and the end of the 1970s for males, when major reductions in mortality over adult ages took root.

These differences in trends speak to one of the problems of relying exclusively on life expectancy as a summary measure of public health. Changes in the underlying age distribution of death, which can be substantial, are hidden. This thesis focuses on one of these other components of longevity that has been less examined: variation in individual lifespans.

Why is lifespan variation an important dimension to quantify?

Among the first persons to seriously contemplate the distribution of lifespans over age was the German statistician Wilhelm Lexis. He hypothesized that once non-senescent death could be removed from the population, lifespans would become normally distributed around the adult modal age at death.[22] This line of reasoning featured prominently in the later work of Fries[23] who argued that society was nearing the point where little more could be done to reduce old aged mortality. Improvements in survival would come from reducing non-senescent death and mortality would eventually become compressed into a shorter age span around a fixed upper limit. Fries made the unfortunate mistake of quantifying this upper limit at 85 years, a level which has since been surpassed by Japanese females. Although

the idea of a fixed upper limit to lifespan has largely been discredited by the accelerating pace of survival improvements among nonagenarians and centenarians,[24-27] the basic notion that we should monitor how lifespans are distributed over age within the population is gaining traction.

At similar levels of life expectancy, two populations can have different underlying lifespan distributions. This is illustrated in Figure 2, comparing males in the United States and Sweden when they had a life expectancy at birth of 75.5 years (this happened earlier for Sweden). At this level of life expectancy, a larger proportions of American males were dying over both younger adult ages (especially ages 20 to 60), and oldest adult ages. In Sweden lifespans were far more compressed around the modal age-at-death.

Life expectancy at birth has become the single most important summary indicator of the population health. It allows for direct comparisons between populations that are not confounded by differences in the age structure of the population. Also it is an objective absorbing state, unlike measures of morbidity such as self-rated health, which can be influenced by personal characteristics such as gender[28 29] and ethnicity[30 31] and can change over the life course. But as Figure 2 aptly demonstrates, only looking at the mean can hide important differences in the distribution, some of which may confound our subjective assessments of population health.

On the basis of longevity alone, the choice between living in the United States in 2006 and Sweden in 1993 is indeed a normative decision. Would individuals prefer the chance at a longer life, but with an elevated risk of premature mortality? Risk averseness to the timing of death is an underexplored research area, and one that will not be pursued in this thesis.

However if we can imagine that society and individuals would have an interest in knowing the timing in death with greater certainty, tools to quantify the variation around an average lifespan would be needed. Beyond the subjective reasons, a number of objective reasons come to mind. Quantifying lifespan variation is important for accurate forecasts in insurance and annuity markets, for public provisioning of medical care and pensions, and would factor into individual life course decisions, particularly as regards savings and investments.

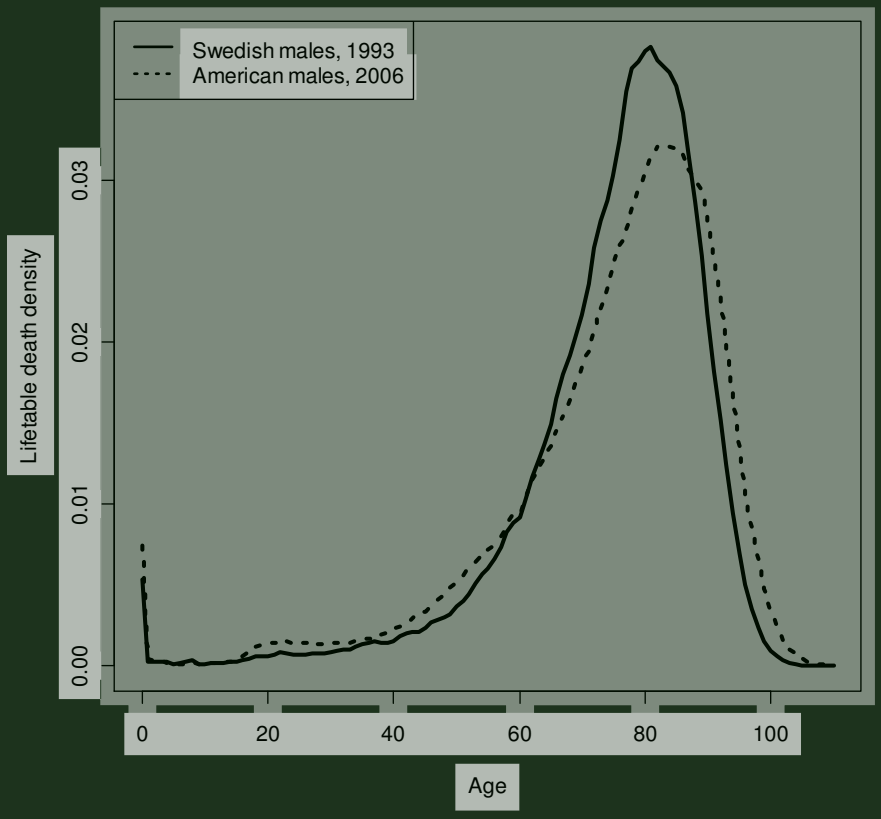

Figure 2: The period lifetable death density for Swedish and American males (smoothed), corresponding to a life expectancy at birth of 75.5 years. Data is from the Human Mortality Database.[27]

Distributive justice and mortality

While the last section offered some compelling reasons to quantify the distribution in lifespans, in this section some of the normative concepts in comparing distributions are explored. The primacy of life itself dictates that inequality in lifespans is perhaps the most fundamental distributional issue we face. Yet far less theorizing over what entails an equitable distribution of lifespans has taken place as compared to the store of distributive justice theories designed around the distribution of goods such as income. There are two interrelated reasons for this. The first is that life years cannot be 'detached' from one individual and transferred to another, blurring the line between what Cohen[32] called receipts and recipient units. The second is that the human lifespan is an outcome, or as Rawls[33] argued, a 'natural good', that cannot be distributed in the same way as other 'social primary goods' since much of its distribution owes to an underlying natural, biological distribution.

Yet this reasoning misses the point that in addition to its intrinsic value, health also has instrumental value that can greatly affect one's position in life.[34] In Sen's capability framework,[31] escaping premature mortality can be thought of as a basic capability necessary for elementary functioning, alongside other basic capabilities such as the ability to be well fed, well sheltered and to escape avoidable morbidity. Additionally, while causality may at times run from income to health, the reverse possibility exists that health itself is a prime determinant of income.[35 36] If this were true than we should be equally concerned with how health was distributed within society, as it impacts on an individual's total welfare. Furthermore, despite the fact that some of the underlying distribution may have natural or genetic underpinnings, the large differences in lifespan variation between countries and populations cannot be due to natural differences alone. Studies on Danish twins revealed that only about one quarter of the total variation in age at death was attributable to genetic factors.[37 38] Thus the social context in which we live must play a role in determining how lifespans are distributed over the population.

With an aim to review *class* inequalities in health within the literature of distributive justice, Marchand et *al.*[39] identified four main strands: (1) maximizing the sum total of individual health; (2) equalizing the prospects for a long and healthy life across social groups; (3) maximin—maximizing the health status of the most disadvantaged classes; and (4) giving priority to the sickest individuals regardless of class. These could equally be modified in a non-differentiating manner to individuals, with equity in lifespans over age replacing equity in health over social class as the distribution of interest. As such, these concepts would broadly fall under the following categories: utilitarian, egalitarian, pro-poor, and achieving a minimum

threshold. The potential to apply each of these distributive justice concepts to lifespan distributions would be as follows:

Utilitarian (Maximising the sum total of health)

Maximizing approaches are rooted in utilitarian views on welfare: essentially, *the greatest happiness for the greatest number*. If we can equate happiness with living longer, applying this tenet to individual mortality would mean following strategies to maximize the total person-years lived, regardless of whether the individual was rich or poor, sick or healthy. Since maximizing the total life-years for a population is the same as maximizing the population life expectancy, the policies pursued would be the same under either concept. The equity here comes in the idea that each individual is treated in the exactly the same way. Strict utilitarian approaches are criticized for their potential for repression. If a majority group holds power or controls resources, they might pursue policies that are strictly to their benefit, at the cost of the minority being held back. So long as the total person-years are maximized, it would not matter who was receiving the benefit, or how large the inequalities were between groups.

Egalitarian (Equalizing the prospects for a long and healthy life)

This view takes as a basis that individuals are morally equal and the same life prospects, including longevity, should be available to all. Thus the inequities of a health distribution would stem from inequalities arising from outside of the individual's control,[41] Since individuals are not born into the same circumstances, egalitarian policies might direct effort toward individuals most in need of help to achieve a long life. This is similar to Sen's capability approach where priority is given to ensure that everyone is given the same capabilities in life to achieve elementary functioning.[40] Whether they choose to do so is not at issue.

Pro-poor (maximizing the lifespans of the youngest)

In age-at-death distributions the 'poorest' individuals are those who die youngest. Thus pro-poor approaches would give absolute priority to raising the survival probabilities of the youngest individuals. Gains in survivorship to the elderly, on the

other hand, would be welcome, but inconsequential in terms of judging the equity of the distribution. Renowned among pro-poor theories is Rawls' general conception of distributive justice.[33] In defending such a distributional outcome, Rawls argues that our high aversion to being in poor circumstances would lead us, if put under a 'veil of ignorance' to choose such an outcome.

Minimum threshold (prioritizing a minimum threshold achievement in lifespans)

Somewhere in between egalitarianism and pro-poor concepts, another approach could be to set some minimum threshold achievement in health. Williams' concept of *fair innings* is one such approach,[42] which is based on a view that each individual is entitled to achieve some 'normal' span of life, implying that "anyone failing to achieve this has in some sense been cheated, whilst anyone getting more than this is 'living on borrowed time'". Unlike a strict pro-poor approach which would always give priority to the least well-off, namely the youngest, a minimum threshold approach would prioritize individuals according to how they were valued in society. Williams notes that while survey responses eliciting views of such nature tend to overwhelmingly value the young over the old, older children are generally more valued than infants, while individuals caring for young children should be saved over the childless.

These four identified distributive justice strands differ primarily in their aversion to inequality. Utilitarianism is blind to the underlying age distribution provided that total person years are maximized. Egalitarianism aims for equalizing the life expectancy prospects of individuals. Finally pro-poor and minimum threshold frameworks contain specific aversions to inequality according to the value placed on saving lives at different ages. Although distributive justice concepts have not been directly linked to the age distribution of death, to a large extent they are already implicit in the ongoing debates over the age rationing of health care. In this thesis I will not be adopting any particular normative concept of inequality with regards to the lifespan distribution, but rather will aim to describe patterns of age-at-death variation across countries and within social groups.

Socioeconomic inequality

The above section recognized that part of what makes the age distribution of death of normative importance might be the extent to which lifespan variation arises beyond an individual's control. Individuals do not always choose the social circumstances in which they are raised or the socioeconomic group to which they later belong. Socioeconomic inequalities in health and mortality are regarded as being particularly unfair if they are avoidable and owing to an unjust distribution of society's resources.[43] As a result, a good part of this thesis is devoted to exploring the links between socioeconomic inequalities in mortality and lifespan variation.

In all countries that have been examined, there is a socioeconomic gradient to mortality. This applies regardless of whether income, wealth, education, occupational status or even housing tenure is used a proxy. Even living in a poorer neighbourhood is associated with a higher risk of mortality than living in a more affluent neighbourhood.[44] Although socioeconomic inequalities in mortality seem ubiquitous, the level can vary substantially by region. Within Europe, for example, inequalities in mortality were found to be lowest in some southern European countries and larger in eastern and Baltic regions.[45] Outside of Europe, relatively higher levels have been found in the United States[46,47] and Canada[48] as compared to average European levels. In Japan, the occupational gradient to self-rated health among males was similar to English and Finnish levels, but was much lower among females.[49] Unfortunately, few internationally comparable studies exist to assess the magnitude of socioeconomic inequalities across other countries.

Over the past two decades our understanding of the causal pathways linking socioeconomic factors to mortality has greatly improved. The explanations for such inequalities are varied, and can change from one setting to the next. Generally they can be divided into material factors, behavioural factors, and psychosocial factors, however these factors are often interrelated. An individual's position in the social strata may influence their housing conditions,[50,51] access to health care,[52,53] occupational health risks,[54,55] and the affordability of nutritional food and sport facilities.[56-58] Moreover, lower socioeconomic groups adopt less healthy behaviours, especially concerning cigarette smoking,[59-62] but also with regards to diet and exercise.[62-65] Although alcohol consumption is generally greater in higher socioeconomic groups, alcohol abuse has been linked to socioeconomic differences in mortality.[66-68] Finally inequalities in mortality have been linked to different levels of stress accumulated over the life course stemming from insecure employment, financial problems, general feelings of helplessness, and feelings of relative deprivation.[69-72]

To date, how socioeconomic inequalities relate to individual variation in lifespans is a topic that has not been extensively explored.

What is known about lifespan variation?

It is only in recent years that demographers have taken an interest in measuring the variation in the lifespan distribution. However there are still some hurdles to be overcome in our understanding of the concept that we would like to measure, before summarizing dispersion in lifespans is likely to become commonplace.

At the moment, no real consensus exists in the choice of measure. Although it is recognized that measures differ in their sensitivities to changes in mortality at different ages, outside of comparing trends in measures and performing age decompositions of differences,[73 74] little formal demographic work has been carried out to elicit these sensitivities. A few studies have carried out surveys of available methods used to measure the rectangularization of the survival curve—itself a concept of variation.[74-79] Mostly these studies tend to find high correlations between measures, using this as a basis to argue that the choice of measure is not an important one.

On the other hand there is a large body of literature examining ways to quantify the distribution of income.[80-86] Many of these measures and decomposition techniques could readily be applied to examine and decompose trends in lifespan variation. However, the income distribution differs considerably from the distribution of lifespans. While the upper age limits of lifespans are dictated by biological processes, the highest incomes have no such limit, and can differ rather substantially from median levels. As a result many of the familiar arguments in the economics literature for using ranking or percentile-based measures may not be as important in demography. In fact, well-known statistical measures such as the standard deviation and the variance have become popular techniques explored to measure lifespan variation.[87-90]

Empirically, it has been observed that falling lifespan variation has accompanied rising life expectancy at birth, in virtually all countries.[74] Trends at other ages have been mixed. Using all countries and years of the Human Mortality Database, a low Gini coefficient in lifespans conditional upon survival to age 15 was shown to be associated with a high remaining life expectancy at age 15.[91] This goes against trends observed in many high income countries showing stagnation in lifespan variation conditional upon survival to some adult age (roughly 10-30) since the 1960s, despite improvements in life expectancy.[19 74 77 88 90 92] Yet many of these

studies are difficult to compare because of differences in measures, time periods, and the age range examined. The starting age of the lifespan distribution examined has been especially shown to make a difference in assessments of trends in lifespan variation.[19 90 93] The role played by the use of different measures to evaluate differences over time in the age-at-death distribution is less clear.

Only a few investigations have been undertaken to account for the observed differences in lifespan variation across populations. One study found that countries achieving a level of life expectancy later than others, did so with lower levels of lifespan variation.[91] Other studies have delved into different causes-of-death, finding that the high lifespan variation in the United States is at least partly attributable to higher levels of external mortality.[88 94 95] Different levels of lifespan variation have also been found by socioeconomic subgroups such as educational groups (Russia,[73] USA[88 96]), race (USA[88]), and income (USA[88]). Meanwhile a relationship between income inequality and lifespan variation was found to be significant, but weak, over time in the United States and in England and Wales.[94] Moreover, this study suggested that over time, differences in lifespan variation within a country may come about for other reasons than differences at any given time between countries, the latter being more influenced by differences in socioeconomic characteristics.

This thesis

In this thesis I aim to undertake a comprehensive study of the variation in human lifespans. More specifically I set out to answer the following research questions:

1. What is the most appropriate way to measure variation in age-at-death?
2. What is the relationship between lifespan variation and life expectancy?
3. How much are educational differences contributing to lifespan variation?

I address these questions by splitting the thesis into three sections. Of course no study of lifespan variation can begin without addressing the concept of variation being measured, thus Section I is aimed at understanding the available tools to quantify dispersion in age at death. Using perturbation analysis, the sensitivities and elasticities of seven measures of lifespan variation are derived. These derivations are applied to empirical data, to demonstrate how the sensitivities of all measures have changed in moving from high to low mortality regimes. Finally, a new decomposition technique to determine the age contribution of mortality change to

changes in the measures over time is introduced. The measures being compared in this chapter are applied in later chapters.

Section II moves to describing macro level trends in lifespan variation. In Chapter 3, a large dataset is used to examine the relationship between life expectancy at birth and lifespan variation. Newly derived demographic equations are applied to understand why the two phenomena are so highly correlated. In Chapter 4, a specific examination of the Japanese female population is undertaken. As Japanese females are currently the world's longevity leaders, understanding recent trends in the development of its age pattern of mortality is crucial for forecasting trends in other regions.

In Section III the emphasis is put on determining the relationship between socioeconomic inequalities and lifespan variation in Europe. In Chapter 5 a comparison of adult lifespan variation by level of education is performed for 10 different European countries, to determine whether there is a socioeconomic gradient to lifespan variation. Age and cause-of-death decompositions are performed to describe the differences in the dispersion of death between low and high educated individuals. In Chapter 6, the contribution of socioeconomic inequality to lifespan variation is estimated using additive decomposition techniques. This level is compared across 11 European countries. Chapter 7 examines how this contribution might have changed over time in Lithuania and Estonia over the 1990s, given the major socioeconomic upheavals accompanying the transition to a market economy. It further disentangles the role played by an upward shift in the educational composition from direct changes to the mortality levels of the educational subgroups.

Finally in Chapter 8 this thesis concludes with a general discussion of the findings and implications of this study.

Terminology

To finish, I would just like to add a short note on terminology. Lifespan variation in the literature has been known by many names (lifespan inequality, length of life inequality, dispersion in age at death, rectangularity of the survival curve, and mortality compression to name a few). All of these terms are generally measuring the same phenomenon, but it is likely that some terms carry heavier connotations. For instance, in public health circles the term 'inequality' is generally used to describe differences in health outcomes which are often thought of as inequitable. In this thesis the aim is to describe the differences between individuals in age at death without having any further connotation. For this reason the term 'lifespan

variation' is generally used, but any other descriptions are not intended to carry any difference in meaning.

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CHAPTER 2

Perturbation analysis of measures of lifespan variability

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in prep

CHAPTER 2

Abstract

Background A number of measures have been used in recent years to calculate lifespan variation, each with different underlying properties. Although these measures are assumed to be interchangeable, little research has been conducted to show under which conditions this assumption is appropriate, or how to compare their responses to the underlying mortality schedule.

Methods We compare seven measures of lifespan variation: life disparity, the Gini coefficient, the standard deviation, the variance, Theil's index, the mean logarithmic deviation, and the inter-quartile range. We derive the sensitivity and elasticity of each measure by applying Markov chain theory and matrix calculus. Using empirical French and Russian male data we compare the underlying sensitivities to mortality change under different mortality regimes in order to test under which conditions the measures might differ in their conclusions about the magnitude of lifespan variation. Finally we demonstrate how integrating these sensitivities can be used as a method of age decomposition.

Results The measures were highly correlated and the sensitivities of the measures to mortality change followed similar general age patterns. The primary differences between the measures were in the sensitivity to infant mortality, the slope of the decline from birth to late adulthood, and in the age at which the sensitivities cross the x-axis. The interquartile range had the most qualitatively different sensitivity pattern from the others.

Conclusions This paper presents an easily computable method for calculating the properties of this important class of longevity measures. By examining the sensitivity of the measures to changes in age-specific mortality, researchers can match their choice of lifespan variation measure to their normative preferences for mortality reduction at different ages.

Introduction

Summarizing the variation in lifespans is a natural complement to describing the average length of life, giving greater insight into the age pattern of mortality. Measures of lifespan variation have been used to describe and compare the level of lifetime uncertainty across populations.[1-6] A related strand of research measures the rectangularity of the survival curve for humans and non-humans.[7-14] Finally lifespan variation measures are used to determine whether old-age mortality is being compressed, or whether these deaths are shifting to higher ages.[12 14-23] Besides the differences in research objectives, these studies are often difficult to compare both because of different age ranges being examined, as well as the different measures used to summarize the lifespan variation.

Turning to the last point, a handful of studies have compared lifespan variation measures.[5 14 16-19 24] These studies generally conclude that the measures are for the most part interchangeable due to their high correlation. Little attention has been paid to the different underlying sensitivities, or to understand when measures can be expected to disagree. Rarely are reasons for choosing one measure over another tied in to any normative concept of inequality or to social preference for the weights placed on deaths at different ages.[24-26]. A notable exception to this is the WHO attempt to quantify inequality over individuals as part of World Health Report 2000, using a Gini-like measure modified by expert opinion.[26-28]

The aim of this paper is to make the underlying formal properties of these measures explicit, allowing researchers to better tie their choice of measure to their research aims. Using perturbation analysis, we derive the analytic expressions for the derivatives of seven measures of lifespan variation, by expressing the problem in terms of an absorbing Markov chain and applying matrix calculus. We compare both the sensitivity (a response to small additive perturbation), and the elasticity (the proportional response to a small proportional perturbation) of the measures under changing age-at-death distributions. Using empirical examples we illustrate instances where these different sensitivities cause measures to disagree on the magnitude or even direction of change in lifespan variation. Finally, we demonstrate how integrating sensitivities can be used to decompose measures by age and time, using Life Table Response Experiments (LTRE).

CHAPTER 2

Measures

We make comparisons of the following measures of variability:

- Life disparity ($e^{\dagger}$)
- Gini coefficient (G)
- Theil's index (T)
- Mean logarithmic deviation (MLD)
- Standard deviation in lifespans (S)
- Variance in lifespans (V)
- Inter-quartile range (IQR)

These seven measures come from different disciplinary backgrounds. The $e^{\dagger}$ measure is a life table based measure that can be interpreted as the average remaining life expectancy at death, or alternatively the average life years lost in a population due to death. When multiplied by the life expectancy it becomes the elasticity of life expectancy with respect to mortality change,[29-31] also known as Keyfitz' H.[32] The G measure is often used in economic inequality research. It ranges from 0 to 1, with higher numbers signalling greater inequality. It is also reasonably easy to interpret demographically. It is simply the average inter-individual difference in age at death, divided by the life expectancy.[5] The T and MLD are entropy measures developed in the field of information theory by Henry Theil in the 1960s.[33] Originally intended to calculate the rate of transfer of information in a particular message, Theil himself noted the correlation with inequality measures, and was the first to apply them to economic inequality research. Unfortunately neither measure has an intuitive demographic interpretation. The well known statistical measures S, V and IQR follow a distance concept. While V is the average squared distance in age at death from the mean, S is the square root of V, measured in years. Finally IQR measures the distance in years between the 1st and 3rd age quartiles of death.

Given these different disciplinary backgrounds, a reasonable question to ask is what properties we should deem important in measuring lifespan variation. The distribution of lifespans is obviously different from the distribution of income, particularly at the upper end where death is governed by biological processes but the highest incomes can deviate a great deal from the median. The bi-modal shape of the lifespan distribution might also call into question measures that compare each individual's age at death to the mean, which particularly in historical or contemporary developing countries can differ sharply from either mode.[34] Finally

why we should interest ourselves in entropy measures is also not immediately clear. Having said that these measures were selected for inclusion in this paper because they had either desirable formal properties, were easy to calculate, or had reasonable demographic interpretations.

Different research objectives often call for usage of one measure over another due to their underlying formal properties. The *V*, *T* and *MLD* measures are all additively decomposable into between- and within-group variation.[35] This type of decomposition is used to determine how much between-group differences are accounting for the total level of lifespan variation. The *G* measure can also be decomposed in this way, but contains an overlap term.[36] The *MLD* measure can additionally be additively decomposed over time, to account for compositional change to the between- and within-group variation components.[37] The $e^{\dagger}$ measure has an interesting relationship to life expectancy: The product of $e^{\dagger}$ and the average rate of progress in reducing age specific death rates is equal to the change in life expectancy.[31]

Another consideration in choosing a measure is whether absolute inequality (the level of variation would be unaffected by additive gains to everyone's lifespan) or relative inequality (the level of variation would be unaffected by proportional gains to everyone's lifespan) should be measured. For instance when measuring the dispersion in lifespans above age 30, should the average age at death conditional upon survival (roughly ages 30-110) or the remaining life expectancy (roughly 0-80) be used? For additive measures it would not make a difference, but relative measures would find greater variation in the latter distribution. Additive measures have the advantage of being more easily interpretable, as they are normally expressed in years. An overview of these formal properties can be seen in Table 1.

Finally the sensitivity of measures to changes in mortality at different ages is perhaps the most important and least understood property of the measures. As economist Paul Allison noted: "The choice of an inequality measure is properly regarded as a choice among alternative definitions of inequality rather than a choice among alternative ways of measuring a single theoretical construct." [38]

Despite differences in backgrounds and formal properties, all seven measures can be expected to pick up most of the general patterns in lifespan variation resulting from changing age patterns of mortality. This can be seen by the high Pearson coefficient correlations between many of the included measures.[14] We have extended this to all seven measures we compare, both from birth (Table 2) and

	demographic definition	absolute or relative measure	additional formal properties
$e^{\dagger}$	average remaining life expectancy at death	absolute	equal to the change in life expectancy divided by the average rate of progress in reducing age specific death rates
G	average distance in years between each individual in age at death divided by life expectancy	relative	additively decomposable, but with overlap term
T	not intuitive	relative	additively decomposable
MLD	not intuitive	relative	additively decomposable, and over time to account for compositional change
V	average individual squared distance from mean age at death in years	absolute	additively decomposable
S	square root of variance	absolute	
IQR	distance in years between the 3rd and 1st quartile in age at death	absolute	

Table 1: Overview of the measures being compared

	$e^{\dagger}$	G	T	MLD	S	V	IQR
$e^{\dagger}$	1.000						
G	0.977	1.000					
T	0.945	0.991	1.000				
MLD	0.964	0.991	0.992	1.000			
S	0.981	0.931	0.890	0.928	1.000		
V	0.987	0.943	0.907	0.941	0.996	1.000	
IQR	0.968	0.965	0.946	0.955	0.921	0.944	1.000

Table 2. Pearson correlation coefficients between pairs of measures. Calculated over all ages (0-110+), for all female and male life tables in the Human Mortality Database (6860 in total). Data accessed 01/02/2010.

	$e^{\dagger}_{10}$	G_{10}	T_{10}	MLD_{10}	S_{10}	V_{10}	IQR_{10}
$e^{\dagger}_{10}$	1.000						
G_{10}	0.984	1.000					
T_{10}	0.979	0.995	1.000				
MLD_{10}	0.967	0.986	0.995	1.000			
S_{10}	0.986	0.958	0.961	0.952	1.000		
V_{10}	0.985	0.960	0.967	0.960	0.998	1.000	
IQR_{10}	0.981	0.978	0.978	0.969	0.958	0.965	1.000

Table 3. Pearson correlation coefficients between pairs of measures. Calculated over ages (10-110+), for all female and male life tables in the Human Mortality Database (6860 in total). Data accessed 01/02/2010.

from age 10 (Table 3), calculated over all female and male life tables currently in the Human Mortality Database.

Calculating the measures of variability

In Table 4 we present the conventional lifetable notation alongside the less familiar matrix notation for each measure. In conventional notation ℓ_y is survivorship, d_y the death density, and $\dot{e}_y$ remaining life expectancy for the age interval y to y+1. We further denote a_y as the length of the age interval lived by those who died. An overbar, for example $\bar{e}_y$, is used when adjustments to the variable are necessary to account for the portion of the age interval lived by those who died, i.e.

$$\bar{e}_y = e_y + a_y\left(e_{y+1} + e_y\right) \tag{1}$$

By this same logic, $\bar{x}_y$ is the average age at death over the interval. Generally it is the age halfway in between the two age intervals, but in the first year of life $\bar{x}_0 = a_0$. The oldest age interval is denoted by ω.

Finally in the *IQR* formula, $\hat{x}_1$ and $\hat{x}_3$ are the interpolated first and third age quartiles, at which 25 and 75 percent of the total deaths have occurred.

Expressing each measure in matrix notation was necessary for deriving the sensitivities. We denote matrices by capital letters in bold face, vectors by small letters in bold face and scalers by small letters in regular type face. A superscript $\top$ refers to the transpose of a matrix. The symbol $(\mathbf{A}\circ\mathbf{B})$ denotes the Hadamard element-by-element product of the two matrices, while $(\mathbf{A}\otimes\mathbf{B})$ is the Kronecker product. We also make use of the vec operator, which stacks columns of a matrix into a column vector. Since this study focuses on human demography, we express everything in terms of an age classified model. Nevertheless these models could be generalized for stage classified populations.

We express longevity as an absorbing Markov process, with s transient states (age classes) and a absorbing states. In an age-classified model absorbing states can be death, death by a certain disease, or death classified by any other status. The transition matrix of the Markov chain is given by

$$\mathbf{P} = \begin{pmatrix} \mathbf{U} & 0 \\ \mathbf{M} & \mathbf{I} \end{pmatrix} \tag{2}$$

	conventional LT notation	matrix notation
$e^{\dagger}$	$\sum_{y=0}^{\omega} d_y \overline{e}_y$	$\mathbf{f}^{\top}\eta$
G	$1-\frac{1}{e_0}\sum_{y=0}^{\omega}\ell_{y+1}^2+a_y\left(\ell_y^2-\ell_{y+1}^2\right)$	$1-\frac{1}{\eta_1}\mathbf{e}^{\top}\left[(\mathbf{e}-\mathbf{Cf})\circ(\mathbf{e}-\mathbf{Cf})\right]$
T	$\sum_{y=0}^{\omega} d_y\left(\frac{\overline{x}_y}{e_0}\ln\frac{\overline{x}_y}{e_0}\right)$	$\mathbf{e}^{\top}\operatorname{diag}(\mathbf{f})\frac{1}{\eta_1}\mathbf{x}\ln\left(\frac{1}{\eta_1}\mathbf{xe}\right)$
MLD	$\sum_{y=0}^{\omega} d_y\left(\ln\frac{e_0}{\overline{x}_y}\right)$	$\mathbf{e}^{\top}\operatorname{diag}(\mathbf{f})\ln\left(\eta_1\operatorname{diag}\left(\frac{1}{\mathbf{x}}\right)\mathbf{e}\right)$
V	$\sum_{y=0}^{\omega} d_y\left(\overline{x}_y-e_0\right)^2$	$\left[\mathbf{e}^{\top}\mathbf{N}(2\mathbf{N}-\mathbf{I})-\eta^{\top}\circ\eta^{\top}\right]^{\top}$
S	$\sqrt{V}$	$\sqrt{V}$
IQR	$\hat{x}_3-\hat{x}_1$	$\hat{x}_3-\hat{x}_1$

Table 4. Formulas for calculating measures in conventional life table formulation (discrete, assuming life table radix of 1) and their equivalent formulation in matrix notation.

where $\mathbf{U}$ is an $s \times s$ matrix with the age-specific survival probabilities on the sub-diagonal and zeros elsewhere. The zero in the (s, s) position of $\mathbf{U}$ is analogous to having a probability of death, $q_x = 1$, in the open-aged interval of a lifetable to close it off. The $a \times s$ matrix $\mathbf{M}$ gives the probability of death at each absorbing age, and $\mathbf{I}$ is the $a \times a$ identity matrix. It can thus be readily seen that the columns of $\mathbf{P}$ sum to one. The fundamental $s \times s$ matrix $\mathbf{N}$ represents how long it takes for absorption

$$\mathbf{N} = (\mathbf{I} - \mathbf{U})^{-1} \tag{3}$$

We denote life expectancy by η, which is not to be confused with $\mathbf{e}$, a column vector of ones, length s, used for summations ($\mathbf{e}_1$ is the first element of this vector). Summing the columns of $\mathbf{N}$ we then get the following expression for life expectancy

$$\eta = \mathbf{e}^{\top}\mathbf{N} \tag{4}$$

The first element of η is life expectancy at birth (or age at which the lifetable begins), denoted as η_1.

The age distribution of death is

$$\mathbf{B} = \mathbf{MN} \tag{5}$$

where $\mathbf{f} = \mathbf{B}\mathbf{e}_1$ is simply the age at death distribution from birth.

The vector $\mathbf{x}$ contains the average age at death in the age interval (i.e. for French males in 2005 it is $\{0.06, 1.5, 2.5, ..., 109.5, 111.32\}$.

In *G* calculations, $\mathbf{C}$ is an $s \times s$ matrix for making cumulative sums, containing ones on the diagonal and below, and zeros elsewhere:

$$\mathbf{C} = \begin{pmatrix} 1 & 0 & & 0 \\ 1 & \ddots & \ddots & \vdots \\ \vdots & \ddots & \ddots & 0 \\ 1 & & 1 & 1 \end{pmatrix} \tag{6}$$

The column vector of survivorship ℓ, which assumes a life table radix of 1, is calculated as

$$\ell = \mathbf{e} - \mathbf{Cf} \tag{7}$$

Calculating sensitivity and elasticity

Perturbation analysis was first introduced to demography in the 1970s in assessing the sensitivity of life expectancy to changes in the underlying mortality rates.[8 32 39 40] In recent years this work has been extended and further life table relationships have been derived.[29-31 41 42] Widespread usage of perturbation analysis in demography, however, was somewhat limited by the complexity in deriving the analytic expressions for the derivatives of different measures and in its ability to handle complexities in life history. Expressing the problem in terms of an absorbing Markov chain and applying matrix calculus has expanded the possibilities.[43-47]

The sensitivity of y to a perturbation parameter, θ, is $\frac{dy}{d\theta}$, and the elasticity of y to θ is $\frac{\theta}{y}\frac{dy}{d\theta}$. To assess respectively the absolute and proportional effects on the measures from changes in the underlying mortality rates we needed the analytic expressions for the sensitivity and elasticity of the seven measures of lifespan variation with respect to mortality. The sensitivity of $e^{\dagger}$ was first derived by Zhang and Vaupel in an age-classified model.[48] This was later generalized to an age and stage classified model by Caswell,[46] who also derived expressions for the sensitivity and elasticity of the variance and the standard deviation.[45] The other expressions were newly derived for this paper.

Calculating the elasticity of any measure, y, to a vector of age-specific mortality rates, θ, on which $\mathbf{U}$ and $\mathbf{M}$ depend is

$$\frac{\in y}{\in \theta^{\top}} = \mathrm{diag}(y)^{-1}\frac{dy}{d\theta^{\top}}\mathrm{diag}(\theta) \qquad (8)$$

The formulas themselves are not terribly intuitive, but are easily calculated. We performed all calculations in MATLAB 7.3.0 and would be happy to share the code. Deriving the sensitivity and elasticity of each measure to mortality was done using traditional matrix differentiation techniques.[49] These techniques are also given extensive treatment in recent papers by Caswell, using most of the same notation that we have here.[45 46] The derivation of the sensitivities of *G*, *MLD*, *T* and *IQR* to mortality can be found in the appendix, with the sensitivity expressions for all measures listed in Table 5.

We now turn to the demographic applications, especially in comparing the sensitivities of these measures, examining how they have changed over time as we

Sensitivity of measure	
$e^{\dagger}$	$\left[\left(\mathbf{f}^{\top}\mathbf{N}^{\top}\otimes\eta^{\top}\right)+\left(\mathbf{e}_1^{\top}\mathbf{N}^{\top}\otimes\eta^{\top}\mathbf{B}\right)\right]\frac{d\,\mathrm{vec}\,\mathbf{U}}{d\theta^{\top}}+\left(\mathbf{e}_1^{\top}\mathbf{N}^{\top}\otimes\eta^{\top}\right)\frac{d\,\mathrm{vec}\,\mathbf{M}}{d\theta^{\top}}$
G	$\frac{1}{\eta_1^2}\mathbf{e}^{\top}(\ell\circ\ell)\left(\mathbf{e}_1^{\top}\mathbf{N}^{\top}\otimes\mathbf{e}^{\top}\mathbf{N}\right)\frac{d\,\mathrm{vec}\,\mathbf{U}}{d\theta^{\top}}+\frac{2}{\eta_1}\mathbf{e}^{\top}\,\mathrm{diag}(\ell)\mathbf{C}\frac{d\mathbf{f}}{d\theta^{\top}}$
T	$\frac{1}{\eta_1}\left[\left(\ln\left(\frac{1}{\eta_1}\mathbf{x}\mathbf{e}\right)\right)^{\top}\mathbf{x}\otimes\mathbf{e}^{\top}\right]\mathrm{diag}(\mathrm{vec}\,\mathbf{I})(\mathbf{e}\otimes\mathbf{I})\frac{d\mathbf{f}}{d\theta^{\top}}-\frac{1}{\eta_1}T\frac{d\eta_1}{d\theta^{\top}}-\frac{1}{\eta_1^2}\mathbf{e}^{\top}\,\mathrm{diag}(\mathbf{f})\mathbf{x}\mathbf{e}\frac{d\eta_1}{d\theta^{\top}}$
MLD	$\left[\ln\left(\mathbf{e}^{\top}\eta_1\,\mathrm{diag}\left(\frac{1}{\mathbf{x}}\right)\right)\otimes\mathbf{e}^{\top}\right]\mathrm{diag}(\mathrm{vec}\,\mathbf{I})(\mathbf{e}\otimes\mathbf{I})\frac{d\mathbf{f}}{d\theta^{\top}}+\mathbf{e}^{\top}\,\mathrm{diag}(\mathbf{f})\mathbf{e}\frac{1}{\eta_1}\frac{d\eta_1}{d\theta^{\top}}$
V	$\left[2\left(\mathbf{N}^{\top}\otimes\mathbf{e}^{\top}\right)+2\left(\mathbf{I}\otimes\mathbf{e}^{\top}\mathbf{N}\right)-\left(\mathbf{I}\otimes\mathbf{e}^{\top}\right)-2\left(\mathrm{diag}(\eta)\otimes\mathbf{e}^{\top}\right)\right]\left(\mathbf{N}^{\top}\otimes\mathbf{N}\right)\frac{d\,\mathrm{vec}\,\mathbf{U}}{d\theta^{\top}}$
S	$\frac{1}{2}\mathrm{diag}(S)^{-1}\frac{dV}{d\theta^{\top}}$
IQR	$\frac{d\hat{x}_3}{d\theta^{\top}}-\frac{d\hat{x}_1}{d\theta^{\top}}$

Table 5. Sensitivity of measures, in matrix notation. The derivation of the sensitivity of *T, MLD, G* and *IQR* can be found in the appendix.

have moved from high to low mortality regimes, and using the sensitivities as a decomposition method.

Comparing the underlying sensitivities of the measures

We used French male data to broadly illustrate the underlying sensitivities and elasticities of each measure. We calculated the measures under four very different mortality regimes: high mortality (1888), medium mortality (1948), low mortality (2005) and war/epidemic year (1918). The latter distribution is interesting as the second mode is around young adulthood, and the distribution has a long right tail instead of the long left tail. To help visualize these differences, all four distributions are plotted in Figure 1.

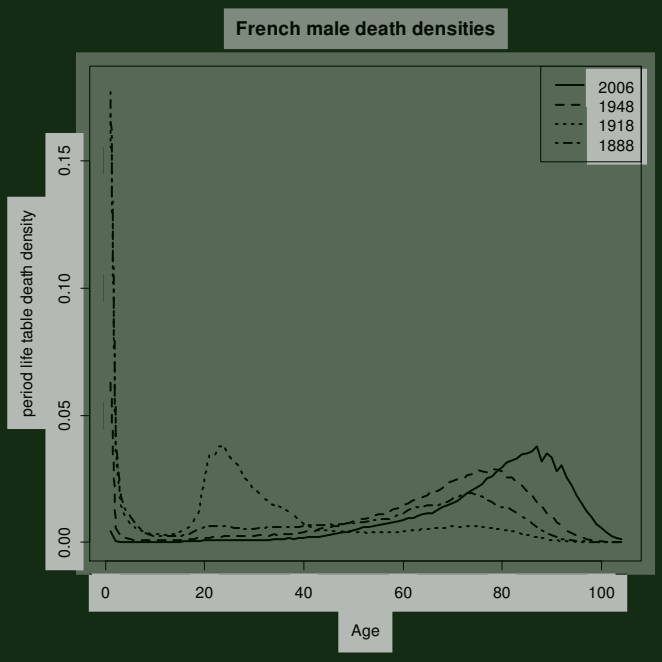

Figure 1: The age at death distributions of French males over the four different mortality regimes that we examine.

We standardized the sensitivity of each measure to its initial value, i.e. $\frac{1}{y_0}\frac{dy}{d\theta}$, to make them comparable. In a conventional plot, differences in the sensitivity and elasticity of the measures over adult ages are obscured by the high sensitivity to infant mortality, which are presented for the most recent period (Figure 2). This might lead one to believe that interventions to reduce adult mortality are unimportant for reducing lifespan variation. However, such interventions would generally impact a range of ages, unlike interventions in prenatal care, which might impact infant mortality alone. To better make out the differences in the sensitivity of measures to mortality over adult ages, we separately plotted ages 0 to 4 (Figure 3a) from ages 5 to 105+ (Figure 3b).

We also plotted the elasticities of each measure to mortality change in Figure 4a (ages 0 to 4) and Figure 4b (ages 5 to 105+). Given the different units for each measure, elasticities are generally easier to interpret, as they are simply the proportional change in the measure from a one percent change in mortality at each age.

As we would expect from the high correlations between measures, the sensitivities follow similar general age patterns. The primary differences were in the sensitivity to infant mortality, the slope of the decline from birth to late adulthood, and in the age at which the sensitivities cross the x-axis. Improvements in mortality below this age reduce lifespan variation, while improvements after this age increase the variation. The age itself has been termed the threshold age or $a^\dagger$ due to its original derivation for the $e^\dagger$ measure.[48] This age has pushed itself out to later and later ages with time, and the differences between threshold ages of the measures have considerably diminished.

Conditioning the measures upon survival to age 10 only resulted in minor changes to the pattern of sensitivity to mortality at different ages—although it did remove some of the differences between measures found when examined from birth (results not shown). This was particular the case for the *MLD* and *T* measures which are highly sensitive to changes at birth, so much so that changes at other ages were largely masked.

The *IQR* measure produced the most unique sensitivity patterns. It is only sensitive to transfers between quartiles and not to transfers within quartiles. Transfers of course are an awkward concept in mortality research, particularly as there are no finite life years that need to be distributed within the population. But in practice the idea of age rationing in health care, sacrificing facilities and medicine for older individuals to save younger individuals, comes close.

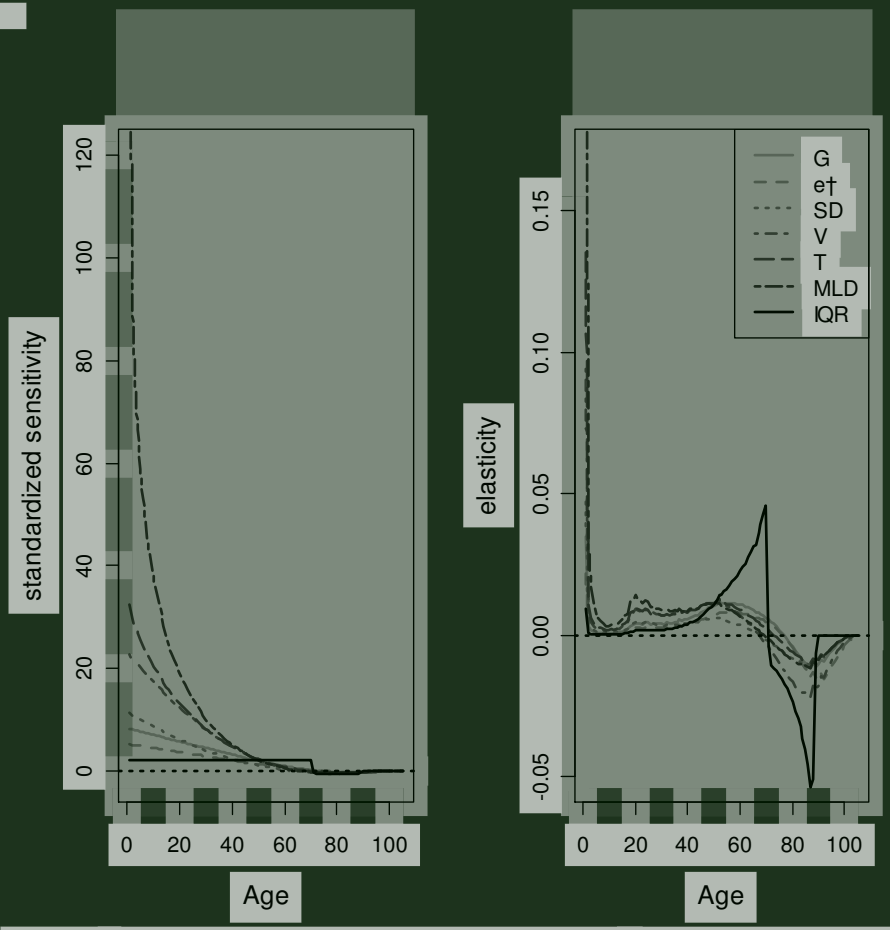

Figure 2: The sensitivity and elasticity of the measures to changes in mortality for the French male 2006 period age-at-death distribution.

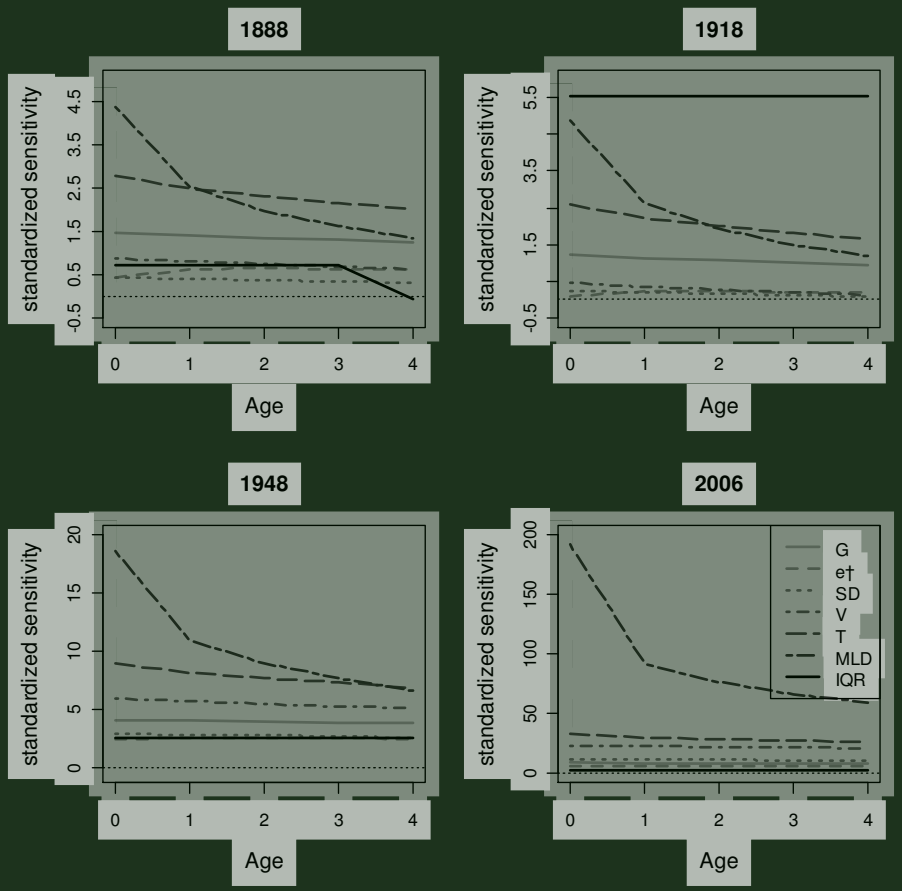

Figure 3a: The sensitivity of each measure to changes in mortality over young ages

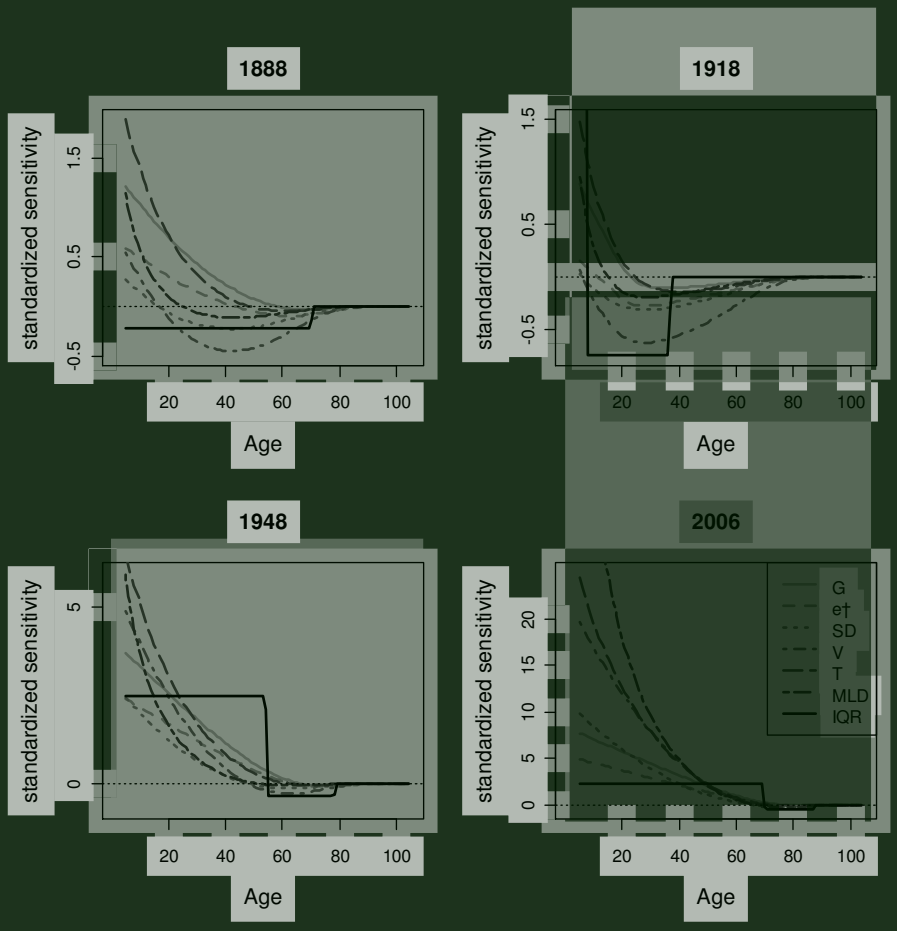

Figure 3b: The sensitivity of each measure to changes in mortality over ages 5 to 105.

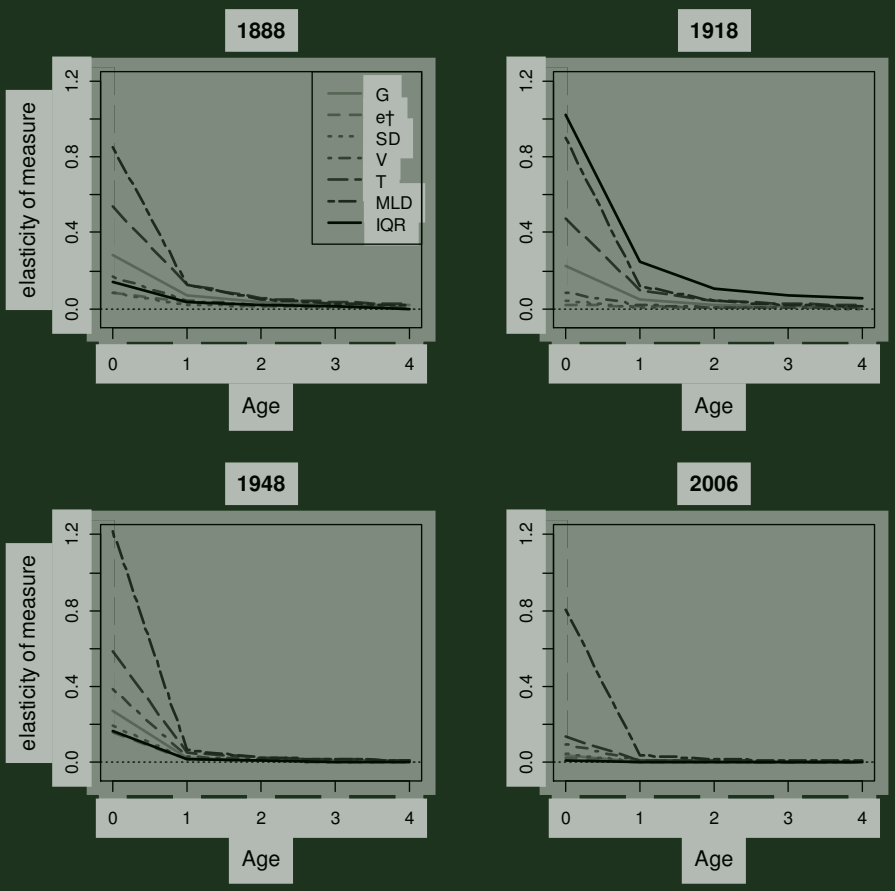

Figure 4a: The elasticity of each measure to changes in mortality over ages 5 to 105.

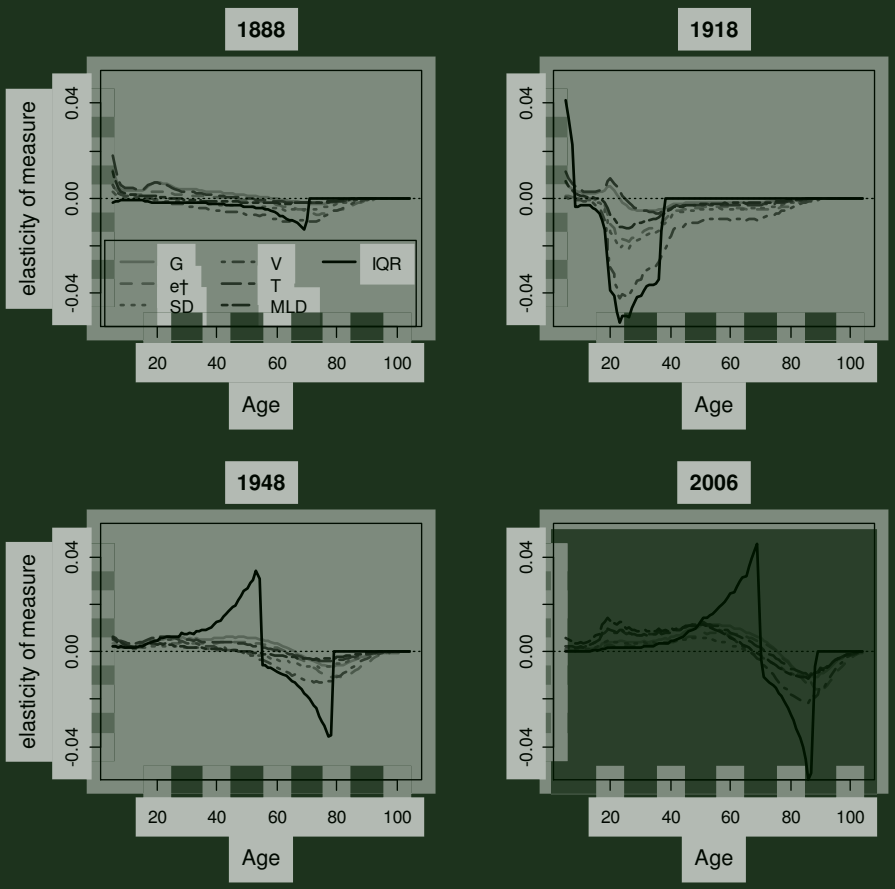

Figure 4b: The elasticity of each measure to changes in mortality over ages 5 to 105.

Decomposing differences over time

Another reason to calculate the sensitivities of measures is to perform Life Table Response Experiment (LTRE) decompositions. This type of decomposition was initially introduced to decompose growth rates into contributions from age-specific survival and reproductive rates.[50] It can be extended to any demographic statistic for which the sensitivity of the underlying vital rates on which it depends can be determined. Further examples are given in Chapter 10 of Caswell's book.[43]

In this case we needed the derivative of the measure of lifespan variability, y, with respect to time, t, which again depended on the θ vector of age-specific death rates with s age classes

$$\frac{dy}{dt} = \sum_{i=1}^{s} \frac{dy}{d\theta_i} \frac{d\theta_i}{dt} \qquad (9)$$

The time derivate of mortality $\frac{d\theta}{dt}$ can be numerically derived using statistical software such as the MATLAB function 'gradient'.

We applied this decomposition method to Russian male mortality data, 1958 to 2006, by integrating the yearly contributions obtained using equation 9. The differential age pattern of mortality change experienced by Russian males makes them an interesting example to examine how measures differ in their sensitivity.[5 24] During this period infant mortality declined substantially, from around 47 to 12 deaths per thousand live births. This decline was particularly rapid in the first 10 years. Meanwhile, adult mortality over ages 20 to 70 fluctuated a great deal, especially in the 40 to 50 age range. Until the middle of the 1980s mortality over these ages mostly experienced a slow but steady increase. Then it rapidly declined between 1984 and 1987 following the anti-alcohol campaigns, only to rise steeply with the mortality crisis brought on by the upheavals of transition.[51]

In Figure 4 we compared the change in each measure to its level in 1959, for measures at birth and at age 10. Apart from the *IQR* all measures found that lifespan variation over the entire age range decreased during the period. The high sensitivity of some measures to infant mortality, particularly *MLD*, *T* and *V*, is illuminated by the contrast between the two panels. Most of the measures conditioned upon survival to age 10 showed increased lifespan variation compared to the first year over most of the period, with fluctuation around the 1984 to 1987

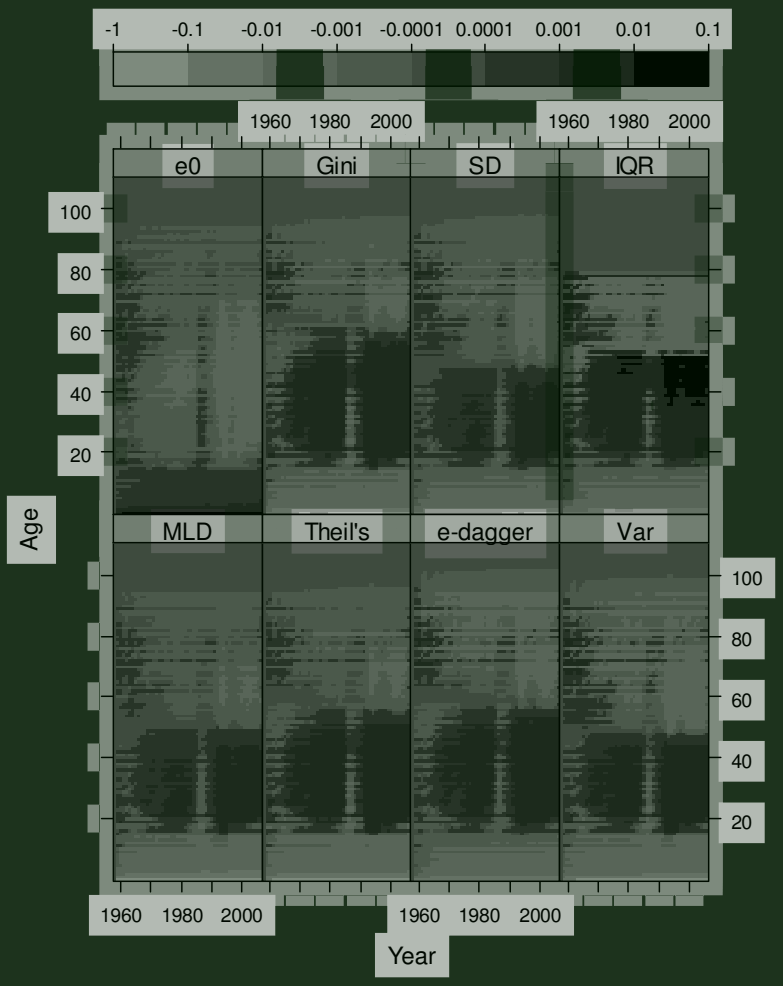

Figure 6: The proportional contribution of the change in life expectancy, and in each measure by age and year, as compared to the first year. Note that the colour scale changes by a factor of 10. This was done because the high sensitivity of all measures to infant mortality masked the contributions from any other age. The calculations were made using the LTRE decomposition method on period lifetable data for Russian males, 1958-2006, from the Human Mortality Database. Data accessed 01/02/2010.

mortality reduction period, and expansion over the later mortality crisis. Also, the differences between measures were less pronounced in the second panel.

The yearly age contributions to the change in the measures calculated from birth are presented in Figure 5. Again the contribution from reductions in infant mortality differed sharply across measures. Over middle adult ages, *IQR*, *G* and *T* were most sensitive to the increases. The generally lower threshold age of *S* meant that the increased mortality over about age 50 actually worked to reduce lifespan variation. Meanwhile the insensitivity to changes in mortality above the third quartile meant that mortality changes above age 80 had no effect on the *IQR*.

Conclusion

We compared seven measures of lifespan variation, all of which largely correlated with one another over the mortality schedules found in the 6860 lifetables of the Human Mortality Database. Using matrix differentiation techniques we derived the expressions for the sensitivities and elasticities of all measures. We compared these sensitivities under different age-at-death profiles and related changes over time in the measures to the underlying sensitivities through a LTRE decomposition.

The aim of this paper was not to come out in favour of any one method of measuring lifespan variation but rather to make explicit the differences in the underlying sensitivities of each measure to age-specific mortality. This is essential for formulating any larger normative concept of inequality or variation. It is also clear from this analysis that some measures are better suited to certain tasks than others. The *MLD*, *T* and *V* measures are so sensitive to infant mortality that they are not ideal candidates for studies over the entire age range, if adult mortality is also of interest. In comparing distributions above childhood, however, they become more suitable measures, particularly if there is a strong aversion to death at younger versus older ages. The *IQR* differs the most from the other measures. Although it has great intuitive appeal, it can be expected to deviate from the other six measures of variation the most often. Moreover, at times it can have a qualitatively different sensitivity pattern from the other six measures. Thus caution should be taken when using this measure. Unless a clearly defined concept of variation is specified outright, we would recommend using two or more measures with different sensitivity patterns before coming to any strong conclusions about the magnitude or direction of change in lifespan variability.

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CHAPTER 2

Chapter 2 Appendix - Deriving the sensitivities of measures

In this section we begin with a summary of matrix calculus techniques used to derive the sensitivities of the measures of lifespan variation, followed by the derivation of *G*, *T*, *MLD* and *IQR*. The sensitivities of *V*, *S* and $e^{\dagger}$ can be found in Caswell's recent papers.[45 46]

1. Matrix calculus preliminaries

The equations listed in Table 3 are a mixture of scalars, vectors and matrices. We will make use of the following relationships between the three. The derivative of scalar y to scalar x is the familiar $\frac{dy}{dx}$. If $\mathbf{y}$ is a $n\times 1$ vector and x is a scalar, then the derivative of $\mathbf{y}$ to x is the n x 1 vector.

$$\frac{d\mathbf{y}}{dx}=\begin{pmatrix}\frac{dy_1}{dx}\\ \vdots \\ \frac{dy_n}{dx}\end{pmatrix} \tag{1}$$

The derivative of a scalar y with respect to a $m\times 1$ vector $\mathbf{x}$ is the $1\times m$ gradient vector

$$\frac{dy}{d\mathbf{x}^{\top}}=\left(\frac{\partial y}{dx_1}\cdots\frac{\partial y}{dx_m}\right). \tag{2}$$

The derivative of the $n\times 1$ vector $\mathbf{y}$ to the $m\times 1$ vector $\mathbf{x}$ is the $n\times m$ Jacobian matrix

$$\frac{d\mathbf{y}}{d\mathbf{x}^{\top}}=\left(\frac{dy_i}{dx_j}\right). \tag{3}$$

The derivatives of matrices can be computed by transforming matrices into column vectors using the vec operator and applying the previous equations. In this way the derivative of the $m\times n$ matrix $\mathbf{Y}$ to the $p\times q$ matrix $\mathbf{X}$ is the $mn\times pq$ matrix

$$\frac{d\mathbf{Y}}{d\mathbf{X}}=\frac{d\,\mathrm{vec}\,\mathbf{Y}}{d\,\mathrm{vec}^{\top}\mathbf{X}}. \tag{4}$$

For notational simplicity we denote $(d\,\mathrm{vec}\,\mathbf{X})^{\top}$ as $d\,\mathrm{vec}^{\top}\mathbf{X}$.

The chain rule can also be applied in matrix calculus. If $\mathbf{Y}$ is a function of $\mathbf{X}$, and $\mathbf{X}$ is a function of $\mathbf{Z}$ then

$$\frac{d\mathrm{vec}\mathbf{Y}}{d\,\mathrm{vec}^{\top}\mathbf{Z}} = \frac{d\mathrm{vec}\mathbf{Y}}{d\,\mathrm{vec}^{\top}\mathbf{X}}\frac{d\mathrm{vec}\mathbf{X}}{d\,\mathrm{vec}^{\top}\mathbf{Z}}. \tag{5}$$

Matrix derivatives are often constructed by forming differentials, where the differential of a matrix (or vector) is the matrix (or vector) of differentials of the elements; i.e.

$$d\mathbf{X} = \left(dx_{i,j}\right). \tag{6}$$

If, for some matrix $\mathbf{Q}$, it can be shown that

$$d\mathbf{y} = \mathbf{Q}d\mathbf{x} \tag{7}$$

then according to the "first identification theorem" of Magnus and Neudecker[52]

$$\frac{d\mathbf{y}}{d\mathbf{x}^{\top}} = \mathbf{Q}. \tag{8}$$

Finally at times we rearranged our equations to make use of a number of well-known techniques. If $\mathbf{Y} = \mathbf{ABC}$ then Roth[53] showed that the vec operator is related to the Kronecker product by

$$\mathrm{vec}\,\mathbf{Y} = \left(\mathbf{C}^{\top}\otimes\mathbf{A}\right)\mathrm{vec}\,\mathbf{B}. \tag{9}$$

Meanwhile,

$$\left(\mathbf{A}\otimes\mathbf{B}\right)\left(\mathbf{C}\otimes\mathbf{D}\right) = \mathbf{AC}\otimes\mathbf{BD}. \tag{10}$$

whenever $\mathbf{AC}$ and $\mathbf{BD}$ are defined.

2. Differentiating measures

2.1 Preliminaries

Differentiating the various measures made use of the following sensitivities.

The sensitivity of life expectancy with respect to mortality is,[45]

$$\frac{d\eta}{d\theta^{\top}} = \left(\mathbf{I}\otimes\mathbf{e}^{\top}\right)\left(\mathbf{N}^{\top}\otimes\mathbf{N}\right)\frac{d\,\mathrm{vec}\,\mathbf{U}}{d\theta^{\top}}. \tag{11}$$

The sensitivity of the death density with respect to mortality is,[46]

$$\frac{d\mathbf{f}}{d\theta^{\top}} = -\left(\mathbf{e}_1^{\top}\mathbf{N}^{\top}\otimes\mathbf{I}\right)\operatorname{diag}\left(\operatorname{vec}\mathbf{I}\right)\left(\mathbf{e}^{\top}\otimes\mathbf{I}\right)\frac{d\mathbf{p}}{d\theta^{\top}}$$
$$+\left(\mathbf{e}_1^{\top}\otimes\mathbf{M}\right)\left(\mathbf{N}^{\top}\otimes\mathbf{N}\right)\frac{d\,\overline{\operatorname{vec}}\,\mathbf{U}}{d\theta^{\top}}. \qquad (12)$$

2.2 Gini coefficient

The expression for the Gini coefficient is

$$G = 1-\frac{1}{\eta_1}\mathbf{e}^{\top}\left[\left(\mathbf{e}-\mathbf{C}\mathbf{f}\right)\circ\left(\mathbf{e}-\mathbf{C}\mathbf{f}\right)\right]. \qquad (13)$$

In deriving the sensitivity of G, we first replace the $(\mathbf{e}-\mathbf{C}\mathbf{f})$ part of equation 13 with ℓ for notational simplicity

$$G = 1-\frac{1}{\eta_1}\mathbf{e}^{\top}\left(\ell\circ\ell\right). \qquad (14)$$

We differentiate equation 14, making use of the product rule

$$-dG = d\frac{1}{\eta_1}\mathbf{e}^{\top}\left(\ell\circ\ell\right)+\frac{1}{\eta_1}\mathbf{e}^{\top}\left(d\ell\circ\ell\right)+\frac{1}{\eta_1}\mathbf{e}^{\top}\left(\ell\circ d\ell\right). \qquad (15)$$

Meanwhile, the differential of $\frac{1}{\eta_1}$ is

$$d\frac{1}{\eta_1} = \frac{-1}{\eta_1^2}d\eta_1. \qquad (16)$$

Substituting equation 16 into expression 15 and simplifying gives us

$$-dG = -\frac{1}{\eta_1^2}(d\eta_1)\mathbf{e}^{\top}\left(\ell\circ\ell\right)+\frac{2}{\eta_1}\mathbf{e}^{\top}\left(d\ell\circ\ell\right). \qquad (17)$$

We next applied the vec operator (equation 9)

$$-dG = -\frac{1}{\eta_1^2}\mathbf{e}^{\top}\left(\ell\circ\ell\right)(d\eta_1)+\frac{2}{\eta_1}\left[\operatorname{vec}\left(\mathbf{e}^{\top}\left(d\ell\circ\ell\right)\right)\right] \qquad (18)$$

$$= -\frac{1}{\eta_1^2}\mathbf{e}^{\top}\left(\ell\circ\ell\right)(d\eta_1)+\frac{2}{\eta_1}\left[\mathbf{e}^{\top}\operatorname{vec}\left(d\ell\circ\ell\right)\right] \qquad (19)$$

$$= -\frac{1}{\eta_1^2}\mathbf{e}^\top(\ell \circ \ell)(d\eta_1) + \frac{2}{\eta_1}\mathbf{e}^\top \operatorname{diag}(\ell)\, d\ell \,. \tag{20}$$

Finally, remembering that $\ell = (\mathbf{e} - \mathbf{C}\mathbf{f})$ the differential of ℓ is

$$d\ell = -\mathbf{C}d\mathbf{f} \,. \tag{21}$$

We substitute equations 11 and 21 into equation 20 and apply equation 8. For computational purposes, we found that it worked better to first apply equation 10 to equation 11, giving us this expression for the sensitivity of G

$$-\frac{dG_1}{d\theta^\top} = -\frac{1}{\eta_1^2}\mathbf{e}^\top(\ell \circ \ell)\left(\mathbf{e}_1^\top\mathbf{N}^\top \otimes \mathbf{e}^\top\mathbf{N}\right)\frac{d\,\mathrm{vec}\,\mathbf{U}}{d\theta^\top} - \frac{2}{\eta_1}\mathbf{e}^\top \operatorname{diag}(\ell)\mathbf{C}\frac{d\mathbf{f}}{d\theta^\top} \,. \tag{22}$$

2.3 Mean Logarithmic Deviation

The mean logarithmic deviation is calculated in the following way

$$MLD = \mathbf{e}^\top \operatorname{diag}(\mathbf{f})\, ln\left(\eta_1 \operatorname{diag}\left(\frac{1}{\mathbf{x}}\right)\mathbf{e}\right). \tag{23}$$

For notational simplicity we replace the $\operatorname{diag}\left(\frac{1}{\mathbf{x}}\right)$ term in equation 23 with $\mathbf{Y}$

$$MLD = \mathbf{e}^\top \operatorname{diag}(\mathbf{f})\, ln(\eta_1 \mathbf{Y}\mathbf{e}). \tag{24}$$

Differentiating equation 24 gives us

$$dMLD = \mathbf{e}^\top d\left(\operatorname{diag}(\mathbf{f})\right) ln(\eta_1 \mathbf{Y})\mathbf{e} + \mathbf{e}^\top \operatorname{diag}(\mathbf{f})\frac{1}{\eta_1} d\eta_1 \mathbf{e}\mathbf{e}^\top\mathbf{e}. \tag{25}$$

In equation 25, we have the term $d\left(\operatorname{diag}(\mathbf{f})\right)$. First, $\operatorname{diag}(\mathbf{f})$ can be written as

$$\operatorname{diag}(\mathbf{f}) = \mathbf{I} \circ (\mathbf{f}\mathbf{e}^\top), \tag{26}$$

while its differential is

$$d\left(\operatorname{diag}(\mathbf{f})\right) = \mathbf{I} \circ (d\mathbf{f}\mathbf{e}^\top). \tag{27}$$

Substituting equation 27 into equation 25 and simplifying gives us

$$dMLD = \mathbf{e}^{\top}\left[\mathbf{I} \circ \left(d\mathbf{f}\mathbf{e}^{\top}\right)\right] \ln\left(\eta_1 \mathbf{Y}\mathbf{e}\right) + \mathbf{e}^{\top} \operatorname{diag}\left(\mathbf{f}\right)\mathbf{e}\frac{1}{\eta_1} d\eta_1 . \tag{28}$$

We apply the vec operator (equation 9) to equation 28, bearing in mind that the transpose of $\mathbf{Y}$ is itself

$$dMLD = \left[\ln\left(\mathbf{e}^{\top}\eta_1 \mathbf{Y}\right) \otimes \mathbf{e}^{\top}\right] vec\left[\mathbf{I} \circ \left(d\mathbf{f}\mathbf{e}^{\top}\right)\right] + \mathbf{e}^{\top} \operatorname{diag}\left(\mathbf{f}\right)\mathbf{e}\frac{1}{\eta_1} d\eta_1 . \tag{29}$$

Meanwhile, the differential of $\ln\left(\eta_1 \mathbf{Y}\right)$ term in equation 29 is

$$d\ln\left(\eta_1 \mathbf{Y}\right) = \frac{1}{\eta_1} d\eta_1 \mathbf{e}\mathbf{e}^{\top} . \tag{30}$$

Additionally, using equation 9, the $\operatorname{vec}\left[\mathbf{I} \circ \left(d\mathbf{f}\mathbf{e}^{\top}\right)\right]$ term can be simplified to

$$\operatorname{vec}\left[\mathbf{I} \circ \left(d\mathbf{f}\mathbf{e}^{\top}\right)\right] = \operatorname{diag}\left(\operatorname{vec}\mathbf{I}\right) \operatorname{vec}\left(d\mathbf{f}\mathbf{e}^{\top}\right) \tag{31}$$

$$= \operatorname{diag}\left(\operatorname{vec}\mathbf{I}\right)\left(\mathbf{e} \otimes \mathbf{I}\right) d\mathbf{f} . \tag{32}$$

Substituting equations 30 and 32 back into equation 29, and applying equation 8 gives us our final expression for the sensitivity of the *MLD*

$$\frac{dMLD}{d\theta^{\top}} = \left[\ln\left(\mathbf{e}^{\top}\eta_1 \operatorname{diag}\left(\frac{1}{\mathbf{x}}\right)\right) \otimes \mathbf{e}^{\top}\right] \operatorname{diag}\left(\operatorname{vec}\mathbf{I}\right)\left(\mathbf{e} \otimes \mathbf{I}\right) \frac{d\mathbf{f}}{d\theta^{\top}} + \mathbf{e}^{\top} \operatorname{diag}\left(\mathbf{f}\right)\mathbf{e}\frac{1}{\eta_1}\frac{d\eta_1}{d\theta^{\top}} . \tag{33}$$

2.4. Theil's index

The expression for Theil's index is

$$T = \mathbf{e}^{\top} \operatorname{diag}\left(\mathbf{f}\right)\frac{1}{\eta_1}\mathbf{x}\ln\left(\frac{1}{\eta_1}\mathbf{x}\mathbf{e}\right) \tag{34}$$

Differentiating this expression gives us

$$dT = \mathbf{e}^{\top}\left[d\left(\operatorname{diag}\left(\mathbf{f}\right)\right)\right]\frac{1}{\eta_1}\mathbf{x}\ln\left(\frac{1}{\eta_1}\mathbf{xe}\right) + \mathbf{e}^{\top}\operatorname{diag}\left(\mathbf{f}\right)\left[d\frac{1}{\eta_1}\right]\mathbf{x}\ln\left(\frac{1}{\eta_1}\mathbf{xe}\right) + \mathbf{e}^{\top}\operatorname{diag}\left(\mathbf{f}\right)\frac{1}{\eta_1}\mathbf{x}\left[d\ln\left(\frac{1}{\eta_1}\mathbf{xe}\right)\right]. \quad (35)$$

We substitute equation 27 into the first term of equation 35 and rearrange it to make it easier to apply Roth's theorem in the next step

$$dT = \frac{1}{\eta_1}\mathbf{e}^{\top}\left[\mathbf{I}\circ\left(d\mathbf{f}\mathbf{e}^{\top}\right)\right]\mathbf{x}\left[\ln\left(\frac{1}{\eta_1}\mathbf{xe}\right)\right] + \mathbf{e}^{\top}\operatorname{diag}\left(\mathbf{f}\right)\left[d\frac{1}{\eta_1}\right]\mathbf{x}\ln\left(\frac{1}{\eta_1}\mathbf{xe}\right) + \mathbf{e}^{\top}\operatorname{diag}\left(\mathbf{f}\right)\frac{1}{\eta_1}\mathbf{x}\left[d\ln\left(\frac{1}{\eta_1}\mathbf{xe}\right)\right]. \quad (36)$$

We apply the vec operator from equation 9

$$dT = \frac{1}{\eta_1}\left[\left(\ln\left(\frac{1}{\eta_1}\mathbf{xe}\right)\right)^{\top}\mathbf{x}\otimes\mathbf{e}^{\top}\right]\operatorname{vec}\left[\mathbf{I}\circ\left(d\mathbf{f}\mathbf{e}^{\top}\right)\right] + \mathbf{e}^{\top}\operatorname{diag}\left(\mathbf{f}\right)\left[d\frac{1}{\eta_1}\right]\mathbf{x}\ln\left(\frac{1}{\eta_1}\mathbf{xe}\right) + \mathbf{e}^{\top}\operatorname{diag}\left(\mathbf{f}\right)\frac{1}{\eta_1}\mathbf{x}\left[d\ln\left(\frac{1}{\eta_1}\mathbf{xe}\right)\right]. \quad (37)$$

The differential of $d\ln\left(\frac{1}{\eta_1}\mathbf{xe}\right)$ is

$$d\ln\left(\frac{1}{\eta_1}\mathbf{xe}\right) = -\frac{1}{\eta_1}d\eta_1\mathbf{e}. \quad (38)$$

Now we substitute equations 32, 16, and 38 into equation 37

$$dT = \frac{1}{\eta_1}\left[\left(\ln\left(\frac{1}{\eta_1}\mathbf{xe}\right)\right)^{\top}\mathbf{x}\otimes\mathbf{e}^{\top}\right]\operatorname{diag}(\operatorname{vec}\mathbf{I})(\mathbf{e}\otimes\mathbf{I})d\mathbf{f}$$
$$-\frac{1}{\eta_1^2}\mathbf{e}^{\top}\operatorname{diag}(\mathbf{f})\mathbf{x}\ln\left(\frac{1}{\eta_1}\mathbf{xe}\right)d\eta_1$$
$$-\frac{1}{\eta_1^2}\mathbf{e}^{\top}\operatorname{diag}(\mathbf{f})\mathbf{xe}\,d\eta_1. \qquad (39)$$

Finally we simplify the middle term of equation 39, and apply equation 8 to get our expression for the sensitivity of T,

$$\frac{dT}{d\theta^{\top}} = \frac{1}{\eta_1}\left[\left(\ln\left(\frac{1}{\eta_1}\mathbf{xe}\right)\right)^{\top}\mathbf{x}\otimes\mathbf{e}^{\top}\right]\operatorname{diag}(\operatorname{vec}\mathbf{I})(\mathbf{e}\otimes\mathbf{I})\frac{d\mathbf{f}}{d\theta^{\top}}$$
$$-\frac{1}{\eta_1}T\frac{d\eta_1}{d\theta^{\top}}$$
$$-\frac{1}{\eta_1^2}\mathbf{e}^{\top}\operatorname{diag}(\mathbf{f})\mathbf{xe}\frac{d\eta_1}{d\theta^{\top}}. \qquad (40)$$

2.5 The interquartile range

Let $f(\mathbf{x})$ be a probability density function, expressed as a horizontal vector.

$$f(\mathbf{x}) = \begin{pmatrix} f(x_1) \\ f(x_2) \\ \vdots \\ f(x_h) \end{pmatrix} \qquad (41)$$

Then $F(\mathbf{x}) = \int_{-\infty}^{x} f(s)\,ds$ is the cumulative distribution. It is also a horizontal vector

$$F(\mathbf{x}) = \begin{pmatrix} \int_0^{x_1} f(s)\,ds \\ \vdots \\ \int_0^{x_h} f(s)\,ds \end{pmatrix} \qquad (42)$$

The q th quantile is the value $\hat{x}$ satisfying

$$F(\hat{x}) = q. \tag{43}$$

Let $F(\hat{x}_1) = q_1$ and $F(\hat{x}_2) = q_2$, assuming that $q_2 > q_1$. The inter-quantile range is

$$R(q_1, q_2) = x_2 - x_1. \tag{44}$$

The special case of the interquartile range would refer to $R(0.25, 0.75)$.

We can choose a set of quantiles of interest

$$\mathbf{q} = \begin{pmatrix} q_1 \\ \vdots \\ q_h \end{pmatrix} \tag{45}$$

Suppose that the distribution $f(\cdot)$ depends on a parameter vector θ, dimension $p \times 1$. Then

$$F\left[\theta, \hat{\mathbf{x}}(\theta)\right] = \mathbf{q}, \tag{46}$$

where $\hat{\mathbf{x}}(\theta)$ defines the vector of quantiles.

Next we differentiate equation 46.

$$\frac{\partial F}{\partial \theta^\top} d\theta + \frac{\partial F}{\partial \hat{x}^\top} d\hat{x} = 0. \tag{47}$$

Solving for $d\hat{x}$

$$d\hat{x} = -\left(\frac{\partial F}{\partial \hat{x}^\top}\right)^{-1} \left(\frac{\partial F}{\partial \theta^\top}\right) d\theta. \tag{48}$$

Then rearranging according to equation 8

$$\frac{d\hat{x}}{d\theta^\top} = -\left(\frac{\partial F}{\partial \hat{x}^\top}\right)^{-1} \left(\frac{\partial F}{\partial \theta^\top}\right). \tag{49}$$

The first term of equation 49 represents

$$\left(\frac{\partial F}{\partial \hat{x}^{\top}}\right)^{-1} = \begin{pmatrix} \frac{1}{f(\hat{x}_1)} & & 0 \\ & \ddots & \\ 0 & & \frac{1}{f(\hat{x}_h)} \end{pmatrix} \tag{50}$$

While the second term of equation 49 can be written as

$$\left(\frac{\partial F}{\partial \theta^{\top}}\right) = \begin{pmatrix} \frac{\partial F(\hat{x}_1)}{\partial \theta_1} & \cdots & \frac{\partial F(\hat{x}_1)}{\partial \theta_p} \\ \vdots & & \vdots \\ \frac{\partial F(\hat{x}_h)}{\partial \theta_1} & \cdots & \frac{\partial F(\hat{x}_h)}{\partial \theta_p} \end{pmatrix} \tag{51}$$

Combining equations 50 and 51 according to equation 49 gives

$$\left(\frac{d\hat{x}}{d\theta^{\top}}\right) = -\begin{pmatrix} \frac{1}{f(\hat{x}_1)}\frac{\partial F(\hat{x}_1)}{\partial \theta_1} & \cdots & \frac{1}{f(\hat{x}_1)}\frac{\partial F(\hat{x}_1)}{\partial \theta_p} \\ \vdots & & \vdots \\ \frac{1}{f(\hat{x}_h)}\frac{\partial F(\hat{x}_h)}{\partial \theta_1} & \cdots & \frac{1}{f(\hat{x}_h)}\frac{\partial F(\hat{x}_h)}{\partial \theta_p} \end{pmatrix} \tag{52}$$

The sensitivity of the inter-quantile range is the difference between row j and row i of 52

$$\frac{dR_{(i,j)}}{d\theta^{\top}} = \frac{d\hat{x}_j}{d\theta^{\top}} - \frac{d\hat{x}_i}{d\theta^{\top}}. \tag{53}$$

When $\mathbf{f}(x)$ is a discrete distribution, the quantiles have to be interpolated. This is what we did to find the sensitivity of the *IQR* with quartiles $\hat{x}_3$ and $\hat{x}_1$.

CHAPTER 3

Life expectancy and disparity

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CHAPTER 3

ABSTRACT

Background: For two centuries life expectancy has increased steadily in prosperous countries. Reductions in mortality have been more rapid at younger vs. older ages, compressing the distribution of lifespans.

Methods: Life disparity is a measure of how much lifespans differ among individuals. We define a death as premature if postponing it to a later age would decrease lifespan disparity. New demographic data and methods permit exact determination of which deaths are premature. For the lifetables from 1840 to 2008 in the Human Mortality Database, about 38% of deaths are premature. Using these lifetables we determined the contribution of progress in postponing premature deaths to older ages to the increase in life expectancy and the decline in life disparity.

Results: In 89 of the 170 years from 1840 to 2009, the country with the highest male life expectancy had the lowest male life disparity. This was true in 86 years for female life expectancy and disparity. In all years, the top several life expectancy leaders were also the top life disparity leaders. Fully 84% of the increase in life expectancy resulted from averting premature deaths. The reduction in life disparity resulted from reductions in early-life disparity, i.e., disparity caused by premature deaths; late-life disparity levels remained roughly constant.

Conclusions: The countries that have been the most successful in averting premature deaths have consistently been the life expectancy leaders. Greater longevity and greater equality of individuals' lifespans are not incompatible goals. Countries can achieve both by reducing premature deaths.

Introduction

The rise in life expectancy, from under 40 years in all areas of the world two centuries ago to over 80 years today in many developed countries, has fundamentally improved the human condition.[1,2] Equally significant and closely linked to the increase in life expectancy has been the reduction of differences among individuals in the age at death.[3-6] Even in the most egalitarian societies before the mid-19th century the fate of most newborns was to die young but a fortunate minority survived to old age. Death rates today in health leaders such as Japan, Spain and Sweden imply that three-quarters of babies will survive to celebrate their 75th birthdays.[2]

The negative correlation between high life expectancy and low lifespan variation has been investigated for several countries, including the United States,[4,6,7] England and Wales[7], Sweden[6] and Japan[6]. The correlation is strong but there are discrepancies. Some countries, notably the United States, have substantially greater lifespan dispersion than might be predicted from their high levels of life expectancy.[3-5]

Progress in reducing premature deaths reduces variation in lifespans, whereas progress in reducing deaths at older ages increases variation in lifespans. A recently-developed demographic formula permits ready determination of the ages at which deaths are premature.[8] We use this new formula and apply it to a large dataset on developed countries to gain a deeper understanding of the relationship between high life expectancy and low lifespan variation. We find that the countries that have been the most successful in reducing premature deaths, and consequently in reducing lifespan variation, have consistently been the life expectancy leaders.

Methods

Our calculations are based on all period lifetables of the Human Mortality Database (*HMD*), from 1840 to the most recent year available in the data set.[2] This is a freely available database with reliable, comparable data covering 40 countries and areas. (Table 1 in the appendix to Chapter 3 lists the countries or regions and years used in the analysis.)

We measure dispersion in age-at-death by the life disparity measure, $e^{\dagger}$ (technical description in the appendix).[8,9] Life disparity is defined as the average remaining life expectancy at the ages when death strikes; it is a measure of life years lost due to death. The more egalitarian the lifespan distribution is, the lower the life

disparity. In the Swedish female life table for 2008 life expectancy reached 83 years; for those women who survived to age 83, remaining life expectancy was 7.5 additional years. Hence a death shortly after birth would contribute 83 years whereas a death at age 83 would contribute 7.5 years. The average of such values over the Swedish female population, weighted by the number of deaths at each age, gives a life disparity of 9. In 1840 life expectancy for Swedish women was only 46 and life disparity was 24. Over time, as deaths became concentrated at later ages, the average gap was reduced between the age at which a person died and the remaining lifespans of people who survived beyond this age.

Saving lives (i.e., averting deaths) at any age increases life expectancy. Lifespan disparity, on the other hand, narrows or widens depending on the balance between saving lives at 'early' ages, which compresses the distribution of lifespans, and saving lives at 'late' ages, which expands this distribution. Separating the two is a unique threshold age, $a^{\dagger}$. Henceforth, we refer to deaths occurring before the threshold age as 'premature deaths', while those occurring after this age are 'late deaths'. This definition implies that deaths at surprisingly old ages can be premature deaths. In 2008 deaths up to age 82 were premature deaths for Swedish females (Table 1).

The life disparity measure has the property that it can be additively decomposed at any age such that the components before and after this age sum to the total life disparity.[8] When it is decomposed at the threshold age, the components are defined as 'early-life disparity' and 'late-life disparity'.

While it is known that high life expectancy is associated with low lifespan variation, we wanted to establish whether life expectancy leaders had the most egalitarian lifespan distributions. For each sex, year, and for up to 40 countries depending on the year, we determined the male and female record high life expectancy and record low life disparity. We calculated how many fewer years of life expectancy and additional years of life disparity each country experienced compared with the record-holding country in that year.

We next investigated the relative importance of premature vs. late deaths in determining the relationship between high life expectancy and low life disparity. To do so, we calculated first the number of premature and late deaths as a proportion of all deaths, measured by 10-year averages across all countries and years. We then compared this to the respective contributions of averting premature and late deaths to increases in life expectancy, using a 20-year moving average to smooth mortality trends over exceptional years of war, pandemics or famine.

Finally, we ranked countries according to their life expectancy and life disparity for the latest year for which we had data.

Results

Populations with high life expectancy enjoy low life disparity. In 89 out of 170 years, holders of record life expectancy for males also enjoyed the lowest life disparity (Figure 1). For females this happened 86 times (Figure S1 in the appendix). These countries increased life expectancy not because of a general decrease in life disparity at all ages, but because of a decrease in early-life disparity. Figure 2 shows that the reduction in life disparity—from around 25 years in 1840 to between 9 and 12 years at present—is overwhelmingly due to reductions in early-life disparity. Although mortality at old ages has come down considerably (which might cause one to expect increases in late-life disparity), the shifting of the threshold age to higher ages has caused late-life disparity to stay roughly constant at around or just under five years.

For females since 1840, premature deaths have accounted for only 38 percent of all deaths, but fully 84 percent of the increase in life expectancy resulted from decreases in premature deaths (Figure S2 in the appendix). During this time the threshold age rose considerably, rising from 47 for Swedish women in 1840 to 85 for Japanese women in 2009. Historically (and today in less developed countries) infants, children and younger adults suffered most premature deaths. In today's more developed countries, premature deaths have shifted primarily to older adults in their sixties and seventies. The rise in the threshold age is highly correlated with the rise in life expectancy.

Table 1 displays the latest period life expectancy, threshold age and life disparity calculated for each country. In Russia life expectancy is extraordinarily low and life disparity is very high. In the United States, life expectancy is much longer than in Russia but short compared with countries of similar income per capita. Life disparity in the U.S. is worse than in many Eastern European countries for both males and females. In contrast Japanese females are remarkably successful. They hold the record for life expectancy, 86.4 years in the lifetable for 2009. Half of deaths occurred after age 88 and the most common age of death was 93: deaths up to age 85 were premature in the sense that averting such deaths would decrease life disparity.

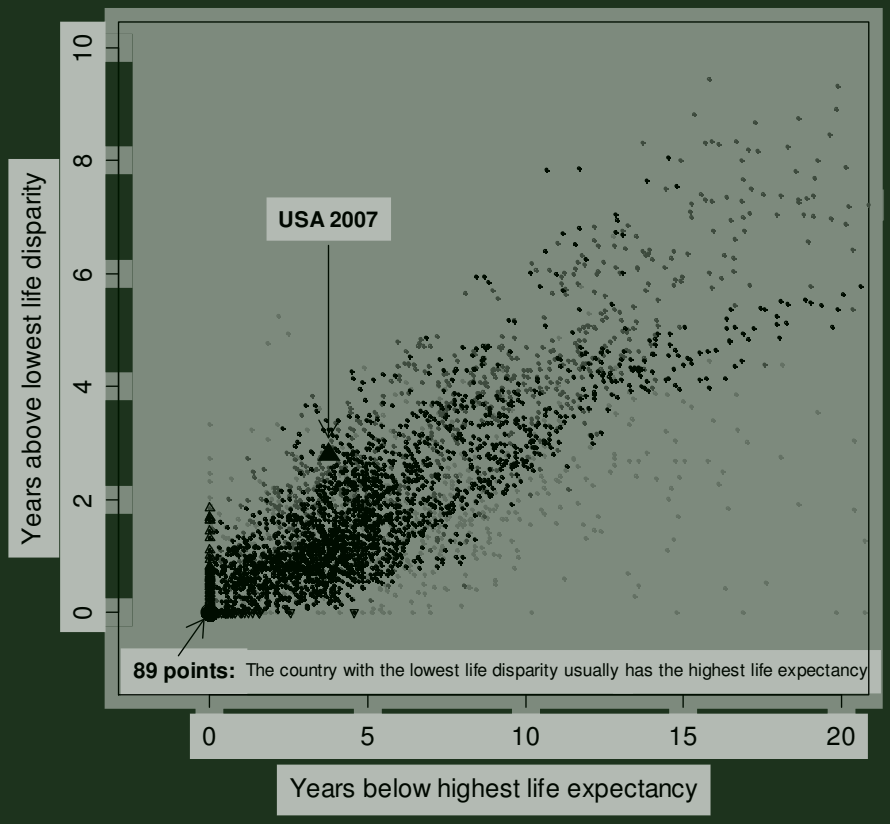

Figure 1: The association between life disparity in a specific year and life expectancy in that year for males in 40 countries and regions, 1840-2009 (Table 1 in the Chapter 3 appendix). The correlation coefficient between them is 0.77 (95% confidence interval 0.76 to 0.78). The large black triangle represents the United States in 2007: the U.S. had a male life expectancy 3.78 years lower than the international record in 2007 and a life disparity 2.8 years greater. The black points denote years after 1950, the dark grey points 1900-1949 and the light grey points 1840-1900. The inverted triangles represent countries with the lowest life disparity but with a life expectancy below the international record in the specific year; the small triangles indicate the life expectancy leaders in a given year, with life disparities greater than the most egalitarian country in that year. The black point at (0,0) marks countries with the lowest life disparity and the highest life expectancy. During the 170 years from 1840 to 2009, 89 holders of record life expectancy also enjoyed the lowest life disparity. The equivalent figure for females is presented as Figure S2 in the appendix.

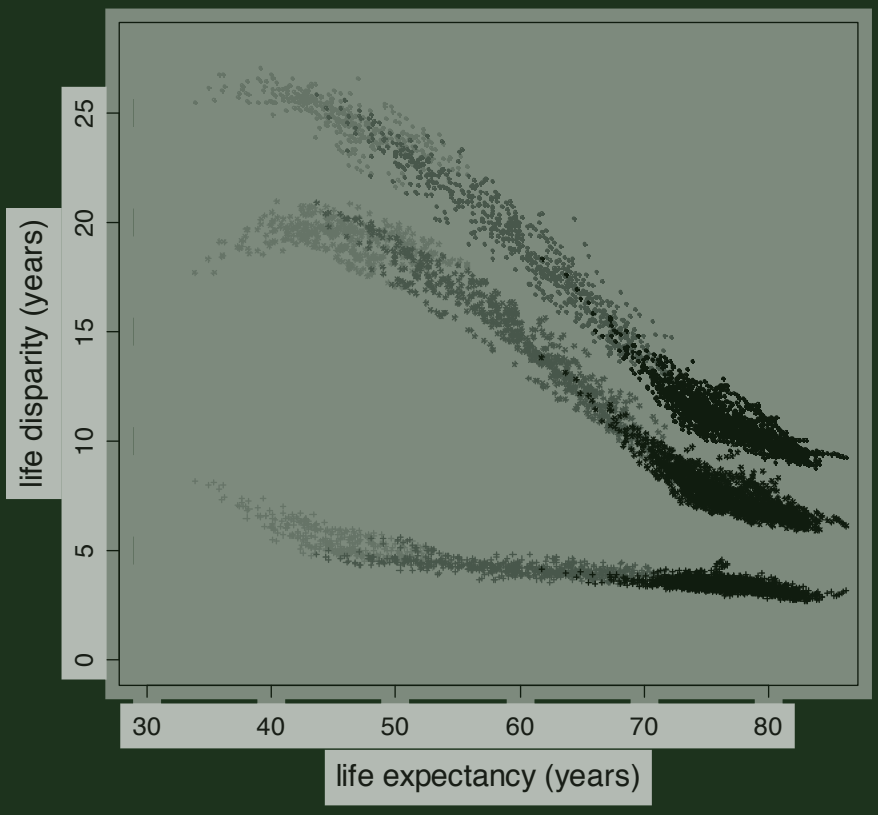

Figure 2: The relationship between total life disparity (top curve - points), early life disparity up to the threshold age (middle curve - stars) and late life disparity after the threshold age (bottom curve - crosses). The darkest hues relate to data from 1950-2007, middle hues 1900-1949 and lightest hues 1840-1899. Total disparity is an additive function of early life disparity and late life disparity. Since 1840 the decrease in total life disparity has resulted from reductions in early life disparity. Late life disparity has remained remarkably constant at about 5 years across a wide range of life expectancies. Hence, according to this measure, there has been neither a marked compression nor expansion of mortality at advanced ages as life expectancy has increased. Data are for females from the 37 countries and regions of the Human Mortality Database (Table 1 in the Supplementary Material).

Country or region	Females			Males		
	e_0	$e^\dagger$	$a^\dagger$	e_0	$e^\dagger$	$a^\dagger$
Japan	86.4	9.2	85.3	79.6	10.6	78.0
	(86.3, 86.4)	(9.1 ,9.2)	(85.3, 85.4)	(79.6, 79.6)	(10.6, 10.6)	(77.9, 78.0)
France	84.4	9.3	83.8	77.4	11.4	76.5
	(84.3, 84.4)	(9.3, 9.4)	(83.7, 83.8)	(77.4, 77.5)	(11.3, 11.4)	(76.5, 76.6)
Switzerland	84.1	9.0	83.2	79.3	10.2	78.0
	(83.9, 84.2)	(8.9, 9.1)	(83.1, 83.4)	(79.2, 79.5)	(10.1, 10.3)	(77.9, 78.3)
Italy	84.1	8.8	82.9	78.8	10.2	77.3
	(84.0, 84.1)	(8.8, 8.9)	(82.8, 82.9)	(78.8, 78.9)	(10.1, 10.2)	(77.2, 77.3)
Spain	84.1	8.8	82.9	77.6	11.1	76.0
	(84.0, 84.1)	(8.7, 8.8)	(82.9, 83.0)	(77.5, 77.6)	(11.0, 11.1)	(76.0, 76.1)
Australia	83.7	9.3	82.9	79.3	10.6	78.0
	(83.7, 83.8)	(9.2, 9.3)	(82.8, 83.0)	(79.2, 79.4)	(10.5, 10.6)	(77.9, 78.1)
Finland	83.1	9.1	82.4	76.5	11.2	75.3
	(83.0, 83.3)	(9.0, 9.2)	(82.2, 82.6)	(76.3, 76.7)	(11.1, 11.3)	(75.1, 75.5)
Sweden	83.1	8.9	82.2	79.1	9.8	77.9
	(83.0, 83.2)	(8.8, 8.9)	(82.1, 82.3)	(79.0, 79.2)	(9.7, 9.8)	(77.7, 78.0)
Austria	83.0	8.9	82.1	77.6	10.6	76.6
	(82.9, 83.0)	(8.8, 8.9)	(82.0, 82.2)	(77.5, 77.7)	(10.5, 10.7)	(76.4, 76.8)
Norway	83.0	9.1	81.9	78.3	10.0	77.2
	(82.8 ,83.1)	(9.0, 9.2)	(81.7, 82.0)	(78.2, 78.5)	(9.9, 10.1)	(77.0, 77.4)
Iceland	83.0	8.7	81.9	79.7	9.8	78.2
	(82.5, 83.7)	(8.3, 9.1)	(81.4, 82.6)	(79.0, 80.4)	(9.3, 10.3)	(77.1, 79.2)
Canada	82.9	10.0	81.9	78.3	11.0	76.9
	(82.9 ,83.0)	(9.9, 10.0)	(81.8, 82.0)	(78.3, 78.4)	(10.9, 11.0)	(76.8, 77.0)
Israel	82.9	9.2	81.1	79.0	10.9	77.0
	(82.7, 83.0)	(9.1, 9.3)	(81.0, 81.3)	(78.8, 79.2)	(10.7, 11.0)	(76.8, 77.2)
England & Wales	82.5	9.8	81.1	78.3	10.9	76.6
	(82.4, 82.5)	(9.8, 9.8)	(81.1, 81.2)	(78.3, 78.4)	(10.9, 10.9)	(76.6, 76.7)
West Germany	82.4	8.9	81.7	77.5	10.5	76.1
	(82.4, 82.5)	(8.9, 9.0)	(81.7, 81.8)	(77.5, 77.6)	(10.4, 10.5)	(76.0, 76.1)
East Germany	82.4	9.1	81.2	76.5	11.0	74.8
	(82.2, 82.5)	(9.0, 9.1)	(81.1, 81.2)	(76.4, 76.6)	(10.9, 11.1)	(74.7, 74.9)
Portugal	82.4	8.9	81.5	76.4	11.0	75.5
	(82.4 ,82.5)	(8.8, 8.9)	(81.4, 81.6)	(76.3, 76.5)	(10.9, 11.0)	(75.3, 75.6)
Belgium	82.3	9.5	81.6	76.9	10.9	75.7
	(82.2, 82.4)	(9.4, 9.5)	(81.5, 81.7)	(76.8, 77.0)	(10.8, 10.9)	(75.5, 75.8)
Netherlands	82.3	9.6	80.9	78.3	9.8	76.7
	(82.3, 82.4)	(9.5, 9.7)	(80.8, 81.0)	(78.2, 78.4)	(9.8,9.9)	(76.6, 76.8)
Slovenia	82.2	8.9	81.0	75.7	11.0	73.9
	(82.0, 82.5)	(8.8, 9.1)	(80.7, 81.2)	(75.5, 76.0)	(10.8, 11.2)	(73.5, 74.2)
Luxembourg	82.1	9.2	81.4	76.6	10.0	76.0
	(81.6, 82.6)	(8.9, 9.6)	(80.8, 82.0)	(76.1, 77.2)	(9.7, 10.4)	(75.2, 76.7)
New Zealand (NM)	82.1	9.6	81.2	77.8	10.4	76.6
	(81.9, 82.3)	(9.4, 9.7)	(81.0, 81.4)	(77.6, 78.0)	(10.3, 10.6)	(76.3, 76.8)
Taiwan	82.0	10.1	80.5	75.9	12.6	73.7
	(81.9,82.1)	(10.0, 10.2)	(80.4, 80.6)	(75.8, 76.0)	(12.5, 12.7)	(73.6, 73.9)
Ireland	81.9	9.4	80.3	77.3	10.2	75.5
	(81.7, 82.1)	(9.3, 9.6)	(80.1, 80.6)	(77.1, 77.4)	(10.1, 10.4)	(75.3, 75.8)
Northern Ireland	81.3	9.9	80.6	77.2	11.0	76.1
	(81.0,81.6)	(9.7, 10.1)	(80.3, 80.9)	(76.9, 77.5)	(10.8, 11.3)	(75.7, 76.4)
Denmark	80.9	9.9	79.4	76.5	10.7	74.9
	(80.8, 81.0)	(9.8, 10.0)	(79.2, 79.6)	(76.3, 76.6)	(10.6, 10.8)	(74.7, 75.1)

Country or Region	Females			Males		
	e_0	$e^\dagger$	$a^\dagger$	e_0	$e^\dagger$	$a^\dagger$
USA	80.8 (80.7, 80.8)	11.1 (11.0, 11.1)	79.8 (79.8, 79.8)	75.6 (75.6, 75.6)	12.5 (12.5, 12.5)	74.5 (74.4, 74.5)
Chile	80.7 (80.6, 80.8)	10.7 (10.6, 10.8)	78.9 (78.8, 79.0)	75.0 (74.9, 75.1)	12.7 (12.5, 12.8)	72.3 (72.0, 72.5)
Scotland	80.4 (80.3, 80.6)	10.3 (10.2, 10.4)	78.9 (78.7, 79.1)	75.9 (75.7, 76.0)	11.6 (11.5, 11.7)	74.0 (73.8, 74.2)
Czech Republic	80.3 (80.2, 80.4)	9.3 (9.2, 9.4)	78.9 (78.8, 79.0)	74.0 (73.9, 74.1)	11.2 (11.2, 11.3)	71.7 (71.6, 71.9)
Estonia	80.0 (79.7,80.3)	9.9 (9.6, 10.1)	79.0 (78.8, 79.3)	69.7 (69.4, 70.1)	12.9 (12.7, 13.2)	66.3 (65.8, 66.9)
Poland	79.9 (79.8, 80.0)	10.0 (9.9, 10.0)	78.9 (78.8, 79.0)	71.5 (71.4, 71.5)	12.5 (12.5, 12.6)	68.7 (68.6, 68.8)
Slovakia	78.8 (78.7, 79.0)	9.8 (9.6, 9.9)	77.2 (77.0, 77.3)	70.8 (70.6, 71.0)	12.2 (12.1, 12.3)	67.8 (67.6, 68.0)
Lithuania	78.6 (78.4,78.7)	10.2 (10.1, 10.4)	78.0 (77.8, 78.2)	67.5 (67.3, 67.7)	13.6 (13.4, 13.7)	64.0 (63.6,64.3)
*China**	*78.2*	*11.7*	*76.5*	*73.4*	*12.6*	*72.0*
Latvia	78.0 (77.8,78.3)	10.5 (10.4, 10.7)	77.5 (77.2, 77.7)	68.3 (68.0, 68.5)	13.2 (13.0, 13.3)	64.8 (64.5, 65.1)
Hungary	77.7 (77.5, 77.8)	10.7 (10.7, 10.8)	76.1 (76.0, 76.2)	69.2 (69.0, 69.3)	12.9 (12.8, 13.0)	65.0 (64.8, 65.1)
Bulgaria	77.3 (77.1,77.4)	10.1 (10.0, 10.2)	76.3 (76.1, 76.4)	70.0 (69.9, 70.2)	12.6 (12.5, 12.7)	67.3 (67.2, 67.5)
Belarus	76.1 (76.0, 76.3)	10.9 (10.8, 11.0)	74.7 (74.6, 74.9)	64.5 (64.4, 64.7)	13.7 (13.6, 13.7)	60.4 (60.2, 60.6)
Russia	74.2 (74.1, 74.2)	11.9 (11.9, 11.9)	73.4 (73.4, 73.5)	61.8 (61.7, 61.8)	15.0 (15.0, 15.1)	57.4 (57.3, 57.4)
Ukraine	73.8 (73.7, 73.9)	11.6 (11.6, 11.7)	72.9 (72.8, 72.9)	62.3 (62.2, 62.4)	14.7 (14.7, 14.8)	58.0 (57.9, 58.0)
*India**	*63.8*	*18.2*	*72.8*	*61.8*	*18.2*	*69.7*
*South Africa**	*52.6*	*20.7*	*60.6*	*50.0*	*19.8*	*56.8*

Data sources: * Data for 2006 from World Health Organization (WHO), not used in the analysis but shown here for comparative purposes. All other data are from the Human Mortality Database 2011; see Table 1 in the Supplementary Material for latest year available.

Table 1: Countries and regions of the Human Mortality Database[2] used in our analysis, ranked by female life expectancy for the latest year available. Life expectancy is denoted by e_0, the threshold age separating 'premature' from 'late' deaths by $a^\dagger$, and life disparity by $e^\dagger$, with 95% confidence intervals given in brackets (see appendix). The countries used in our analyses are in regular type face. The countries in italics are shown for comparison; data are less reliable for these countries.

CHAPTER 3

Discussion

These findings make clear that the correlation between high life expectancy and low lifespan variation is due to progress in reducing early-life disparity. The countries that have the highest life expectancy today are those who have been most successful at postponing the premature deaths that contribute to early-life disparity.

In addition to life disparity, several other measures of lifespan dispersion have been proposed.[3 4 6 10] We analyzed the extent to which our findings depend on our use of life disparity as our measure of lifespan variation. We calculated Pearson correlation coefficients between pairs of the more commonly used measures of lifespan variation, based on all male and female period life tables available from the Human Mortality Database (Table S1 in the appendix). As shown in Table 2 of the Supplementary Material, these measures are highly correlated with each other. In particular, the correlation of life disparity with the other measures never falls below 0.966 for females and 0.940 for males. Hence life disparity can be viewed as a surrogate for the other measures. Although the various measures are highly correlated, they differ somewhat in their sensitivity to deaths at different ages in the lifespan distribution.[4 11] The use of an alternate measure of lifespan variation would result in some changes in the ranking of countries with similar life disparity levels, but the high correlation between measures implies that such changes would be minor.

Some researchers have examined whether lifespan variation above the modal age at death has changed with increased survivorship. These studies also tend to find a gradual decline in later life mortality variation.[10 12-14] More generally, whether expansion or compression of the lifespan distribution is observed over time can depend on the age range examined.[15-17] While being a life expectancy leader is associated with low life disparity when the entire lifetime is examined, this relationship might not hold for selected age ranges.

Reducing early life disparities helps people plan their less-uncertain lifetimes. A higher likelihood of surviving to old age makes savings more worthwhile, raises the value of individual and public investments in education and training, and increases the prevalence of long-term relationships. Hence, healthy longevity is a prime driver of a country's wealth and well-being.[18] While some degree of income inequality might create incentives to work harder, premature deaths bring little benefit and impose major costs.[19]

Moreover, equity in the capability to maintain good health is central to any larger concept of societal justice.[20] The tenet that everyone should be entitled to a long, healthy lifespan has gained support as mortality at younger ages has declined. Currently, rates of change for adult mortality vary more across countries than those

for infants and children.[21] In Williams' concept of *fair innings*,[22] individuals dying early are "cheated" while those living beyond a "normal" lifespan are "living on borrowed time". Groups and areas with lower socioeconomic status account for a disproportionate share of lifespan variation:[3 4 7] this compounds the inequity of premature death.

If death rates continue to decline, most babies born in advanced nations today may live to enjoy their 100th birthday.[23] As we celebrate this progress in extending lives it is reasonable to question whether we ought to continue aiming for ever longer lives *on average* or to ensure that more individuals avoid premature death. Policymakers face a choice of where to target health care spending. Reducing life disparity would lead to health policies that prioritize early mortality and to social protection schemes designed to shield vulnerable individuals and groups. We are not the first to make this argument. Heath poignantly reasoned that if health care services were serious about reducing health inequality they should direct their attentions to reducing premature mortality—even if this meant reducing expensive medical treatments for the elderly.[24] The accompanying editorial in this journal proclaimed that "premature deaths should be the priority for prevention".[25]

Russia, the U.S. and other laggards can learn much from research on the reasons why various countries (including Japan, France, Italy, Spain, Sweden and Switzerland) have been more successful in reducing premature deaths. The reasons involve health care, social policies, personal behavior (especially cigarette smoking and alcohol abuse), and the safety and salubriousness of the environment.[26-33] Genetic variation plays a modest role in determining variation in how long we live[34 35] and cannot account for the major declines in life disparity and increases in life expectancy or the large differences in life expectancy and disparity among countries.

Smits and Monden[5] recently showed that countries achieving some level of life expectancy earlier than others did so with higher levels of lifespan variation. This led them to conclude that "reducing inequality and gaining increases in life expectancy might be alternative goals that require different policy measures to be achieved". Our results differ because we examine differences between countries in lifespan variation for each year whereas they examine differences over time in lifespan variation within each country. These different set-ups can lead to different conclusions. In a study comparing the United States to England and Wales, reductions in circulatory diseases were causing most of the changes in lifespan variation over time (in each country) whereas differences in external mortality were causing differences in life disparity between countries at any given time.[7] As can be seen in Figure 3 the relationship between being pioneers in life expectancy and having high life disparity is weak, especially after 1960. We take issue with Smits

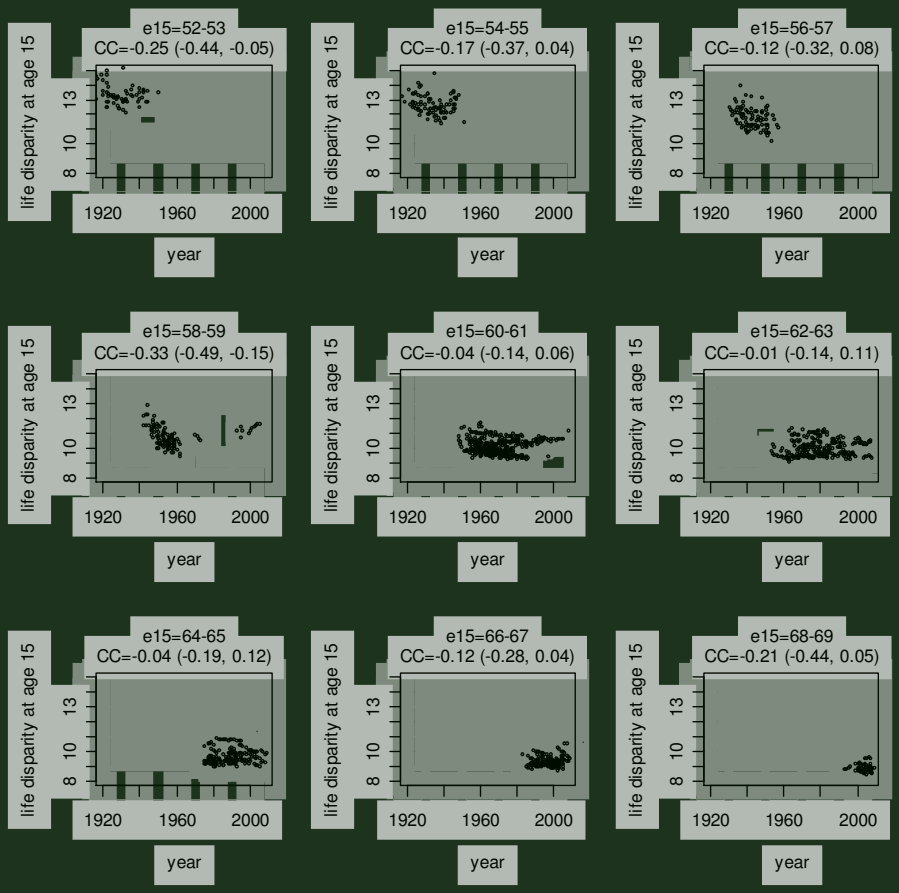

Figure 3: The relationship between remaining life expectancy at age 15 (e_{15}) and life disparity at age 15, according to the year in which e_{15} was first reached. Up until 1960 and for e_{15} from 54 to 59, the pioneers in first attaining a level of remaining life expectancy did so with higher levels of life disparity than the laggards. Since 1960 and at higher remaining life expectancies, the relationship between remaining life expectancy and life disparity at age 15 are not correlated. Ages 15 and over were examined to make the results comparable to those obtained by Smits and Monden.[5] Data are for females from the 40 countries and regions in the Human Mortality Database (Table 1 in the appendix).

and Monden's conclusion which our cross-sectional results do not support. Over the past 170 years, the country with the lowest life disparity most often had the highest life expectancy. Even today, the most egalitarian countries are all among the longest living.

The increase in life expectancy is given by the product of two factors—life disparity and the rate of progress in reducing age-specific death rates.[9] The lower life disparity is, the greater is the rate of progress needed to achieve an additional year of life expectancy. Consequently it might be thought that countries with long life expectancy would tend to have high life disparity. The opposite is true (Figure 1). The reason is that the countries with long life expectancy have gained this victory by focusing on reductions in premature deaths—and reductions in premature deaths reduce life disparity. It is not a question of either long life or low disparity: countries can achieve both by averting premature deaths.

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Appendix to Chapter 3

Life Disparity

Life disparity, $e^{\dagger}$, is the life expectancy lost due to death,

$$e^{\dagger} = \int_0^{\omega} e(x,t) f(x,t) dx$$

where

$$e(a,t) = \frac{\int_a^{\omega} l(x,t) dx}{l(a,t)}$$

is remaining life expectancy at age a and time t,

$$l(a,t) = \exp(-\int_0^a \mu(x,t) dx)$$

gives the probability of survival to age a and $\mu(a,t)$ denotes the age-specific hazard of death. The life table distribution of deaths is given by $f(a,t) = l(a,t)\mu(a,t)$. Maximum lifespan is denoted by ω.

Conceptually, this measure is similar to Greville's 1948 variant of the Potential Years of Life Lost (PYLL) measure,[S1] which weights the death counts from a given disease at each age by remaining life expectancy in order to assess the importance of major causes of death. In this way the age profile of disease mortality is taken into account, which can lead to different conclusions than assessments that compare diseases strictly on the basis of death counts or on their average effect on lifespan.

When all causes of death are taken into account, life disparity functions in much the same way. Saving lives at ages with both many remaining life years and a high number of death counts has the greatest impact on lifespan variation. This was first observed by Keyfitz,[S2,S3] who derived the formula for the elasticity of life expectancy to a proportional change in mortality (also known as the entropy of the life table, or Keyfitz' H), which he observed was related to variation in age-at-death. Life disparity equals the entropy of the life table multiplied by life expectancy.[S4-S6] It is only in recent years that the full potential of life disparity as a measure of lifespan variation has been realized.[S7,S8]

Methods to obtain confidence intervals

To be sure that random fluctuation was not substantially affecting our rankings of life expectancy, life disparity and the threshold age in Table 1, we estimated 95% confidence intervals around our results. This was done by Monte Carlo simulation, assuming a binomial distribution of death counts. For each age interval the number of observations in each simulation round was based on the observed number of deaths, D_x, divided by the probability of dying, $\hat{q}_x$. The simulated death counts, d_x^{sim}, divided by the observed population at risk, N_x, gave us simulated death probabilities q_x^{sim}. From these values we simulated 1000 life tables that we used to generate confidence intervals around our life-table-based estimates. Others have used similar methods to generate confidence intervals around life expectancy and healthy life expectancy for small populations.[S9-S12]

Country or region	Earliest year	Latest year
Australia	1921	2007
Austria	1947	2008
Belgium*	1841	2007
Bulgaria	1970	2009
Belarus	1970	2007
Canada	1921	2007
Switzerland	1876	2007
Chile	1992	2005
Czech	1950	2009
West Germany	1956	2008
East Germany	1956	2008
Denmark	1840	2008
Spain	1908	2006
Estonia	1959	2009
Finland	1878	2009
France	1840	2007
England & Wales	1841	2009
North Ireland	1922	2009
Scotland	1855	2009
Hungry	1950	2006
Ireland	1950	2006
Iceland	1840	2008
Israel	1983	2008
Italy	1872	2007
Japan	1947	2009
Latvia	1970	2009
Luxembourg	1960	2007
Lithuania	1959	2009
Netherlands	1850	2008
Norway	1846	2008
New Zealand non-Maori	1901	2008
Poland	1958	2009
Portugal	1940	2009
Russia	1959	2008
Slovakia	1950	2009
Slovenia	1983	2009
Sweden	1840	2008
Taiwan	1970	2009
Ukraine	1970	2006
USA	1933	2007

*No data was available for 1914-1918.

Table S1: Countries and regions of the Human Mortality Database used in our analysis. We used data from the earliest year given in the table through the latest year.

	$e^{\dagger}$	σ^2	σ	σ_{10}	$e^{\dagger}/e_0$	G	IQR	AID
Life disparity (e†)	1.000							
Variance (σ^2)	0.993	1.000						
Standard deviation (σ)	0.985	0.996	1.000					
Standard deviation past age 10 (σ_{10})	0.972	0.964	0.961	1.000				
Entropy of life table ($e^{\dagger}/e_0$)	0.966	0.936	0.919	0.916	1.000			
Gini coefficient (G)	0.983	0.961	0.946	0.937	0.997	1.000		
Inter-Quartile Range (IQR)	0.967	0.944	0.917	0.921	0.966	0.974	1.000	
Inter-individual difference (AID)	0.995	0.998	0.996	0.973	0.937	0.962	0.945	1.000

Table 2(a): Pearson correlation coefficients between pairs of measures of lifespan variation, based on all 3474 female period life tables available from the Human Mortality Database, 1840-2009.

	$e^{\dagger}$	σ^2	σ	σ_{10}	$e^{\dagger}/e_0$	G	IQR	AID
Life disparity (e†)	1.000							
Variance (σ^2)	0.986	1.000						
Standard deviation (σ)	0.979	0.996	1.000					
Standard deviation past age 10 (σ_{10})	0.940	0.909	0.908	1.000				
Entropy of life table ($e^{\dagger}/e_0$)	0.958	0.913	0.898	0.879	1.000			
Gini coefficient (G)	0.979	0.946	0.933	0.898	0.996	1.000		
Inter-Quartile Range (IQR)	0.965	0.937	0.913	0.890	0.948	0.964	1.000	
Inter-individual difference (AID)	0.992	0.997	0.995	0.930	0.917	0.950	0.941	1.000

Table 2(b): Pearson correlation coefficients between pairs of measures of lifespan variation, based on all 3474 male period life tables available from the Human Mortality Database, 1840-2009.

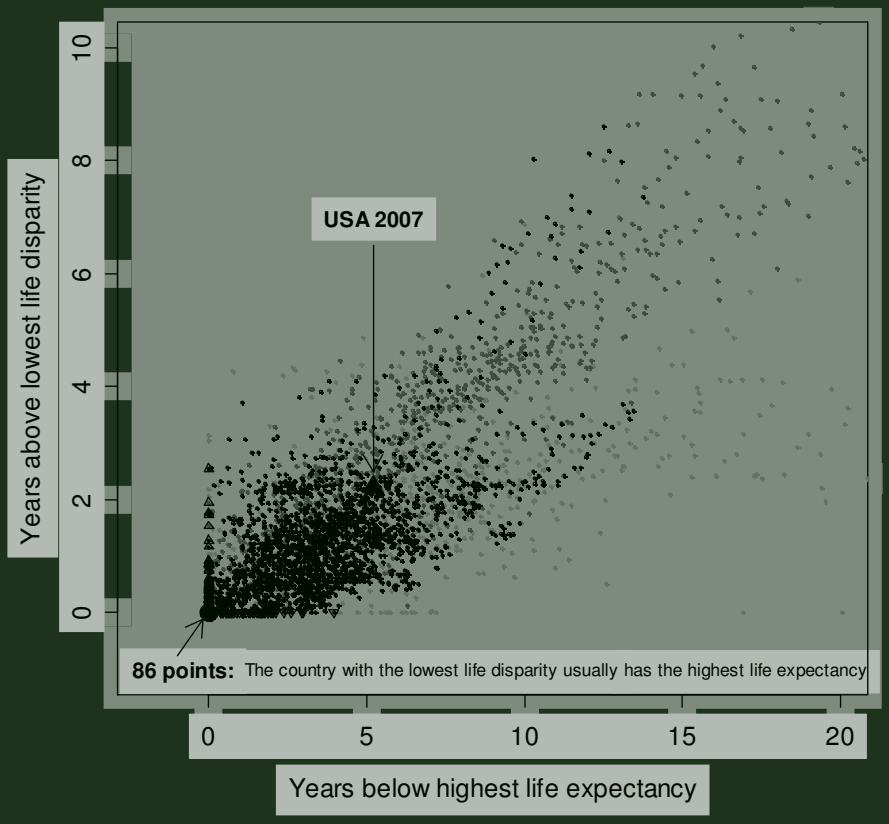

Figure S1: The association between life disparity in a specific year and life expectancy for females in that year for the 40 countries and regions in the Human Mortality Database, 1840-2009 (Table S1). The correlation coefficient between them is 0.75 (95% confidence interval 0.73 to 0.76). The black triangle represents the United States in 2007: the U.S. had a female life expectancy 5.2 years lower than the international record in 2006 and a life disparity 2.2 years greater. The black points denote years after 1950, the dark grey points 1900-1949 and the light grey points 1840-1900. The inverted triangles represent countries with the lowest life disparity but with a life expectancy below the international record in the specific year; the small black triangles indicate the life expectancy leaders in a given year, with life disparities greater than the most egalitarian country in that year. The black point at (0,0) marks countries with the lowest life disparity and the highest life expectancy. During the 170 years from 1840 to 2009, 86 holders of record life expectancy also enjoyed the lowest life disparity.

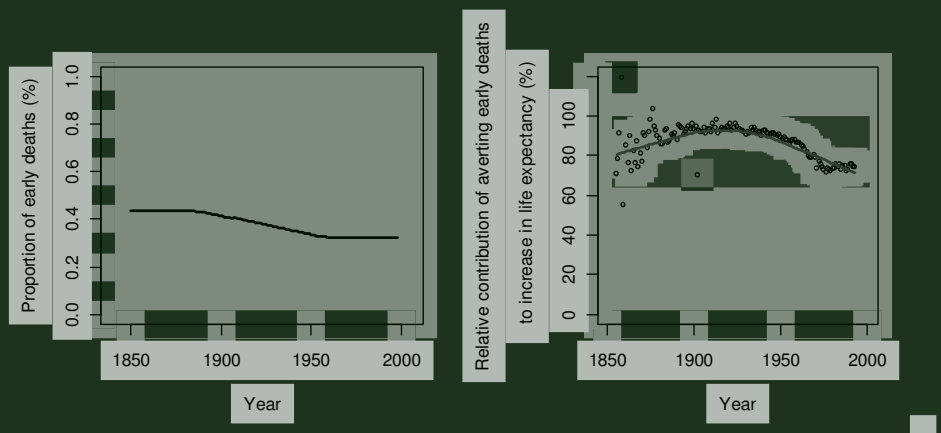

Figure S2: The left panel expresses early deaths as a proportion of all deaths, smoothed by 20-year averages. The right panel displays the 30-year moving average of the relative contribution of averting early deaths to the increase in life expectancy, with the grey solid line marking the trend. The data pertain to females, 1840-2009, all 40 countries and regions of the HMD.

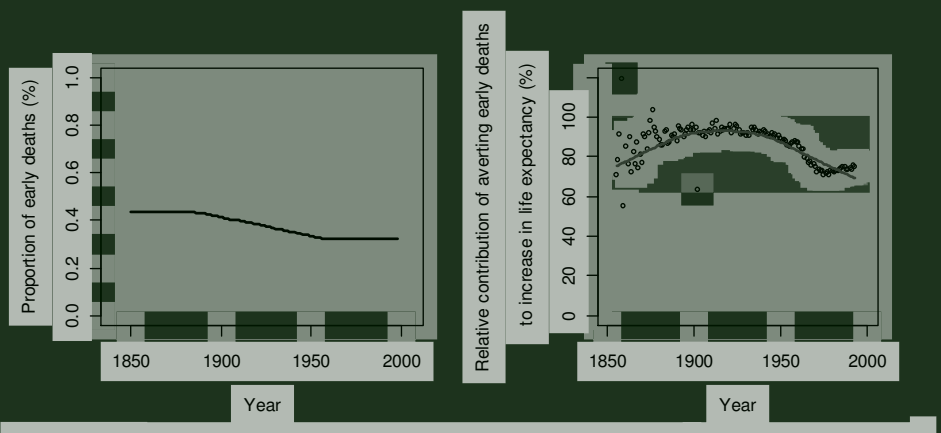

Figure S3: To show that the trends in Figure S2 above are not due to compositional change from new entrants into our dataset, we plotted the two relationships using only the eleven countries for which we had over 100 years of data (see Table S1).

Alternative calculations using the AID measure

Some concern might be raised about whether artefactual correlations are present in our findings since calculation of life disparity involves prior knowledge of life expectancy. We showed the high correlation between life disparity and other measures of lifespan variation in Table 2 of the supplementary material. Some of these other measures do not contain life expectancy in their formulation. To be sure that our results were robust to other measures, we ran our analysis with an alternative measure of lifespan variation, the absolute inter-individual difference (AID). While AID and life disparity are highly correlated, AID tends to be more sensitive to mortality change at younger ages than life disparity.[S7]

The AID is an alternative measure of lifespan variation that is related to the well-known Gini coefficient of inequality. There are many equivalent formulations to the AID, but the Kendall and Stuart definition is the most helpful for understanding the nature of the statistic, which essentially measures the average absolute distance in years between each pair of individuals' age at death (length of life) in the population.[S13] From the life table, it can be calculated as follows:

$$AID = \frac{1}{2}\int_0^\omega\int_0^\omega |x-y| f(x) f(y) dxdy \quad (1)$$

where $|x-y|$ is the absolute value of the distance in years between age x and age y, and $f(x)$ and $f(y)$ are the probabilities of death at ages x and y respectively.

Using the AID measure, the country with the highest life expectancy also had the lowest AID 74 times for females (Figure S3), and 67 times for males (Figure S4) out of 170 years. Differences in this relationship between the two measures were mostly owing to differences in historical populations, especially during war, famine and epidemic years, when certain countries had qualitatively different age at death distributions from other countries.

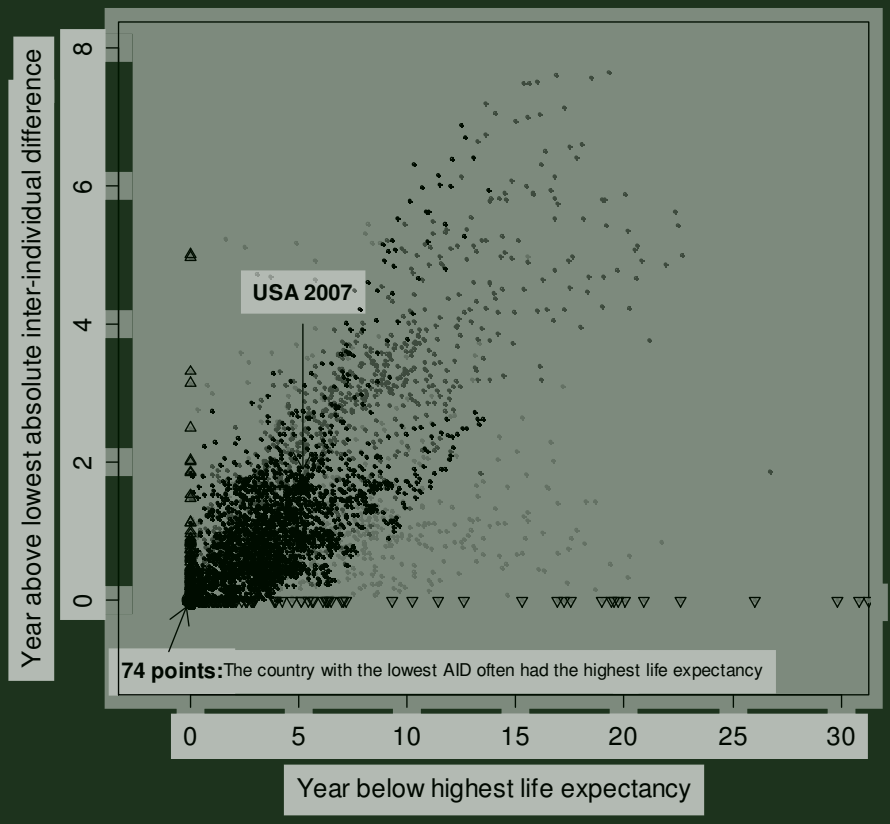

Figure S4: Alternative calculations using the AID measure, females. The correlation coefficient between them is 0.60 (0.58 to 0.62).

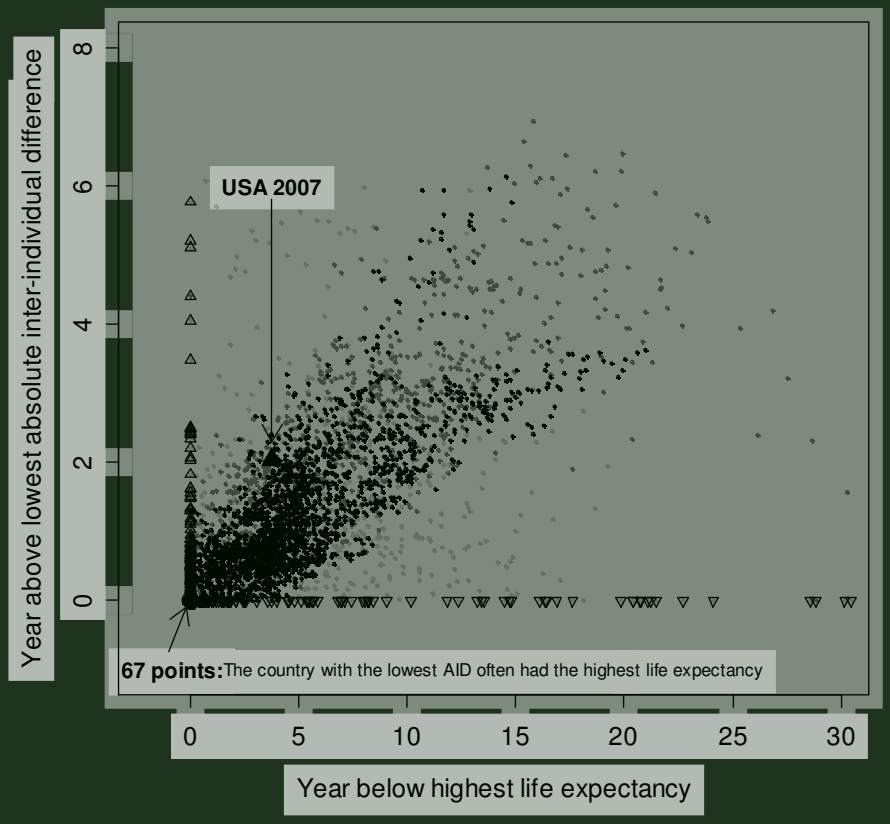

Figure S5: Alternative calculations using the AID measure, males. The correlation coefficient between them is 0.62 (0.60 to 0.64).

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CHAPTER 4

The Japanese female age pattern of mortality decline

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CHAPTER 4

ABSTRACT

Background: Previous research has argued that countries with high life expectancy also have low lifespan variation because of progress at postponing premature deaths. Japanese females have held the record for period life expectancy since 1986, but far less is known about their patterns in lifespan variation.

Methods: We examined trends in life expectancy and life disparity for Japanese females since the Second World War. We calculated the contribution of each age to changes in both measures for each decade since 1950. We compared the actual age profile of mortality change to what would have been expected under the most efficient profiles—that is, age profiles of mortality improvement that would have the maximal impact on increasing life expectancy and those which would reduce early life disparity the most. The 'efficiency gap', which measures the difference in years for achieving both goals, was compared among low mortality countries.

Results: Japanese females held the record lowest life disparity from 1980 to 1995. This was because the pace of mortality reduction was faster at ages below the threshold age, compressing deaths into a shorter age interval. Since the 1990s, this pattern has reversed leading to stagnation in life disparity. Additionally, the age profile of mortality decline has closely resembled the most efficient pattern of decline for increasing life expectancy throughout the last 60 years. For many of these years Japanese females also had the lowest efficiency gap.

Conclusions: The Japanese experience shows the benefit of improving mortality conditions at ages with a high concentration of deaths and many years of remaining life expectancy. Moreover, their low efficiency gap means that as life expectancy has increased so too has the certainty of achieving these longevity gains by all members of society.

Introduction

By any measure of longevity Japanese women are the clear leaders. In the lifetable for 2008, their life expectancy at birth was 86 years, they had a median age at death of 89, and the most common age at death was 91.[1] Despite speculation in the late 1990s that the pace of Japanese mortality decline might converge to international levels,[2] the latest figures show that period female life expectancy increased by 2.2 percent from 1999 to 2006, a rate surpassing that of four out of the five next longest living female populations (France, Spain, Switzerland, Sweden but not Italy).

Only 60 years ago, Japan had a mortality level well above western countries, particularly owing to high levels of infant and child mortality. Over the ensuing years Japanese females experienced some of the fastest recorded gains to life expectancy, in excess of 5 years per decade until the late 1960s, before gradually slowing to current levels. Not only have Japanese females maintained the record life expectancy since 1986, they have also managed to increase their life expectancy lead in absolute terms, from less than one year through the 1980s to more than a year and a half since 2000.

Equally exceptional is the homogeneity in lifespans achieved by Japanese females in their ascent to becoming the longest-lived population. According to the life disparity measure $e^{\dagger}$, the average remaining life expectancy at death,[3] [4] Japanese females had the most egalitarian death distribution (i.e. the lowest lifespan variation) for a 15-year period from 1980 to 1995, compared with females from all other countries listed in the Human Mortality Database. While in 1967 Japanese women were dying with an average remaining life expectancy of 11.3 years, by 2007 this life disparity had fallen to 9.3 years. Moreover, as life expectancies increased throughout the country, differences between prefectures actually declined.[5]

In a recent study, Vaupel et al.[6] showed that the observed relationship between high life expectancy and low lifespan variation could be explained by progress in reducing 'early deaths'—i.e. deaths at ages which both increased life expectancy and reduced life disparities. Furthermore, countries with high life expectancies have generally undergone more 'efficient' age patterns of mortality decline in order to increase their life expectancy and reduce their lifespan variation.[7] [8] As the current longevity leaders and the country with among the longest records of having the lowest lifespan variation, the Japanese female population provides an excellent case to examine these concepts.

We begin this chapter by examining the course of mortality improvement since the Second World War. To understand these trends we examine the age profile

of mortality decline by calculating age decompositions of life expectancy gains. We follow with a similar examination of trends in lifespan variation. Finally we match the observed age pattern of mortality change to what would be expected under scenarios of efficiency, both in terms of life expectancy gains and life disparity losses.

Data and Methods

We used female period mortality data from the Human Mortality Database, from 1960 to 2007. Lifespan variation was measured using the 'life disparity' measure $e^{\dagger}$. From the life table, life disparity is calculated according to the formula,

$$e^{\dagger} = \int_0^{\omega} e(x)d(x)dx, \quad (1)$$

where $e(x)$ and $d(x)$ are respectively the remaining life expectancy and death density at age x.[3 4 9 10] Life disparity can be interpreted as the average remaining life expectancy at death in the population. As deaths become compressed into a shorter age interval, on average individuals are dying with fewer years of remaining life expectancy, and $e^{\dagger}$ decreases. If everyone were to die at the same age $e^{\dagger}$ would be zero.

Whether life disparity increases or decreases depends on the changing age profile of deaths. Zhang and Vaupel showed that so long as $e^{\dagger} < e_0$, there existed one unique age, $a^{\dagger}$, that would separate early deaths which compress the age-at-death distribution (and decreases life disparity) from older deaths that expand this distribution.[4] This age is found by interpolation, setting the following function $k(a)$ equal to zero,

$$k(a) = e^{\dagger}(a) - e(a)(1 - H(a)) \quad (2)$$

where $H(a)$ is the cumulative hazard function, $H(a) = \int_0^a \mu(x)dx$ with $H(0)=0$.

We calculated e_0, $e^{\dagger}$, $a^{\dagger}$ and the modal age at death (using the Kannisto method)[13] for all female populations in the Human Mortality Database from single year period life tables, 1947 to 2007. For each year we determined the population with the highest life expectancy, the highest mode, and the lowest life disparity, and additionally compared these trends to Japan, Sweden, France, Italy, USA (some of whom were often the record-holders).

Moving specifically to the Japanese situation, we decomposed life expectancy by age using the Arriaga[11] method between the first and last year of each decade (i.e., 1950 and 1959 up to 2000 and 2005). We followed this with an age decompositions of life disparity between the same years according to the following method. Zhang and Vaupel showed that life disparity could be additively decomposed between any age intervals.[4] In their equation 4, they decomposed $e^{\dagger}$ into early and late life disparity components according to the threshold age $a^{\dagger}$,

$$\dot{e}^{\dagger}(t) = \frac{de^{\dagger}(t)}{dt} = \int_0^{a^{\dagger}(t)} k(a,t)d(a,t)\rho(a,t)da + \int_{a^{\dagger}(t)}^{\omega} k(a,t)d(a,t)\rho(a,t)da. \quad (3)$$

where $\rho(a,t)$ is the rate of progress in reducing death rates, and ω is the oldest age interval (in our case 110+). In addition to the above decomposition, we decomposed $e^{\dagger}$ into single year age intervals in a similar spirit to formula 3,

$$\dot{e}^{\dagger}(t) = \frac{de^{\dagger}(t)}{dt}$$

$$= \int_0^1 k(a,t)d(a,t)\rho(a,t)da + \int_1^2 k(a,t)d(a,t)\rho(a,t)da + ... + \int_{\omega-1}^{\omega} k(a,t)d(a,t)\rho(a,t)da\,. \quad (4)$$

The age-specific profile of mortality improvement can also be thought of in terms of efficiency. Building on work from Keyfitz,[12] and Vaupel,[9] Oeppen[7] proposed that mortality reductions at any given time period were most effective at improving life expectancy over ages having a higher density of deaths and greater remaining life years, according to the function,

$$\varepsilon(a) = d(a)e(a)\,. \quad (5)$$

The function $\varepsilon(a)$ thus determines the efficiency of mortality improvement with respect to improving life expectancy at each age, specifically by measuring the absolute change in life expectancy caused by a proportional reduction in mortality. We contrasted the relative contribution of each age to life expectancy increases over several successive 5-year periods, with the efficiency function calculated from the life table of the initial period using the 5-year pooled period life table data from the Human Mortality Database. We compared how 'efficient' Japan had been in their changing mortality patterns to females France and the United States.

Similarly, changes in life disparity are determined by the specific age patterns of mortality reduction. The function, $\varphi(a) = d(a)\kappa(a)$, measures the efficiency of mortality changes to changes in life disparity.[8] Noticing that improvements in mortality up to the threshold age are efficient both for increasing

life expectancy and reducing life disparity, Zhang et al. introduced the concept of a 'gap in efficiency'.[8] Namely, they calculated that any given age pattern of mortality reduction up to the threshold age would increase life expectancy by δ years more than it would decrease life disparity. In calculating this gap for a number of countries, they noticed that over the past 150 years, the country with the highest life expectancy also had among the lowest gaps in efficiency. We replicated their findings for the same six populations and the record population.

Results

Development of Japanese female longevity

Age decompositions of life expectancy gains show a clear shift in importance from reductions in infant and child mortality to increasing the life years of the elderly population (Table 1). In the 1950s more than half of the gains to life expectancy were gained by reductions in mortality to women aged less than 20 years of age. By the 1970s it was the survivorship of women over 60 years of age who were contributing the greater half of life expectancy gains, and in the 1990s it shifted to women above age 75.

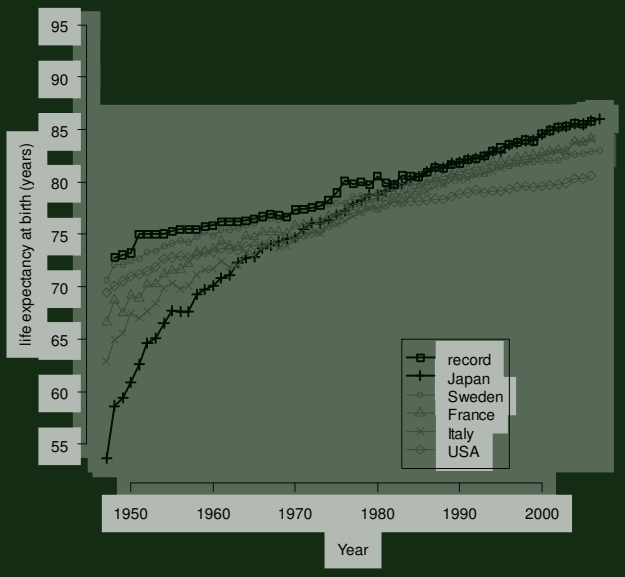
Although older age survivorship only became important to life expectancy gains in the latter half of the period, this is not to say that mortality reductions of the elderly were non-trivial in the initial half. To more clearly illustrate this in Figure 1 we plotted the trends in life expectancy and modal age at death for a selection of mostly low mortality female populations. The mode is a better measure of adult mortality improvement, since it is not influenced by changes in infant and child mortality.[13,14] As compared to the life expectancy trajectory, the improvement in the mode over the past 60 years was close to linear (R^2=0.964) with an improvement of 2.5 years per decade—the same pace as the record life expectancy since 1840. Thus although older ages were contributing more years to life expectancy gains, the linear trend line of the mode suggests that the pace of mortality improvement among the elderly was actually rather stable over this time.

	1950-1959	1960-1969	1970-1979	1980-1989	1990-1999	2000-2005
Relative contribution of age interval						
0	0.19	0.26	0.10	0.06	0.03	0.05
1-4	0.19	0.08	0.03	0.02	0.01	0.02
5-19	0.11	0.06	0.03	0.01	0.02	0.01
20-39	0.26	0.14	0.09	0.04	0.01	0.00
40-59	0.15	0.17	0.19	0.13	0.06	0.10
60-69	0.07	0.12	0.20	0.17	0.11	0.15
70-79	0.03	0.12	0.24	0.31	0.26	0.25
80-89	0.01	0.04	0.11	0.23	0.37	0.31
90+	0.00	0.00	0.01	0.04	0.12	0.13
Life expectancy gain in years						
	9.24	**4.53**	**4.08**	**3.11**	**2.73**	**0.91**

Table 1: Age decomposition of life expectancy changes between the first and last year of each decade, 1950-2005, Japanese females, data from HMD.

Age group	1950-1959	1960-1969	1970-1979	1980-1989	1990-1999	2000-2005
0	-1.29	-0.96	-0.34	-0.17	-0.08	-0.04
1-4	-1.28	-0.31	-0.09	-0.05	-0.03	-0.01
5-19	-0.70	-0.23	-0.11	-0.04	-0.04	-0.01
20-39	-1.52	-0.48	-0.29	-0.11	-0.03	0.00
40-59	-0.62	-0.45	-0.48	-0.26	-0.11	-0.06
60-69	-0.06	-0.16	-0.33	-0.26	-0.16	-0.08
70-79	0.11	0.10	-0.02	-0.18	-0.22	-0.08
80-89	0.12	0.21	0.38	0.36	0.28	0.03
90+	0.01	0.05	0.12	0.26	0.58	0.15
TOTAL	**-5.22**	**-2.23**	**-1.17**	**-0.45**	**0.18**	**-0.10**

Table 2: Age decomposition of life disparity changes, over successive 10-year periods, 1950-2005, Japanese females, data from HMD. The contribution is presented in years.



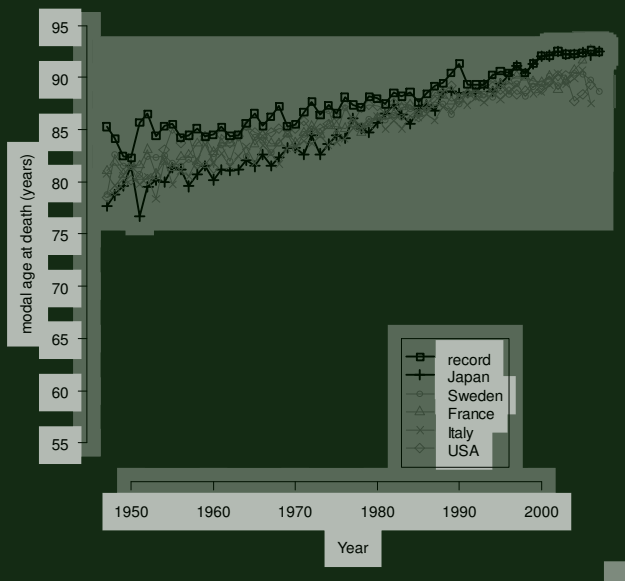


Figure 1: Period life expectancy at birth (left panel) and modal age at death (right panel), females, selected countries. The record country refers to the country with the highest female life expectancy or modal age at death out of all countries in the Human Mortality Database.

Development of low lifespan disparities

Alongside sharply rising life expectancy, female lifespan variation in Japan fell steeply. In Figure 2 we plotted the female life disparity ($e^{\dagger}$) for the same countries of the Human Mortality Database as we plotted in Figure 1.

Again we see that Japan started the post-war period as the worst performer of the group owing primarily to its high levels of infant and child mortality, ages which contributed many lost years of remaining life expectancy. Just over a quarter-century later, Japan overtook Norway to have the world's lowest life disparity, a position it held from 1976 (apart from 1979) until being overtaken by Finland in 1996.

Age decomposition results show that the same young ages that were increasing life expectancy in the 1950s and 60s were also contributing the most to decreases in life disparity during this period (Table 2). From the mid-1970s to the early 1990s, years when Japanese females had the most egalitarian lifespan distribution, improvements in mortality at middle ages (from age 40 to 80) were contributing the most to continued low levels of life disparity. From the 1990s onward, however, continued reductions in mortality under age 80 were offset by improvements in mortality to women over age 80. In fact, the accelerating improvement in mortality at these oldest ages outpaced all other ages and led to stagnating or even small increases in life disparity. This stagnation can also been observed in other low mortality female populations but is more pronounced among Japanese females (Figure 2).

Meanwhile, the threshold age rose considerably throughout the period of study, from age 64 in 1950 to age 84 in 2005. Thus saving the life of an 80 year old would have increased life disparity until 1990, after which time, death at 80 years of age was an 'early death', such that averting the death would decrease life disparity. If we consider lifespan variation before and after this moving threshold age, early life disparity (life disparity from age 0 to age $a^{\dagger}$) fell from 17.0 years in 1947 to 6.2 years in 1988. Late life disparity (life disparity from age $a^{\dagger}$ to age 110+) fell from 4.5 in 1947 to 3.2 years in 1988. The two: early and late life disparity, sum to total life disparity. Although mortality has continued to come down at all ages, life disparity has since stagnated, and the breakdown into early and late life disparity levels has remained constant.

In addition to the low lifespan variation found at the national level, regionally, women could expect to live on average to the same age regardless of the prefecture in which they lived. In comparison with the 26 régions of France and the 51 states of the United States of America (includes the District of Columbia), the 47

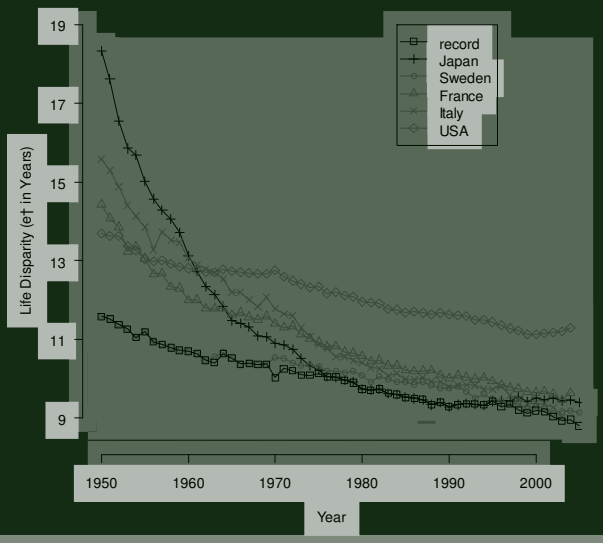


Figure 2: Period life disparity, females, selected countries. The record country refers to the lowest observed life disparity during the year. Data is from the Human Mortality Database.

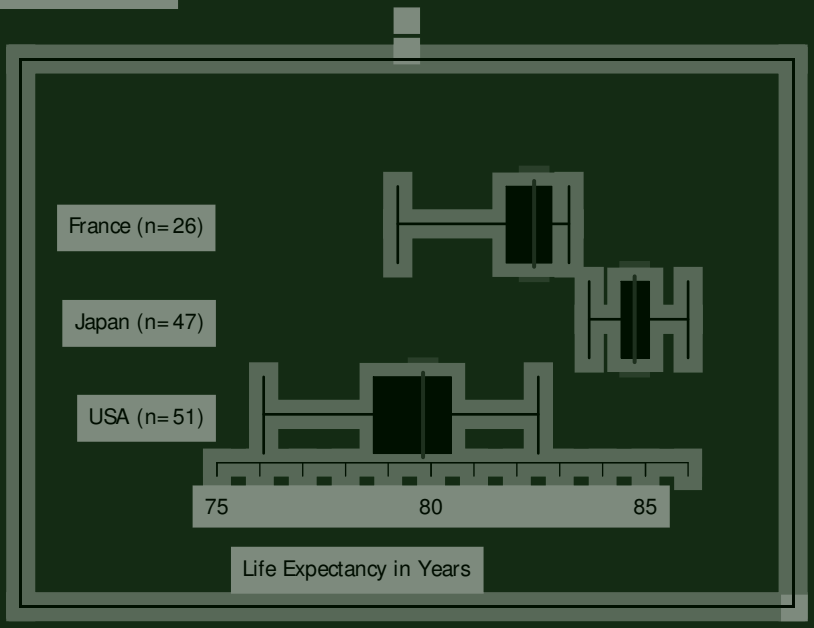


Figure 3: Box plot of female period life expectancy at the regional level in France (26 Régions), Japan (47 Prefectures) and the United States (51 States or Districts) in the Year 2000. The black box contains all regions having life expectancies between the 25th and 75th percentile while the central vertical bar pertains to the region with the median life expectancy. We are grateful to Roland Rau for providing the figure.

prefectures showed little difference in life expectancy (Figure 3). In fact the female life expectancy in every one of the Japanese prefectures lay above even the longest living région in France or best performing American state.

Efficiency of mortality decline

We have shown how the age-specific mortality improvement of Japanese females led to both a rapid and sustained increase in life expectancy, and to a sharp initial decrease in life disparity, followed by recent stagnation. We also examined whether these particular patterns, which led them to attain record life expectancy and for some years the lowest life disparity levels, were efficient patterns of mortality improvement both in terms of improving life expectancy and reducing life disparity.

In Figure 4 we plotted the efficiency function over successive 5-year pooled life tables, overlaid by the actual age pattern of mortality reduction during this time, obtained by age decomposition. We can see that as the efficiency function has shifted to changes in mortality at older ages, Japanese females have changed their age profile of mortality decline to match these changes. This can also be seen in France and other successful low mortality countries. It is less apparent in the United States, where mortality has stagnated in early adulthood, or in many Eastern European countries which failed to shift the age pattern of mortality decline to higher ages. In Figure 5 we plotted the change in the age profile of efficiency and mortality decline for France and the United States by way of comparison.

Of course, only improvements below the threshold age, $a^{\dagger}$, reduce life disparity while improvements in mortality above this age increase the dispersion in lifespans. Recently, age specific contributions to changes in life disparity for Japanese females have been overly concentrated in the ages 60 to 100, which have both positive and negative impacts on the overall life disparity level. This makes efficiency a controversial concept to measure for lifespan variation: lowering life disparity would require both decreasing premature mortality and *increasing* late life mortality.

However, by focussing only on mortality up to the threshold age which both increases life expectancy and decreases life disparity, we could determine the efficiency gap for a group of countries. This gap measures how many additional years are gained in life expectancy than reduced in life disparity. Thus a falling gap would imply that progress in prolonging lives was becoming more equally allocated within society. In Figure 6 we plotted the efficiency gap for six populations and the record population. Japanese females had the lowest efficiency gap for most of period from the mid 1970s to the mid 1990s.

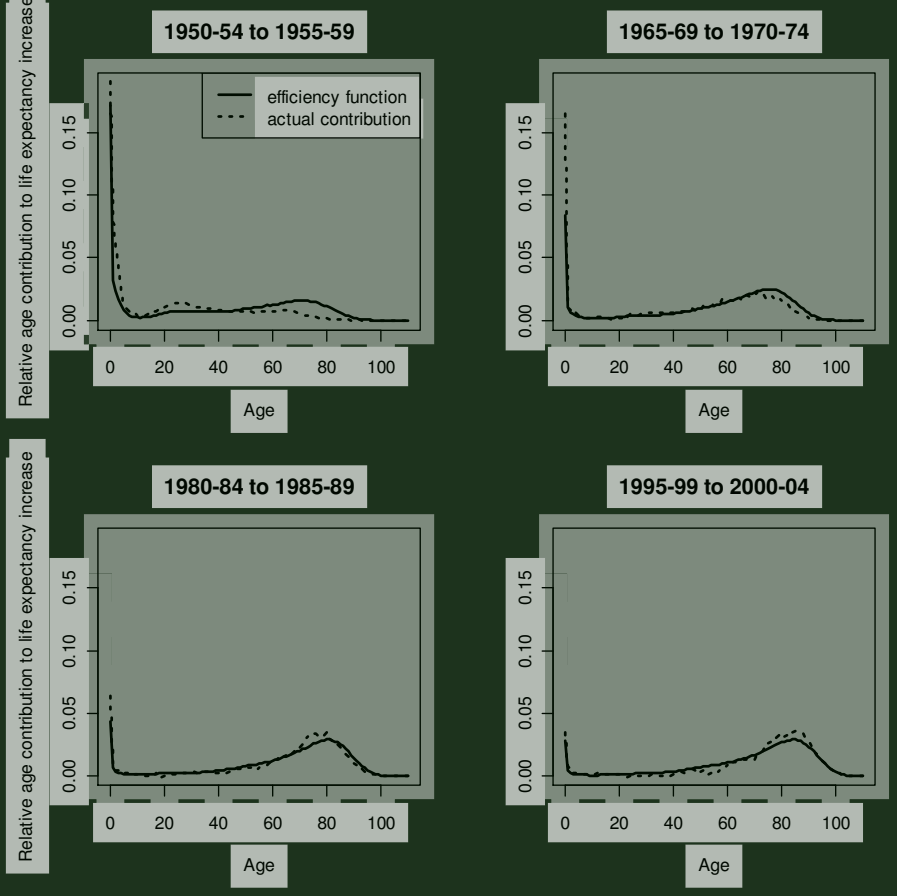


Figure 4: The relative contribution of each age to life expectancy increases (dotted line) over the period is contrasted with the efficiency function (solid line) calculated from the life table of the initial period. Data is the 5-year pooled period life table data from the Human Mortality Database, Japanese females.

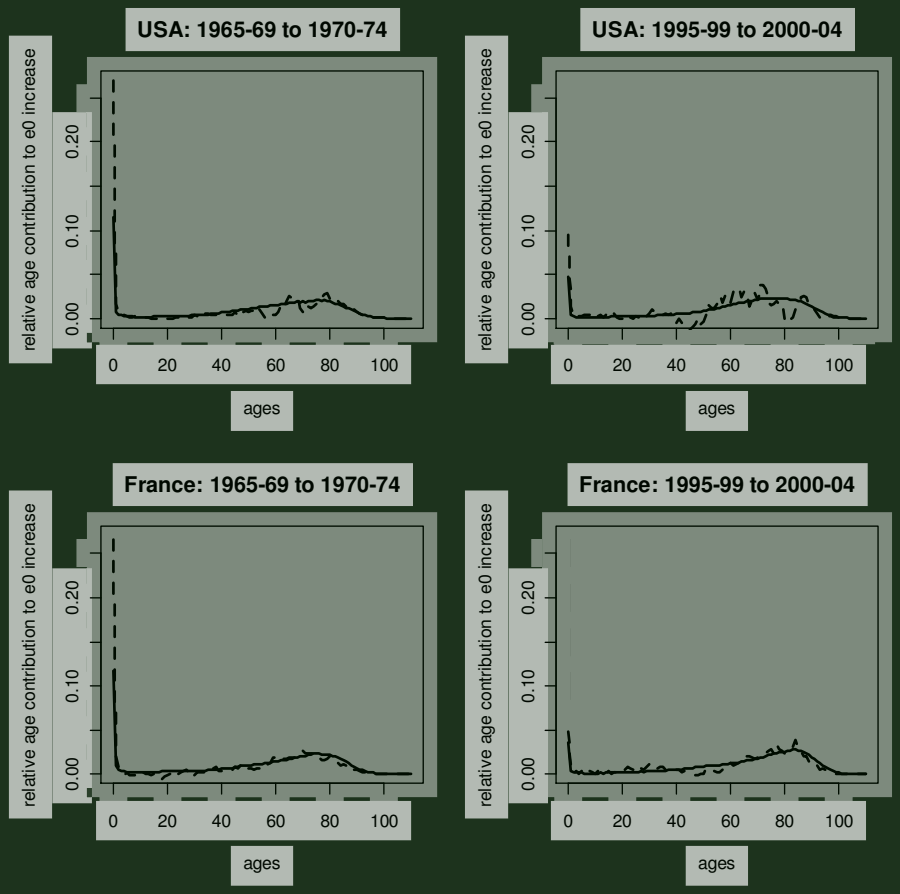


Figure 5: The relative contribution of each age to life expectancy increases (dotted line) over the period is contrasted with the efficiency function (solid line) calculated from the life table of the initial period. Data is the 5-year aggregated life table data from the Human Mortality Database, females.

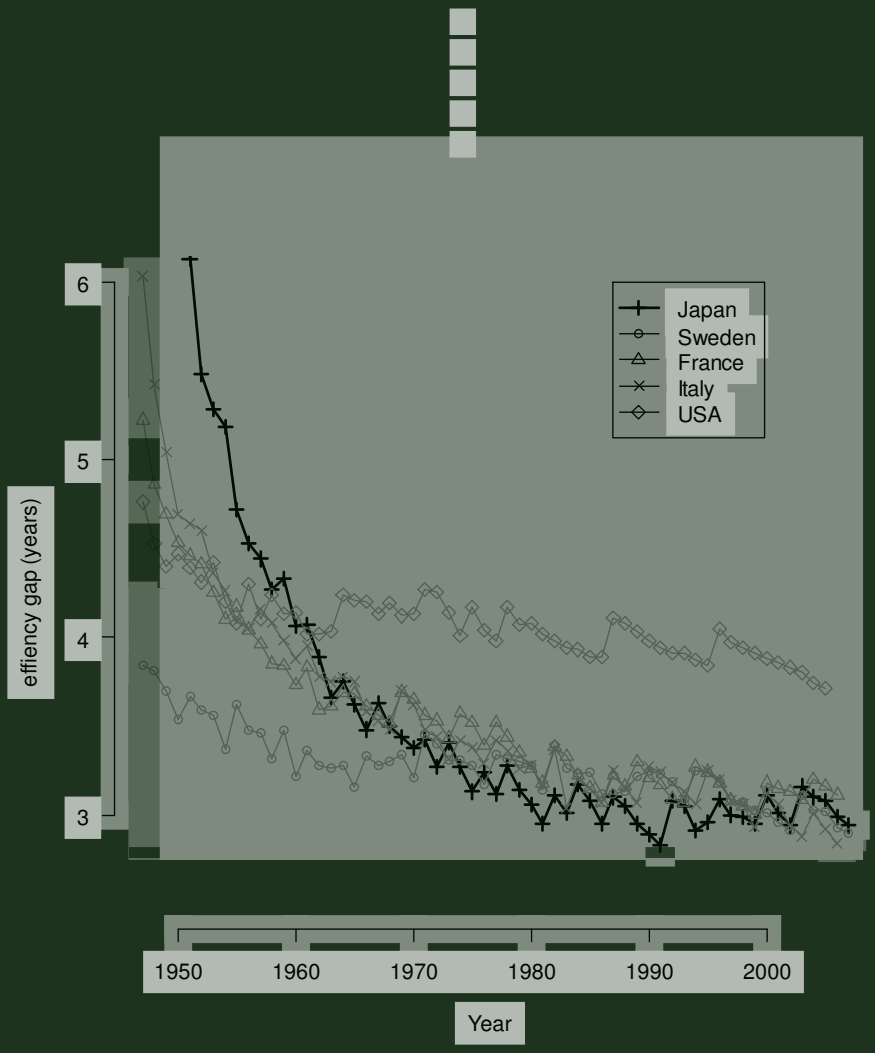


Figure 6: Efficiency gap of mortality improvement, females, selected countries of the HMD. It is remarkable that for most of this period Japan had the lowest efficiency gap, meaning that gains to life expectancy were also helping to reduce life disparities.

Discussion

The general phenomenon of the most pronounced mortality decline shifting to older ages has been labelled an "aging of mortality decline" by Horiuchi and Wilmoth cited in Wilmoth.[15] From a theoretical perspective, Tuljapurkar et al.[16] argue that societies typically direct resources proportionally to observed mortality levels at different ages. As causes of death have become more complicated with time, the marginal effectiveness of the resource level has decreased. In order to compensate for this process the level of resources has had to increase. This process can be seen through shifts in Japanese health policy, which has directed resources to ages with higher efficiency in mortality reduction. These policy shifts are consistent with the changing mortality patterns brought about through the epidemiologic transition.[17]

Following the Second World War new public health laws were created which established health centres, enacted controls against tuberculosis and provided basic vaccinations. At the same time, the prior education reforms of 1890 mandating female education [5] meant that the cohorts of women coming into childbearing age were among the first educated female cohorts, the importance of which has often been highlighted as a factor in reducing infant deaths.[18 19] Together these measures, along with imported medical technology from the west, were particularly effective in reducing mortality from infectious diseases.

In the mid to late twentieth century, although survivorship was beginning to catch up with western countries at younger ages, Japan was far from exceptional when it came to survival probabilities at advanced ages. In a comparison of remaining life expectancy at age 80 of the 1880-1894 birth cohorts, Japanese females were the worst performers of a 5-country group which included England, Sweden, France and the US.[20]

By the 1960s attention shifted to expanding coverage of the health care system, and a new focus on treatment of chronic diseases emerged. Health insurance covered the entire population by 1961 (initially with 50 percent benefit coverage, expanded to 70 percent benefit coverage in 1968). Around this time the National Cancer Centre was established and control programmes begun for cancer (1965) and cardiovascular disease (1968). The number of hospital beds per 10,000 population doubled between 1954 and 1977 in Japan, in a period where it stagnated or even decreased in many other industrialised countries.[21]

Already by 1982 the focus of the health system shifted to treating the rapidly expanding elderly population with the passing of the Health Promotion Law. As resources shifted to the elderly, mortality reductions at older ages became

prominent. Christensen et al.[22] remarked that the probability of surviving from age 80 to 90 had by the mid-2000s increased to over 50 percent. Rau et al.[23] noted that the mortality of females above age 90 dropped on average by around 3 percent per year in the period 2000-2004, a time when other countries experienced only "rather modest" improvements. In this vein, the 12-fold increase in centenarians per 10,000 members of each birth cohort from 1973 to 2000 led to the most rapidly observed decrease in centenarian doubling time, although the increase started from a low base.[24] [25] The more general trend of mortality shifting to older ages among Japanese females was documented by Iishii, who noted the problem of fitting conventional projection methods to such a pattern of mortality change.[26]

These recent changes account for the recent stagnation in lifespan variation—mortality declines at older ages are keeping pace with declines at younger ages. A similar dynamic is observed in mortality above the modal age at death. Japanese females have experienced stagnation in lifespan variation above the mode since around the mid-1980s,[27] a process not yet clearly pronounced in other countries except for maybe France.[28]

Yet health policy alone does not appear to tell the whole story; behavioural, economic and selection factors must also be taken into account. Japanese females are thought to have a range of healthy behaviours which contribute to their longevity (for an overview see Marmot and Smith).[29] Most importantly, females in Japan, like in other leading life expectancy countries such as France, Spain and Italy took up smoking later than in other high income countries. However recent estimates put the smoking attributable fraction of deaths to females aged above 50 in Japan at 13 percent – a sharp increase from 2 percent in 1980 and higher than in most western and southern European countries.[30] While the low initial levels coincide with the earlier ascent to high life expectancy levels and low life disparity levels, this more recent increase in the smoking attributable fraction might be part of the reason why life disparity has been stagnating in recent years.

In terms of economic factors, Japan started the post-war period with low income levels by western standards but experienced rapid economic growth in the half century that followed. Even in the latter part of the period, Japanese per capita GDP increased by 83 percent from 1970 to 1989. By comparison the EU-15 and the United States saw per capita GDP growth of 58 and 54 percent respectively over this same period.[i] While between countries we would not necessarily expect economic

i Calculated using GDP per head, US$, constant prices, constant PPP, reference year 2000. OECD Economic Indicators Database. Available at http://stats.oecd.org/Index.aspx (data downloaded on 19/06/2009).

growth to translate into mortality reductions of the same magnitude given the curvilinear relationship between income and life expectancy,[32] within Japan economic factors were shown to have played a role in mortality decline. Up until the 1980s regression analysis showed the differences in infant mortality between prefectures to be driven more by broad social factors such as education and per capita GDP than by differences in the health sector such as the number of doctors or midwives.[5] Yet the much slower rate of economic growth since the 1990s has not slowed the rate of life expectancy increases. While this time period also coincides with the beginning of the life disparity stagnation, it is difficult to imagine a causal pathway, unless economic growth (and access to health care) was only concentrated among certain pockets of the population. However the Gini coefficient of household income showed only a modest increase through this period.[ii] Thus the changing national income of Japan is only likely to have been a factor in the early post-war mortality reductions.

Finally, low mortality among older Japanese could be owing to selection effects. The rapid pace of mortality decline in Japan means that the current elderly population were born and raised through a period of high infectious disease loads. If the frailer individuals died off, the pool of survivors would be selected for robustness and could for some time experience high period life expectancies.[33] This selection effect could also explain the relatively lower life disparity levels in Japan if the group of survivors were generally more robust and homogeneous individuals.

Conclusion

In this chapter we have shown how the high life expectancy and low lifespan disparities enjoyed by the female Japanese population have resulted from changing age specific progress in reducing mortality. Following the Second World War, policies aimed at controlling infectious diseases led to rapid (and relatively cheap) reductions in infant and child mortality. Success at these ages was then superseded by mortality improvement among middle aged Japanese women, through targeted programs to reduce cardiovascular disease and cancers. Finally in recent years, the oldest segments of the female population have enjoyed the greatest pace of mortality decline and policy has turned to care for the elderly.

[i] Data from the World Income Inequality Database, downloaded 25/01/2011.

As the age pattern of mortality improvement has shifted, so too has the relative contributions of each age to life expectancy improvements and life disparity reductions. Initial age patterns of mortality decline that were concentrated in early ages rapidly increased life expectancy and reduced life disparity. As the "aging of mortality decline" process took hold, these patterns moved outwards to older ages, life expectancy continued to improve but the effects on life disparity became mixed. With more deaths being concentrated in 'older ages', life disparity has been stagnating since the mid-1990s, in addition to the previously remarked upon stagnation in the standard deviation in ages at death above the mode. Nevertheless, as compared to other countries, the efficiency gap remains low.

In this chapter we have focussed exclusively on mortality. An interesting avenue for further research is to examine what has happened to the age distribution of healthy or disability-free life years in Japan. A recent study found that much of the gain in life expectancy among Japanese females over the past 20 years were years spent in only average or poor self-rated health.[34] Provided that the proportion of still-living individuals in worse states of health increases with age, the efficiency function for healthy life years would shift to younger ages because the weights of life years in good health is larger at younger ages than it is for total life years. For the same reason we would expect less dispersion in the distribution of healthy life years than in the distribution of lifespans. If indeed the gains in life years have mostly been in poor health, the stagnation in the total life disparity implies that the distribution of healthy life years has compressed over time, to compensate the expansion of life years lived in average or poor self-rated health. The data used in this chapter were from the Human Mortality Database, which sources its data from census-based population estimates and vital registration. In light of the recent discovery of 234,354 "missing centenarians",[35] the quality of Japanese mortality statistics have come under scrutiny. The missing centenarian issue affects the family registry only. The census data, on the other hand, is considered reliable. Censuses are conducted every 5 years by field workers who directly visit households. Death counts are provided by the Ministry of Health, Labour and Welfare. Vital registration has been required by law since 1899, and deaths for the period covered by the database are considered to be "complete and of good quality".[36] Moreover, lifetable data at the oldest ages are smoothed by the Kannisto method.[37] Thus we do not expect the recent "missing centenarian" scandal to have affected any of the conclusions made in this chapter.

In summary, Japanese females continue to benefit from efficient patterns of life expectancy improvement, their recent pace of life expectancy gains remains high by international standards, and their leadership position among nations continues.

Perhaps even more importantly, Japan has done well to ensure that these gains in longevity are being enjoyed by all female members of society.

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CHAPTER 5

More variation in lifespan in lower educated groups: Evidence from Europe

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CHAPTER 5

ABSTRACT

Background: While it is well established that people with a lower socioeconomic position have a shorter average lifespan, it is less clear what the variability surrounding these averages is. We set out to examine whether lower educated groups face greater variation in lifespans in addition to having a shorter life expectancy, in order to identify entry-points for policies to reduce the impact of socioeconomic position on mortality.

Methods: We used harmonized, census-based mortality data from 10 European countries to construct life tables by sex and educational level (low, medium, high). Variation in lifespans was measured by the standard deviation conditional upon survival to age 35. We also decomposed differences between educational groups in lifespan variation by age and cause of death.

Results: Lifespan variation was higher among the lower educated in every country, but more so among men and in Eastern Europe. Although there was an inverse relationship between average life expectancy and its standard deviation, the first did not completely predict the latter. Greater lifespan variation in lower educated groups was largely driven by conditions causing death at younger ages, such as injuries and neoplasms.

Conclusions: Lower educated individuals not only have shorter life expectancies, but also face greater uncertainty about the age at which they will die. More priority should be given to efforts to reduce the risk of an early death among the lower educated, e.g. by strengthening protective policies within and outside the health care system.

Introduction

By now it is well established that *on average* higher socioeconomic groups live longer than lower socioeconomic groups. Regardless if the proxy used for socioeconomic status is education, wealth, income, occupation, or even housing tenure, the more advantaged groups have higher survival probabilities at every age, and from most causes of death.[1-9]

Yet conventional research employing measures of average age of death such as life expectancy overlook the distribution around these averages. This matters because larger lifespan variation (differences between individuals in their age at death) implies greater uncertainty in the timing of death at the individual level, and thus in the planning of life's events. At the societal level larger lifespan variation suggests lack of effectiveness of policies which aim at protecting vulnerable individuals against the vicissitudes of life, such as social safety nets.[10]

To date, a scattering of evidence from Russia[11] and the United States[12] suggests that lower socioeconomic groups indeed face greater lifespan variation. But up until now no large international study has examined the association between socioeconomic status and lifespan variation. It is also not known whether the same causes that are leading to lower life expectancies are also causing the presumed higher lifespan variation. In this paper we set out to examine whether the finding of a socioeconomic gradient to lifespan variation is a replicable finding, across a range of European countries, using educational level as a proxy for socioeconomic status. In order to determine the added value of lifespan variation as a measure of inequalities in health, we also analyse the association between lifespan variation and average life-expectancy by educational group. Finally, we decompose the differences between educational groups in lifespan variation by age and cause of death.

Data and Methods

We used data from 10 European countries, harmonized as part of the Eurothine project. This consisted of census-based death and exposure counts by sex, cause-of-death and level of education. For some countries we had longitudinal data following individuals aged 30+ for 5-10 years beginning around 1990, for other countries we had cross-sectional data aggregated over a few years around 2000. In all cases data was aggregated into 5 year age intervals with an open-aged interval at age 85+. The

education levels were coded according to the ISCED classifications, and split into 3 internationally comparable categories. These corresponded to: less than secondary education (low), complete secondary education (medium), and some tertiary education (high). We regrouped the causes of death into four broad causes: neoplasm (140-239 ICD 9; C00-D48 ICD 10), circulatory diseases (390-459 ICD 9; I00-I99 ICD 10), external causes (E800-999 ICD 9; V01-Y98 ICD 10) and all remaining other causes including missing or ill-defined. More details on the data can be found in the appendix to chapter 5.

In order to get a more continuous age-at-death distribution, we proportioned out the corresponding shares of the total death and exposure counts by level of education from the Eurothine data to the national data reported by single year of age in the Human Mortality Database[13] for the equivalent time period. We assumed that the proportion of the total deaths and exposures by educational level remained constant within each 5-year age interval. We likewise assumed that these proportions found in the open-aged category 85+ (75+ in Sweden) were the same as those observed in the oldest preceding age category. A previous study showed this to be the case for women, but to risk overestimating differences for men, who were shown to have decreasing rate ratios between educational groups up to ages 90+.[14] This gave us death and exposure counts by single year of age (35-110+), sex and educational level, which we used to calculate death rates. From these death rates we constructed life tables using conventional methods, including fitting a Kannisto model of mortality to older ages.[15 16]

The correlation between lifespan variation and average lifespan has led some to argue that one should be examined within the context of the other.[17] Thus, for each subgroup we calculated both the average lifespan conditional upon survival to age 35 (e_{35} + 35 in conventional life table notation) and lifespan variation, measured by the standard deviation (S_{35}), also conditional upon survival to age 35. Both measures were calculated from the life table death density. In this way our age-at-death distributions were not confounded by differences in the underlying age structure of each subpopulation. The standard deviation has become a popular measure to quantify the dispersion in the age-at-death distribution.[12 18-21] From lifetables the standard deviation is calculated by taking the square root of the variance—itself the squared distance of each individual's lifespan to the subgroup average lifespan divided by the population size. S_{35} is measured in years with higher values indicating greater lifespan variation.

The overall level of lifespan variation comes from the balance of postponing premature deaths (compressing the age-at-death distribution) and saving lives at older ages (which expands this distribution). Separating the two is a unique

threshold age, generally found slightly below the population life expectancy.[22] In the countries and years used in our analysis for men this age ranged between 65 and 70 in western European countries and between 60 and 65 in eastern European countries (younger in Estonia). For women the threshold age ranged between 70 and 80 in all countries but Estonia.

To get a better interpretation of the reasons behind differences in lifespan variation by levels of education, we decomposed the Eurothine data by age and cause of death. Decomposition was done by step-wise decomposition[23 24] by modifying a Visual Basic for Applications program developed by Shkolnikov and Andreev.[25] We compared differences in S_{35} between the low educated group and the combined middle and high educated groups over ages 35-69 for men, and 35-74 for women, the same upper age limits used by Shkolnikov et al.[24] These ages broadly corresponded to the middle adult ages that both reduced lifespan variation and increased life expectancy. Moreover, causes-of-death at older ages are generally more difficult to determine because of the interaction of multiple underlying causes,[26 27] and because of the use of an open-aged interval.

Results

A clear educational gradient existed not only in the average lifespan (Table 1) but also in lifespan variation (Table 2). This was the case for both men and women, in every country examined. Low educated men had a standard deviation in lifespans of 9-11 years (and even 12.8 years in Estonia), which is on average 30 % higher (2.4 years) than high educated men in the same country. Low educated women fared a little better—with S_{35} levels of around 8-9 years – but this is still on average 2.2 years higher than the S_{35} of the high educated. Swiss women were the only exception to this gradient, because the high educated there had the same S_{35} as the medium educated group.

Men in the Czech Republic, Sweden, Norway, and Belgium had lower overall levels of S_{35} than men in the rest of the countries. Women always had lower S_{35} than men in the same country. Women also had a less pronounced educational gradient everywhere except for in Estonia, where the S_{35} was exceptionally high among low educated women, and the levels were comparable to Western Europe among the high educated. Generally countries with high overall S_{35} also had a larger educational gradient in S_{35}, with the exception of the Czech Republic, which had low overall lifespan variation but a larger socioeconomic gradient, particularly among

	Male				Female			
Country	**Low**	**Med.**	**High**	**Total pop.**	**Low**	**Med.**	**High**	**Total pop.**
Belgium	73.6	75.9	78.1	**74.5**	80.1	82.2	82.7	**80.9**
Czech Republic†	70.8	77.3	80.8	**73.0**	78.4	81.9	83.9	**79.9**
Estonia†	62.5	67.4	75.3	**67.2**	73.6	77.8	81.6	**76.9**
Finland	72.7	74.9	78.0	**74.1**	79.9	81.7	82.9	**80.7**
France‡	74.0	77.1	80.4	**75.6**	82.3	84.4	84.8	**82.8**
Norway	74.1	76.6	79.2	**76.1**	80.0	82.2	83.6	**81.5**
Poland†	69.4	76.2	79.7	**71.8**	78.4	82.2	83.9	**80.0**
Slovenia	69.6	73.4	77.4	**72.2**	78.4	80.6	82.3	**79.4**
Sweden	75.7	77.8	80.3	**77.1**	80.7	82.7	85.0	**82.3**
Switzerland	74.1	77.3	79.8	**77.0**	81.7	83.4	84.4	**82.7**
Average	71.7	75.4	78.9	**73.9**	79.4	81.9	83.5	**80.7**
Range (years)	13.2	10.4	5.5	**9.9**	8.7	6.6	3.4	**5.9**

† Estimated from census-unlinked data and might be less reliable.
‡ Estimated from a 1% population survey—greater uncertainty surrounding these estimates.

Table 1: Average lifespan conditional upon survival to age 35, for each educational subgroup

	Male				Female			
Country	**Low**	**Med.**	**High**	**Total pop.**	**Low**	**Med.**	**High**	**Total pop.**
Belgium	9.1	8.7	8.1	**8.6**	8.1	7.8	7.5	**7.7**
Czech Republic†	9.1	8.7	7.2	**8.9**	7.5	7.4	4.5	**6.8**
Estonia†	12.8	11.5	9.5	**11.5**	12.2	9.2	6.4	**9.1**
Finland	10.5	10.1	8.0	**9.6**	8.3	7.0	6.4	**7.2**
France‡	10.8	9.8	8.1	**9.9**	8.3	6.8	5.7	**7.5**
Norway	9.5	8.4	7.2	**8.3**	8.7	7.3	6.3	**7.5**
Poland†	10.6	9.8	8.4	**10.2**	8.7	8.1	6.0	**7.7**
Slovenia	10.7	9.6	8.3	**9.8**	8.4	8.1	7.4	**7.9**
Sweden	8.9	8.3	7.0	**8.1**	8.4	7.7	7.2	**7.4**
Switzerland	10.5	8.8	7.6	**8.7**	8.0	7.1	7.1	**7.2**
Average	10.3	9.4	7.9	**9.4**	8.7	7.6	6.5	**7.6**
Range (years)	3.9	3.2	2.4	**3.4**	4.7	2.4	2.9	**2.3**

† Estimated from census-unlinked data and might be less reliable.
‡ Estimated from a 1% population survey—greater uncertainty surrounding these estimates.

Table 2 Standard deviation in lifespans conditional upon survival to age 35, for each educational subgroup

women.

Looking across countries, the range in S_{35} was larger across low educated groups than it was across high educated groups. Lifespan variation was particularly high for low educated men in Eastern Europe (with the exception of the Czech Republic) and in Finland, France and Switzerland.

The main reason for the differences between educational subgroups in S_{35} is the longer left tail of the lifespan distribution among the lower educated, aspects that can be hidden in summary measures such as the mean or median. To better visualize these differences, we plotted the lifespan distributions for men in Sweden and the Czech Republic (Figure 1). Of course saving lives at older ages can also bring about higher lifespan variation. In our data, the highest educated Swiss and Swedish women showed some evidence of this, and this explains why the Swiss high and medium educated women had similar S_{35}.

In Figure 2, we plotted the average lifespan versus S_{35} for each sex, educational group, and country combination. While there is a strong relationship between high lifespans and low S_{35}, some populations show high levels of lifespan variation for their average lifespan (see also Tables 1 and 2 for country points that are not labelled). For example, S_{35} was a full year higher for low educated men in Switzerland than in Norway, despite both groups having the same average lifespan.

The ages and causes of death contributing to the higher lifespan variation of the low educated groups can be seen in Figure 3 for men and Figure 4 for women. In general, differences between educational groups appeared at earlier ages among men. Women had a flatter age gradient, meaning that all ages were equally contributing to the differences in S_{35} between educational groups. Mortality from external causes made substantial contributions to the larger S_{35} among lower educated groups, especially in Estonia, Finland, Slovenia (men), Poland (men) and Switzerland (men). Neoplasms contributed to the educational gradient in S_{35} for both sexes in the Czech Republic, Poland, and Switzerland and also among women in Norway and Sweden. Circulatory diseases played a similar, non-trivial role in all countries.

After around age 65-70 for men and 70-75 for women (in some countries earlier) the age contribution became negative. This coincided with the threshold age separating compression and expansion of the lifespan distribution. The lower survival rates of the low educated group beyond this age reduced the differences in lifespan variation between the groups.

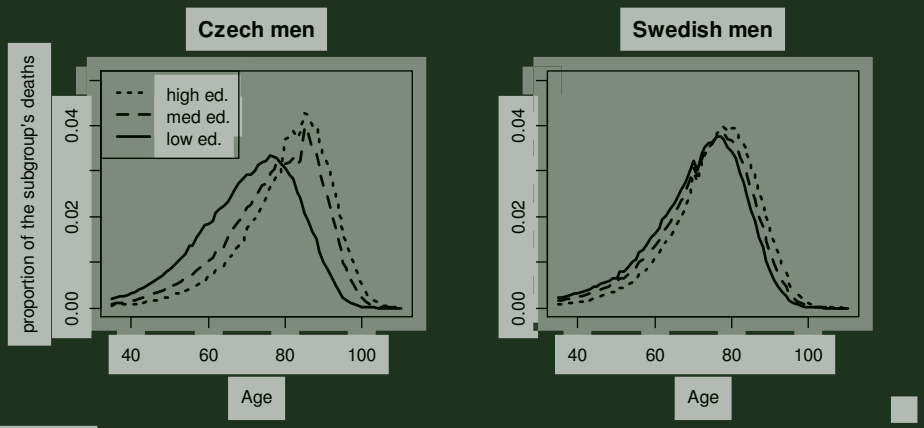

Figure 1: Life table death densities of the different educational subgroups in the Czech Republic and Sweden.

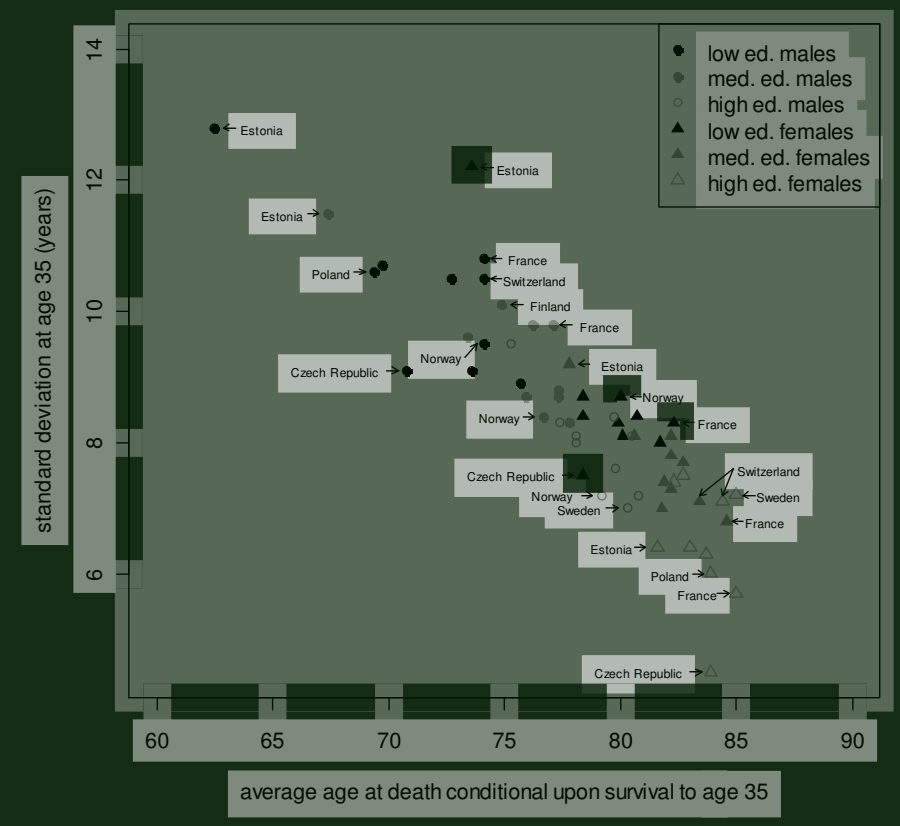

Figure 2: Relationship between uncertainty in the timing of death (life disparity at age 35) and average lifespan (conditional upon survival to age 35) by sex and level of education. All data points in Tables 1 and 2 are plotted, but some are not labelled to avoid clutter.

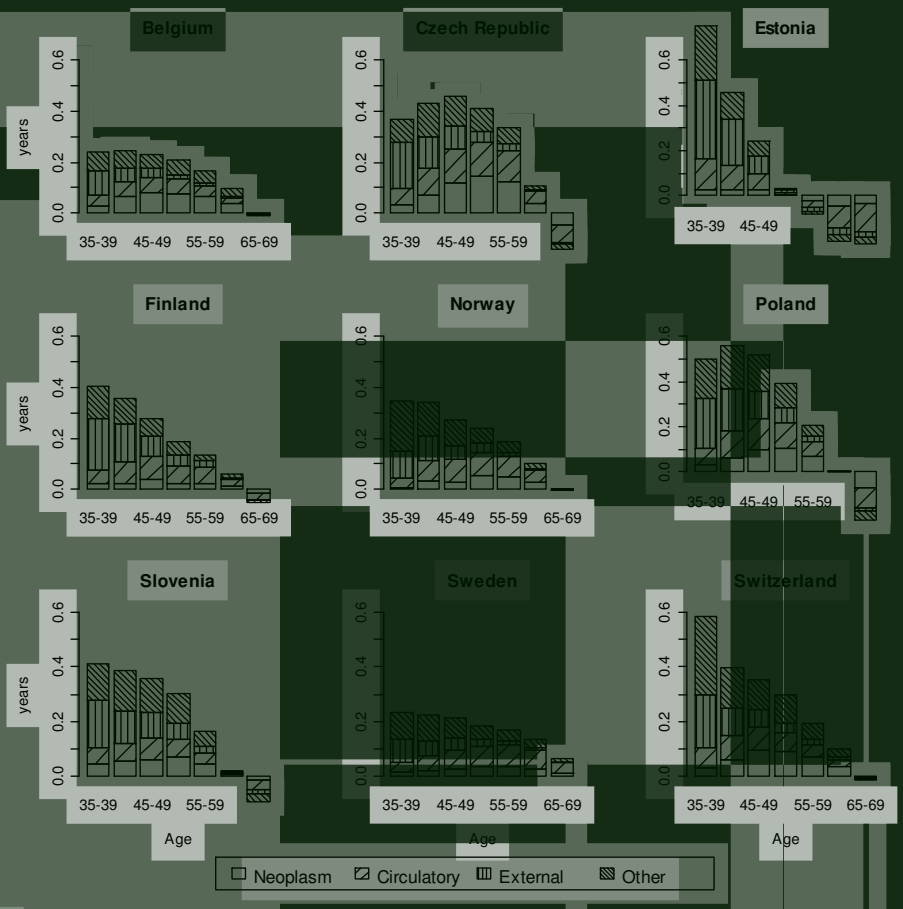

Figure 3: The contribution by age and cause-of-death in years to the greater male S_{35} of the medium and high educated groups combined compared to the low educated group, over age ranges 35-70. The data were collected as part of the Eurothine project.

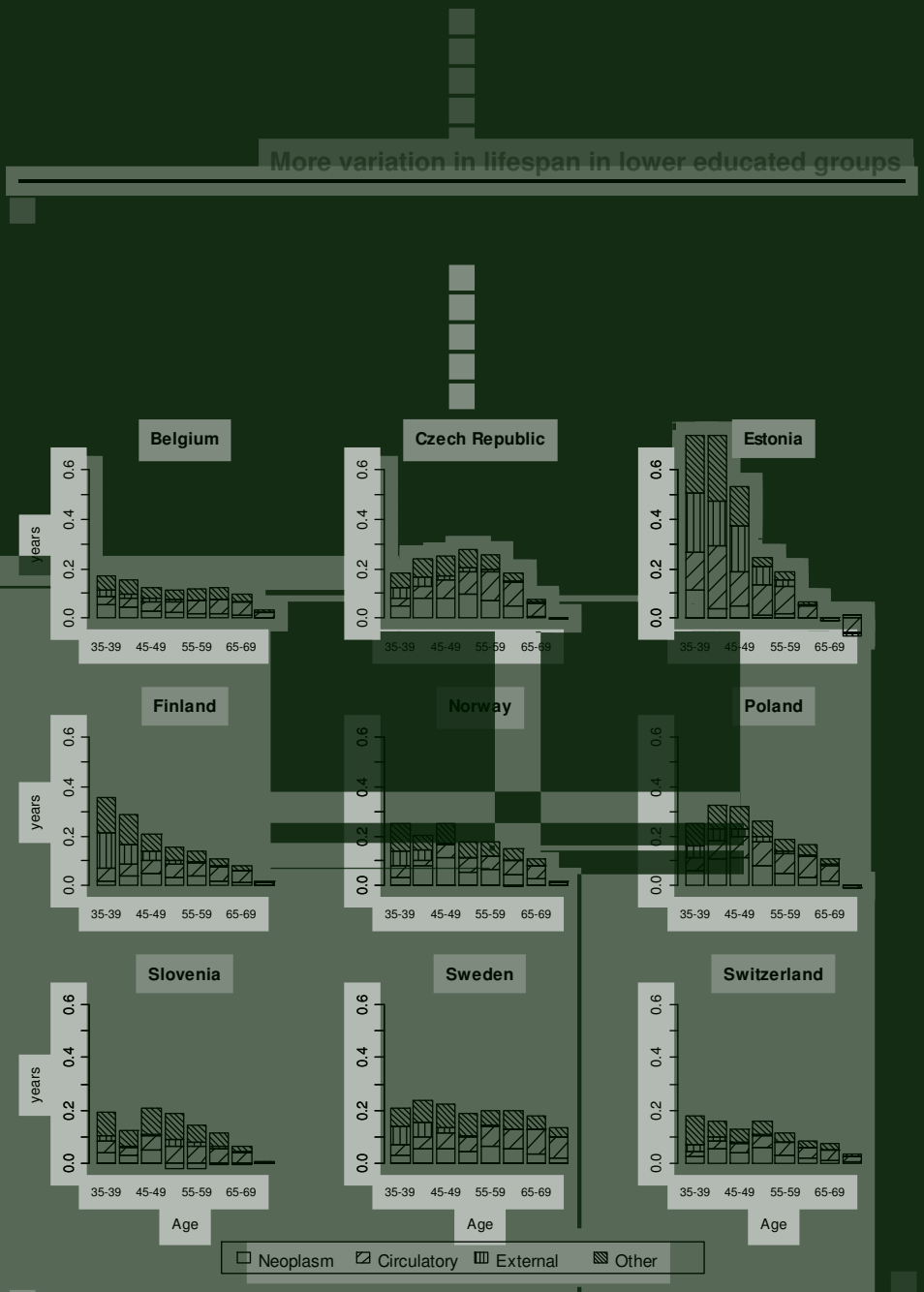

Figure 4: The contribution by age and cause-of-death in years to the greater female S_{35} of the medium and high educated groups combined compared to the low educated group, over age ranges 35-75. The data were collected as part of the Eurothine project.

We also decomposed differences between the low educated and the combined medium and high educated groups in average lifespan, instead of lifespan variation (see Figures S3 and S4 of the appendix). By comparison, educational differences in average lifespan tended to be driven by inequalities in mortality above age 50, and as a result, circulatory diseases played a greater role in explaining the lower lifespans of the low educated than they did for the higher S_{35}.

Discussion

Summary of results

Lifespan variation was higher among the lower educated in every country, but more so among men and in Eastern Europe. Although there was an inverse relationship between average life expectancy and its standard deviation, the first did not completely predict the latter. Greater lifespan variation in lower educated groups was largely driven by conditions causing death at younger ages, such as injuries and neoplasms.

Comparison to other studies

Ours is the first large-scale study of lifespan variation by an indicator of socioeconomic position. It confirms earlier findings from Russia and the United States that lower socioeconomic status is associated with larger variability in the timing of death.[11 12 24] A socioeconomic gradient in lifespan variation was found in all European countries participating in our study, but it was larger in Eastern Europe than in Western Europe

No previous study has examined the contribution of causes of death to socioeconomic inequalities in lifespan variation. A few studies have examined the contribution of causes of death to lifespan variation within whole national populations. Shkolnikov et al. found that external causes explained more of the differences in the Gini coefficient of mortality between the US and England and Wales than it did in explaining differences in average life expectancy.[11] Edwards and Tuljapurkar found that external cause mortality explained as much as 10 % of the total lifespan variation in the United States, but did not explain the differences in rankings between a number of high income countries.[12] Our findings show that

external causes also contribute importantly to socioeconomic inequalities in lifespan variation.

Explanation

Lower educated individuals not only have shorter life expectancies, but also face greater uncertainty about the age at which they will die. This is caused primarily by their elevated premature mortality. Relative inequalities between socioeconomic groups are generally larger in younger age groups than in older age groups.[6] In our study, the especially high lifespan variation of low educated men was due to higher mortality over ages 35-55. This is in contrast to differences in life expectancy between populations, which tend to be driven by mortality over age ranges that have the most deaths,[28 29] as we have also shown here. Thus, reducing premature mortality is even more important for equalizing lifespan variation across educational groups than it is for equalizing average lifespans.

The higher premature mortality of lower socioeconomic groups is caused in part by behavioural differences (especially regarding cigarette smoking, diet, exercise and alcohol abuse),[30-37] material factors (such as financial difficulties and hazardous housing and working conditions),[38-40] and psychosocial pathways (such as psychosocial stress and lack of social support).[41-44] Socioeconomic inequalities in premature mortality, and thus in lifespan variation, are larger among men than among women, because many determinants of premature mortality are more strongly socially patterned among men.[5 45 46]

Country differences in average lifespan as well as in lifespan variation were largest among low educated men. This suggests, first, that the higher educated are less dependent on country-specific circumstances for their survival than the lower educated, and, second, that some countries are more effective than others in protecting lower socioeconomic groups against premature mortality. The much higher lifespan variation among lower educated groups in Eastern Europe could well be a sign of failing social security or failing health and health care policies. It has been found that the considerably higher rate of external mortality among lower educated men in Eastern Europe, which contributes to their higher lifespan variation, is related to a pattern of hazardous drinking,[8 47] which in its turn probably reflects the stress which the lower educated have experienced since the economic and political transition of the early 1990s and the breakdown of former social protection schemes.[48-50] Higher rates of mortality from conditions amenable to medical

intervention in lower educated groups in Eastern Europe[8 51] suggest that lack of access to effective health care also plays a role.

Limitations

For most of the western and northern European countries we had census-linked longitudinal data aggregated over a ten-year period in the 1990s. For the Czech Republic, Estonia and Poland, however, we had census-unlinked data gathered in a cross-sectional manner, aggregated for a few years around the year 2000. During the 1990s relative inequalities in mortality between educational groups increased throughout Europe,[48 52] especially in central and eastern European countries.[49 50] Thus the stronger educational gradient in life expectancy found for Eastern Europe may in part be due to the later time period pertaining to these data. It is more difficult to estimate the possible effects on estimates of lifespan variation, because it depends on the age-specific nature of these widening relative inequalities.

Census-unlinked mortality data are less reliable than census-linked longitudinal data, because of possible numerator/denominator biases. This bias, caused by differences between self-reported information on the census and information reported by next-of-kin on death certificates, could go either way.[53-55] A recent study on unlinked Lithuanian data found an overestimation of inequality,[56 57] but for other Eastern European countries the correspondence between the death and census records seemed better.[49] Thus while there might be some overestimation of the levels of inequality for these countries, we expect the bias to be smaller than that which was found in the Lithuanian studies.

In the analysis reported in this paper we used a readily understandable measure of lifespan variation, the standard deviation. Differences in lifespan variation tend to be driven by premature mortality, which skews the age distribution of death. This might call into question whether the standard deviation is an appropriate measure. In general, different measures of lifespan variation are highly correlated.[58] Nevertheless, we checked whether our findings would change if we would quantify lifespan variation by the Gini coefficient,[11] which compares differences in ages at death between individuals rather than to any average age at death. Moreover, the Gini is sensitive to changes in mortality at older ages than the standard deviation and measures relative rather than absolute differences in lifespan variation.[11] Applying the Gini coefficient produced only few differences in country ranking, and the substantive conclusions remained the same (results not shown).

Implications

Our analysis sheds new light on the frequently studied phenomenon of socioeconomic inequalities in mortality. We show that higher and lower educated groups not only differ in average life expectancy, but also in lifespan variation. Larger lifespan variation in lower socioeconomic groups has potential implications both for individuals and for society as a whole.

At the individual level, greater uncertainty in the timing of death makes long-term investments such as education, healthy behaviour, and retirement planning less sensible. This greater uncertainty could well be one of the determinants of the lower sense of control (or greater powerlessness and fatalism) among lower socioeconomic groups,[59 60] which has been shown to be one of the determinants of their higher rates of unhealthy behaviour.[61-63] Future research should examine whether individuals of lower socioeconomic status indeed perceive their higher lifetime uncertainty, and whether this contributes to a lower sense of control, and indirectly to riskier behaviour.

At the societal level, the existence of substantial inequalities in lifespan variation points to possible failures of social protection policies, particularly those that reduce premature mortality among lower socioeconomic groups. These policies should pay more attention to determinants of premature mortality, such as risky behaviour, hazardous housing and working conditions, psychosocial factors, and lack of access to effective health care. Countries where the low educated had a higher average life expectancy also tended to be the ones with less lifespan variation, both among the low educated and in the population as a whole. Sweden is a clear example. This lends support to the idea that universal social policies protect vulnerable groups by 'raising the floor'.[64] Yet the pattern is less clear for women than for men, and not all countries with universal social policies fare well on these accounts (e.g. Finland). Further research is needed to see whether or not universal social policies are indeed better in reducing lifespan variation than targeted social policies.

In most wealthy countries life expectancy at birth now stands at around 80 years. A reasonable question to ask is what our social preferences are for research and policies directed at increasing our average longevity versus reducing uncertainty in the timing of death. A strong risk aversion to early death would call for more attention to the variability in longevity. Social protection policies would then have to be designed specifically to address the needs of the most vulnerable individuals and social groups. Moreover as early deaths bring down the average, reducing mortality

at early ages would have the double benefit of reducing lifespan variation and increasing life expectancy.

By definition reducing lifespan variation requires that reductions in premature mortality continue at a higher pace than reductions in old age mortality. With finite budgets, targeting premature mortality would imply a degree of age rationing in health priorities. Given a choice, would individuals rather public spending be directed to equalizing life chances or to improving survival probabilities at the oldest ages? Ethical analysis and measuring preferences of the population on this matter would dictate to a large extent how trends in survivorship should be monitored, and where interventions should be prioritized.

Conclusions

Lower educated individuals not only have shorter life expectancies, but also face greater uncertainty about the age at which they will die. More priority should be given to efforts to reduce the risk of an early death among the lower educated, e.g. by strengthening protective policies within and outside the health care system.

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Appendix to Chapter 5

Country	Years	Study Type	Person-years of follow-up	Number of deaths	Missing education (%)
Sweden	1991-2000	Longitudinal, census-linked	48 340 986	919 508	9.8
Norway	1990-2000	Longitudinal, census-linked	22 262 277	433 282	2.3
Finland[1]	1900-2000	Longitudinal, census-linked	27 550 171	473 873	0
Belgium	1991-1995	Longitudinal, census-linked	27 635 206	486 222	6.0
Switzerland	1990-2000	Longitudinal, census-linked	30 728 441	538 619	0.6
France[2]	1990-1999	Longitudinal, census-linked	2 720 978	43 024	0
Slovenia	1991-2000	Longitudinal, census-linked	10 325 537	165 423	1.3
Czech Republic	1999-2003	Cross-sectional, unlinked	30 308 765	535 264	0
Poland	2001-2003	Cross-sectional, unlinked	65 844 117	1 058 745	2.0
Estonia	1998-2002	Cross-sectional, unlinked	4 141 440	60 794	2.3

Special remarks
[1] unknown education is classified as education group 1
[2] survey of national population, causes-of-death were not available

Table S1: Countries and study type included in the analysis

	Male			Female		
Country	Low	Med.	High	Low	Med.	High
Belgium	0.62	0.21	0.16	0.69	0.18	0.13
Czech Republic	0.62	0.25	0.13	0.64	0.29	0.08
Estonia	0.31	0.53	0.17	0.30	0.53	0.17
Finland[1]	0.51	0.28	0.21	0.56	0.26	0.18
France	0.53	0.35	0.12	0.66	0.24	0.09
Norway	0.33	0.47	0.21	0.41	0.44	0.15
Poland	0.61	0.28	0.11	0.54	0.35	0.11
Slovenia	0.39	0.49	0.12	0.59	0.32	0.08
Sweden	0.40	0.43	0.16	0.41	0.40	0.19
Switzerland	0.22	0.55	0.23	0.44	0.49	0.06

[1] unknown education is classified as education group 1

Table S2: Population proportions by educational group

CHAPTER 5

Data description

We used Eurothine data collected in two different formats: census-linked data which followed individuals for around 10 years over the 1990s and census-unlinked data which aggregated age-specific mortality rates over a few years. Comparing these two datasets may have introduced biases relating to the different time periods under study and biases from the data formats. Unfortunately neither dataset could be disaggregated by year. In Figure S1 we illustrate the two data formats on a Lexis surface. The light grey parallelogram refers to the census-linked data, which aggregated deaths and person years in a cohort manner (pictured are those aged 30-31 at the beginning of the study), over a period roughly from 1991 to 2000. The dark grey rectangle represents the census-unlinked studies, which aggregated individuals of each age, illustrated for ages 35-36, over a few years around the year 2000. For the pictured individuals, we assumed an average age-at-death of 35 in the linked studies (more precisely, the age at the start of the study plus half the length of the study) and 35.5 in the unlinked studies.

This might have led to some problems of comparability between the two study types. To get an idea of how this might have biased our results, we computed for Slovenia death densities of the national population over the two methods, longitudinal and cross-sectional from HMD data over the 1990s, plotted in Figure S2. While some differences occur around the modal age at death and at the most advanced ages, the two curves follow each other rather well, giving us confidence that the two study types are indeed comparable.

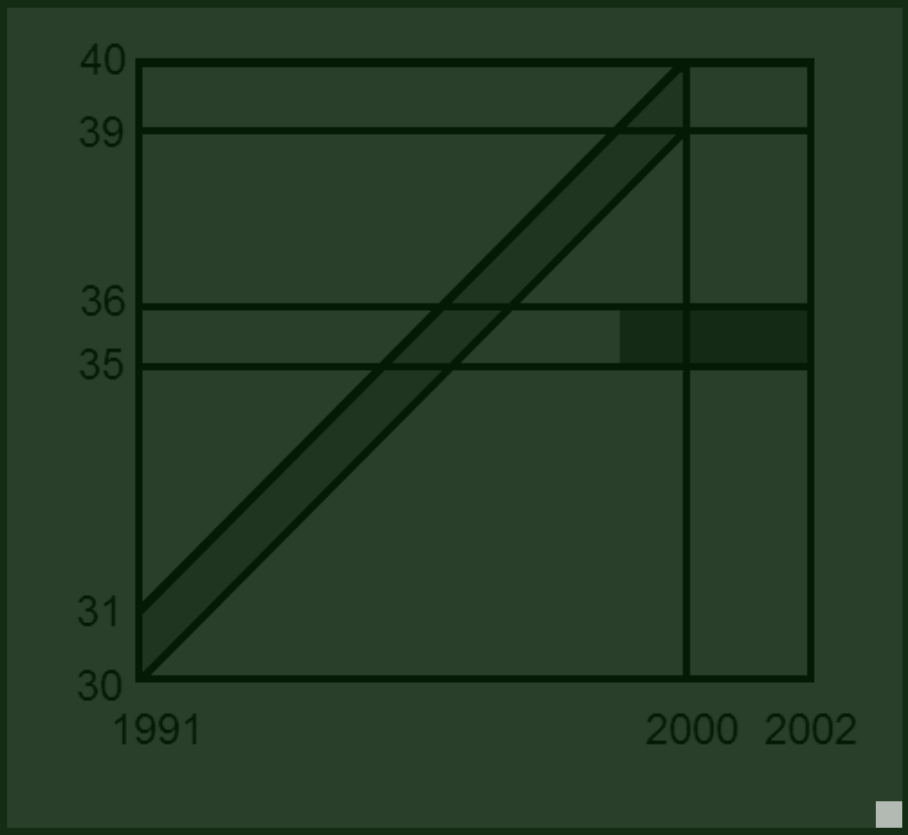

Figure S1: A Lexis diagram illustrating the data formats of the two study types, census-linked in red and census-unlinked in blue. In both cases these data were aggregated to represent the rates of individuals aged 35 to 36

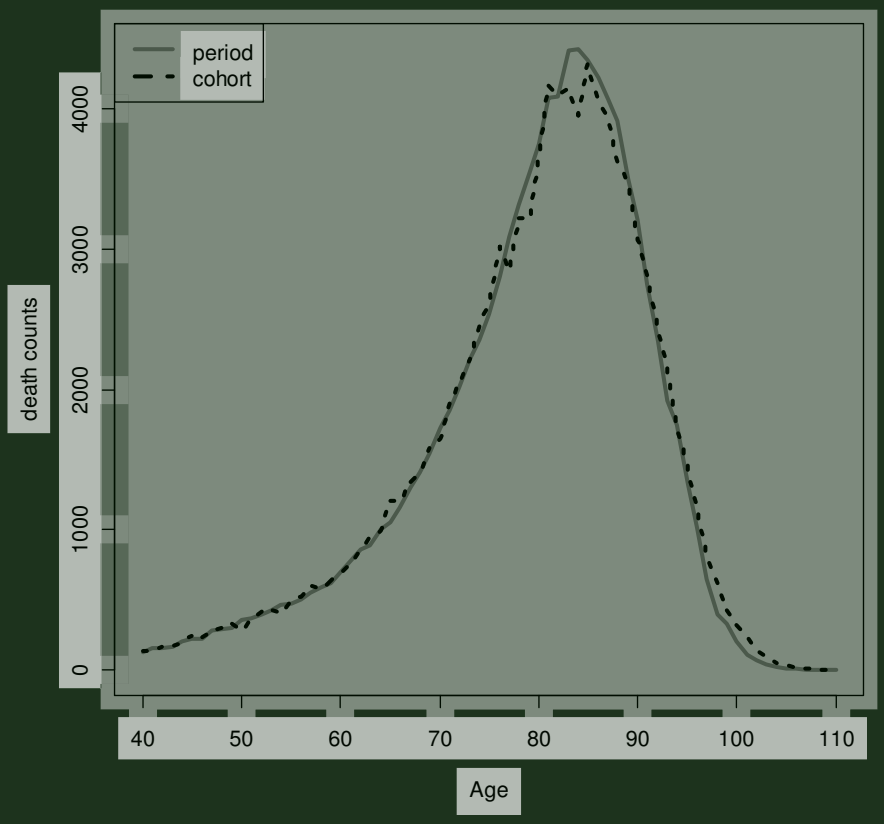

Figure S2: Death densities based on an aggregation of death counts and exposures aggregated over a cohort or period manner, Slovenia females, data from the Human Mortality Database.

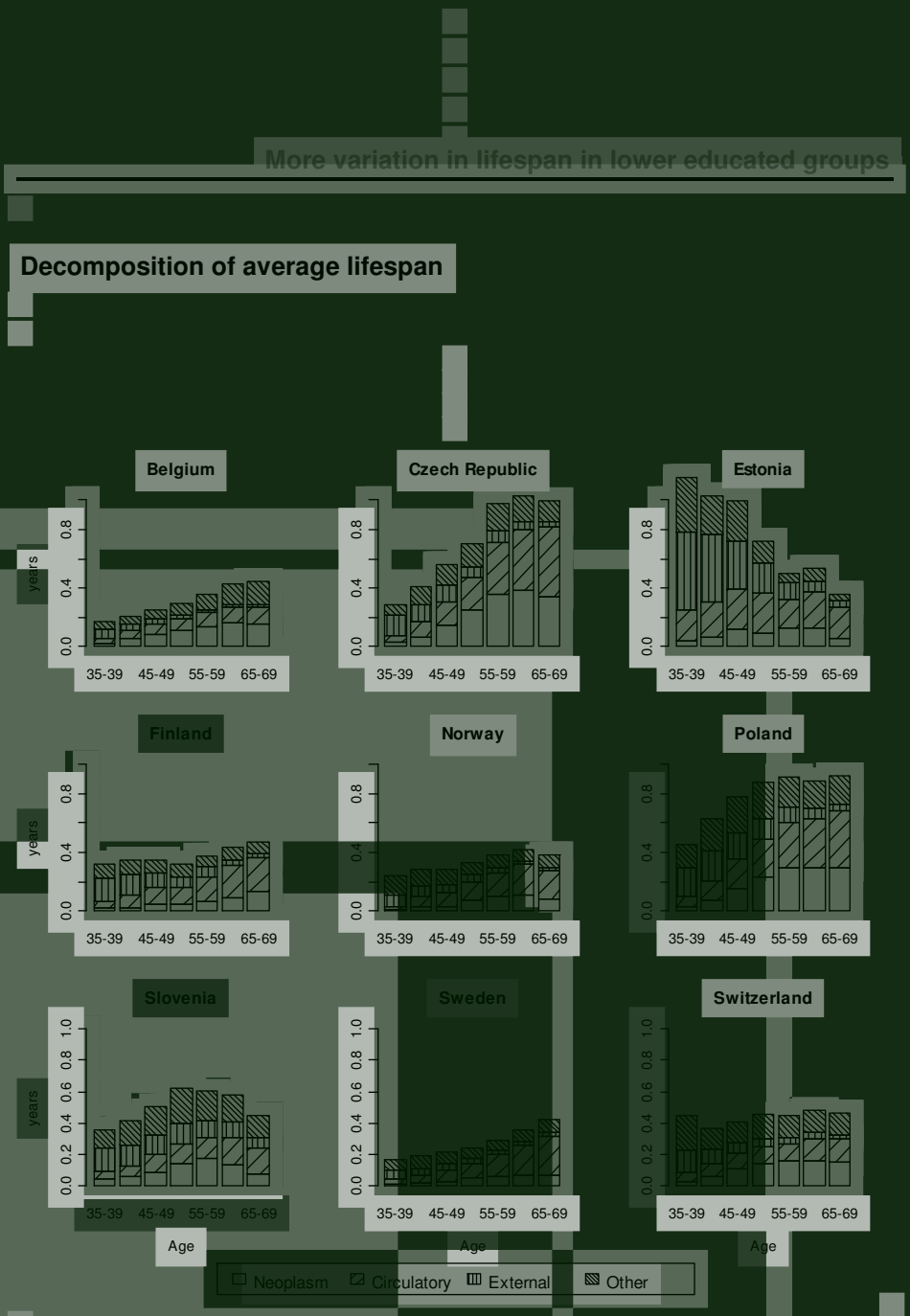

Figure S3: Male age and cause decomposition of life expectancy conditional upon survival to age 35 advantage of the medium and high educated groups combined compared to the low educated group. The contribution is in years, pertaining only to ages 35-70. Data is from the Eurothine project. The equivalent figure for females is presented as online figure 2.

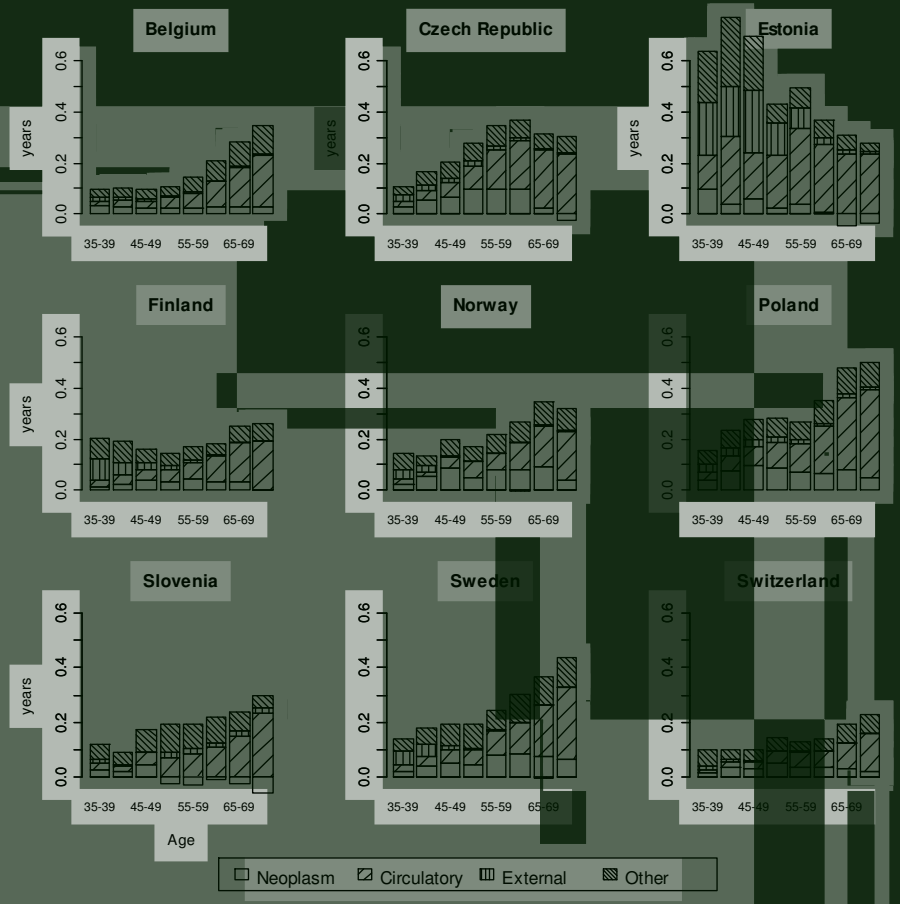

Figure S4: Female age and cause decomposition of life expectancy conditional upon survival to age 35 advantage of the medium and high educated groups combined compared to the low educated group. The contribution is in years, pertaining only to ages 35-75. Data is from the Eurothine project.

CHAPTER 6

The contribution of educational inequalities to variation in lifespan

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submitted

CHAPTER 6

ABSTRACT

Background: Studies of socioeconomic inequalities in mortality consistently point to higher death rates in lower socioeconomic groups. Yet how these between-group differences relate to the total variation in mortality risk between individuals is unknown.

Methods: We used data assembled and harmonised as part of the Eurothine project, which includes census-based mortality data from 11 European countries. We matched this to national data from the Human Mortality Database and constructed life tables by gender and educational level. We measured variation in age-at-death using Theil's entropy index, and decomposed this measure into its between- and within-group components.

Results: The lowest educated groups lived between 3 and 15 years less than the highest educated groups, the latter having a more similar age-at-death in all countries. Differences between educational groups contributed between 0.5 and 2.5 % to total variation in age-at-death between individuals in Western European countries, and between 2 and 10 % in Central and Eastern European countries. Variation in age-at-death is larger, and differs more between countries, among the lowest educated groups.

Conclusions: At the individual level, many known and unknown factors are causing enormous variation in age-at-death, socioeconomic position being only one of them. Reducing variations in age-at-death among lower educated people, by providing protection to the vulnerable, may help to reduce inequalities in mortality between socioeconomic groups.

Introduction

Individuals vary greatly in lifespan. For instance, comparing age-at-death of European males at the individual level to that of every other male in the same population, the average difference is around 7.5 to 10.5 years, depending on the country.[1-3] This variation in lifespan has many sources, including genetic, lifestyle factors, socioeconomic conditions, chance etc. One of these sources, differential mortality by socioeconomic group, has been the subject of much research. A recent European cross-country comparison revealed higher death rates in lower socioeconomic groups in all 16 populations studied, with particularly large socioeconomic differences in mortality in parts of Central and Eastern Europe.[4] What is unknown, however, is the contribution of these *between-group* differences to all *between-individual* differences.

This relates to the debate sparked by the release of the World Health Report 2000 about whether lifespan (or more broadly health) inequality should be measured over individuals or groups, with the report's authors coming out in favour of the former.[5-7] By quantifying the variation of health over all individuals in a population, they contended, a more comprehensive inquiry into the extent of health inequality could be made than by conventional methods which quantify health inequalities as differences between predefined social groups. The authors further criticised methods that exclusively compared group means, speculating that different socioeconomic groups might also have different degrees of within-group variation. Indeed preliminary evidence on Russia,[2] and the United States,[8] suggests that groups with lower socioeconomic status have higher dispersion in their lifespan distributions, in addition to their lower mean lifespans. Criticism of the report centred on whether individuals can replace groups as the unit of analysis. Critics feared that monitoring the full extent of between-individual variation in and of itself would not pinpoint areas requiring public health interventions.[9] Moreover, they noted that between-individual variation in health often correlates poorly with between-group socioeconomic inequalities in health,[10] and reasoned that it would remove equity and human rights considerations from the study of health inequalities.[11]

Although individual- and group-level approaches are indeed not interchangeable,[12] it is important to recognise that differences between individuals and differences between groups are not entirely independent of one another—between-group differences make up one component of total between-individual variation in a population. Analysing how the two are linked would serve to put between-group differences in health within a broader perspective. Lacking in the

WHO report, however, was a clear method of linking between-group differences to total variation in health. In this paper we apply a method commonly used in economic research, but as of yet not attempted in the health sciences, that allows a decomposition of all between-individual variation into two components. By adopting Theil's index total lifespan variation can be decomposed into a between- and a within-group component.[13] Using this method, we determine the contribution of differences in age-at-death between socioeconomic groups, in our case classified by education, to the total between-individual variation in age-at-death. We apply this method to 11 European countries with high quality data.

Data and Methods

Creating synthetic cohort death distributions by age, sex and education

We used census-based data assembled and harmonised as part of the Eurothine project.[14] This comprised sex-specific death counts and exposures by sex, age (aggregated into 5 year age intervals) and level of education. The data covers Sweden, Norway, Finland, France, Belgium, Switzerland, Slovenia, Czech Republic, Hungary, Estonia and Lithuania. For most countries we had longitudinal census-linked studies (individuals aged 30-85+ followed over 10 years between the 1990 and 2000 census rounds) while for Czech Republic, Hungary, Estonia and Lithuania we had cross-sectional unlinked studies (individuals aged 30-85+ pooled for a few years around the 2000 census year). Excluded subpopulations were Åland Island from Finland, non-Swiss nationals from Switzerland, and overseas departments, students, the military, and persons born outside of France from the French data. More information about the data is available in the appendix to Chapter 5.

Comparable educational levels had been created by regrouping national education schemes into four categories of the International System of Classification of Educations (ISCED): primary or no education; lower secondary education; higher secondary education; and tertiary education. For three of the countries studied (Norway, Finland, and Switzerland) the two lowest educational groups had to be combined in the Eurothine harmonization process either because the countries' educational system did not allow for proper differentiation between the two groups or because the proportion of subjects in the lowest educational category was too low to draw meaningful conclusions. The proportion of subjects in each educational category is shown in Table 1.

	Male				Female			
	elementary[1]	lower sec.	upper sec.	tertiary	elementary[1]	lower sec.	upper sec.	tertiary
Sweden	0.30	0.10	0.43	0.16	0.30	0.11	0.40	0.19
Norway	0.33		0.47	0.21	0.41		0.44	0.15
Finland	0.51		0.28	0.21	0.56		0.26	0.18
Belgium	0.44	0.18	0.21	0.16	0.53	0.16	0.18	0.13
Switzerland	0.22		0.55	0.23	0.44		0.49	0.06
France	0.47	0.06	0.35	0.12	0.57	0.09	0.24	0.09
Slovenia	0.20	0.19	0.49	0.12	0.24	0.35	0.32	0.08
Czech Rep.	0.12	0.50	0.25	0.13	0.30	0.34	0.29	0.08
Poland	0.26	0.35	0.28	0.11	0.35	0.19	0.35	0.11
Estonia	0.10	0.21	0.53	0.17	0.13	0.17	0.53	0.17
Lithuania	0.15	0.14	0.55	0.16	0.22	0.10	0.52	0.17

[1] the lowest two educational groups were combined in Norway, Finland and Switzerland

Table 1: Proportion of subjects in each of the following educational categories by country

Unfortunately, the longitudinal census-linked studies could not be disaggregated by year. Thus we started with age 35 because it corresponded to the average age of individuals aged 30 at the beginning of these studies who were followed for 10 years (see appendix to chapter 5). For the cross-sectional unlinked studies, we only used data above age 35. To improve the precision of the age-at-death distribution, the national population death and exposure counts reported by single year of age in the Human Mortality Database (HMD)[3] were proportioned out to each educational group, according to their corresponding shares derived from the Eurothine data for the equivalent time periods. The matching was done per country, sex and 5-year age group for ages 35 to 85+ (75+ in Sweden).

We made the assumption that in the open-aged category (75+ or 85+ years) mortality rate ratios between educational groups were the same as those observed in the oldest preceding age category. A previous study showed this to be the case for females, but to risk overestimating differences for males, who were shown to have decreasing rate ratios between educational groups up to ages 90+.[15] Finally, the small number of subjects surviving to oldest ages led to some random variation in the right tail of the death distributions. To smooth the distribution, we fitted the

Kannisto model of old age mortality to ages above 80, extrapolating death counts for both sexes beyond the first age with fewer than 100 male deaths.[16]

The result of this matching was sex-specific death rates by single year of age (35-110+) and educational level. We then used these death rates to construct male and female life tables for each educational subgroup, thus allowing comparable age distributions of deaths that were not confounded by the age structure of the educational subgroups of the real population.

Measuring and decomposing lifespan disparity

Determining the contribution of educational inequality to total variation in lifespan requires using a measure that is decomposable into its between-group (*BG*) and within-group (*WG*) components, such that total variation = *BG* + *WG*. The *BG* inequality component captures the variation in subgroup average lifespans, while the *WG* component captures the average individual-level variation calculated for each of the subgroups. The contribution of the stratifying variable (in our case education) to the total variation in lifespans then is simply the *BG* component divided by the total variation.

Only a few measures of variation are additively decomposable, and of this subset we decided to apply Theil's entropy index (*T*) because of its sensitivity to changes in mortality in the early part of the age at death distribution—deaths which we consider to be the most unjust. Theil's index was created from information theory to measures the degree of disorder in the distribution, and is widely used in studies of economic inequality.[13] It is a relative measure of variation, meaning that the level of variation would be unaffected by proportional gains to each individual's lifespan. The calculation and decomposition of this measure are presented in the appendix to this chapter. Theil's index takes on greater values with greater dispersion in lifespans. A value of 0 would indicate perfect equality (i.e. everyone died at the same age).

Although measures of lifespan variation are highly correlated,[17 18] they can arrive at different conclusions depending on their sensitivities to changes in mortality at different ends of the age distribution of death (Chapter 2). In particular *T* is sensitive to changes in the early part of the distribution, and becomes progressively less sensitive to changes at older ages. We therefore decided to also calculate the variance in age-at-death (*V*), which is known to be sensitive to changes in both early and late ages, given that it is calculated by squaring the distance to the mean. Moreover *V* is an absolute measure of variation that is unaffected by additive gains to each person's lifespan. As it is unclear whether gains to the lifespan distribution tend to occur in additive or proportional terms, we decided to examine

both. The calculation and decomposition of *V*, as well as the full results for this alternative measure are given in the chapter appendix.

Results

All countries in our study showed large educational differences in average age at death (Table 2). Differences tended to be smaller in Western Europe, where the highest educated women typically lived 3-5 years longer than the least educated women, and differences amounted to 5-7 years among men. In Central and Eastern European countries these educational differences in life expectancy were considerably larger. Men in the Czech Republic had the largest differences: 17 years between the highest and lowest educated groups. These larger differences owed to the substantially poorer performance of the lowest educated groups in Central and Eastern Europe. The tertiary educated lived to a much more similar age in all countries. Differences were always larger for males than for females.

Countries with large educational differences in life expectancy also tended to have higher overall levels of between-individual lifespan variation (Table 3). The differences again tended to follow regional patterns, with Western European countries having the lowest levels of lifespan variation, and some Central and Eastern European countries, particularly Estonia and Lithuania, the highest. Comparing Theil's Index of lifespan variation by educational group, we see that in all countries, the higher the level of education, the lower was the between-individual lifespan variation within the group. The differences between countries in between-individual lifespan variation were also largest among the lowest educated groups. In fact, the highest educated groups in all countries had similar levels of lifespan variation.

Differences between educational groups account for between 1.8 to 10.6 percent of total variation in age-at-death among men, while for females between-group differences account for 0.6 to 9.4 percent of total variation (Table 4). Similar results were obtained using the *V* measure (see appendix). Between-group differences explained more of the total variation in age-at-death in Central and Eastern Europe. This is particularly true for males in the Czech Republic, both because of the high between-group component and, as compared to other countries in its regional grouping, the low within-group component.

	Male					Female				
	elemen -tary[1]	lower sec.	upper sec.	tertiary	**Total**	elemen -tary[1]	lower sec.	upper sec.	tertiary	**Total**
Sweden	75.3	76.2	77.8	80.3	**77.1**	80.3	81.9	82.7	85.0	**82.3**
Norway		74.1	76.6	79.2	**76.1**		80.0	82.2	83.6	**81.5**
Finland		72.7	74.9	78.0	**74.1**		79.9	81.7	82.9	**80.7**
Belgium	73.5	75.3	76.6	78.2	**74.7**	79.9	81.9	82.3	82.8	**80.6**
Switzerland		74.1	77.3	79.8	**77.0**		81.7	83.4	84.4	**82.7**
France	73.4	76.6	77.1	80.4	**75.6**	81.7	83.7	84.4	84.8	**82.8**
Slovenia	69.0	70.4	73.4	77.4	**72.4**	77.7	78.8	80.6	82.3	**79.4**
Czech Rep.	63.7	73.9	77.3	80.8	**73.0**	77.7	79.3	81.9	83.9	**79.9**
Poland	67.9	69.3	76.2	79.7	**71.8**	78.1	77.1	82.2	83.9	**80.0**
Estonia	61.3	62.5	67.4	75.3	**67.2**	70.4	74.0	77.8	81.6	**76.9**
Lithuania	61.7	61.8	69.9	76.8	**68.6**	71.2	72.9	82.1	83.8	**79.1**
Range (years)	14.0	14.8	10.4	5.5	**9.9**	11.3	9.6	6.6	3.4	**5.9**

[1] the lowest two educational groups were combined in Norway, Finland and Switzerland

Table 2: Average age at death (conditional on survival to age 35) for each country, gender and educational group over the period of study; 'Total' refers to all educational groups combined

	Male					Female				
	elemen -tary[1]	lower sec.	upper sec.	tertiary	**Total**	elemen -tary[1]	lower sec.	upper sec.	tertiary	**Total**
Sweden	1.58	1.42	1.29	1.06	**1.37**	1.33	1.23	1.11	1.01	**1.19**
Norway		1.59	1.32	1.10	**1.39**		1.31	1.08	0.95	**1.16**
Finland		1.97	1.80	1.35	**1.83**		1.36	1.16	1.07	**1.27**
Belgium	1.68	1.51	1.45	1.28	**1.56**	1.30	1.21	1.17	1.11	**1.25**
Switzerland		1.78	1.37	1.15	**1.43**		1.17	1.03	1.01	**1.10**
France	2.01	1.66	1.66	1.30	**1.82**	1.31	1.21	1.06	0.95	**1.22**
Slovenia	2.09	1.87	1.61	1.29	**1.77**	1.40	1.25	1.20	1.08	**1.27**
Czech Rep.	2.27	1.80	1.43	1.12	**1.85**	1.37	1.32	1.11	0.82	**1.26**
Poland	2.70	1.89	1.66	1.31	**2.12**	1.60	1.33	1.21	0.94	**1.38**
Estonia	3.66	3.28	2.52	1.65	**2.81**	3.57	2.44	1.53	1.06	**1.93**
Lithuania	4.01	3.52	2.57	1.77	**3.02**	3.60	2.49	1.69	1.12	**2.24**
Range (years)	2.43	2.10	1.28	0.71	**1.65**	2.30	1.32	0.66	0.30	**1.14**

[1] the lowest two educational groups were combined in Norway, Finland and Switzerland

Table 3: Theil's index of lifespan inequality (x 100) by country, gender and educational subgroup; 'Total' refers to the male/female total population Theil's index.

	Variance		Within-group component		Between-group component		BG inequality as % of total	
	Male	Female	Male	Female	Male	Female	Male	Female
Sweden	1.37	1.19	1.35	1.17	0.02	0.02	1.76	1.65
Norway	1.39	1.16	1.36	1.15	0.03	0.01	2.07	1.20
Finland	1.83	1.27	1.78	1.26	0.04	0.01	2.41	1.09
Belgium	1.56	1.25	1.53	1.24	0.03	0.01	1.87	0.96
Switzerland	1.43	1.10	1.40	1.09	0.03	0.01	2.13	0.61
France	1.82	1.22	1.77	1.21	0.05	0.01	2.78	1.03
Slovenia	1.77	1.27	1.71	1.25	0.06	0.02	3.55	1.24
Czech Rep.	1.85	1.26	1.65	1.23	0.20	0.03	10.57	2.49
Poland	2.12	1.38	1.95	1.33	0.18	0.05	8.31	3.53
Estonia	2.81	1.93	2.6	1.83	0.21	0.10	7.43	4.95
Lithuania	3.02	2.24	2.75	2.03	0.27	0.21	8.89	9.37

Table 5: Decomposition of Theil's index of lifespan inequality into its between-group and within-group components by country and gender

Figure 1 visualizes the between-group and within-group differences in age-at-death for two sample countries, and illustrates that most of the total variation in age-at-death comes from within the groups. The male Czech population has the highest contribution of the between-group component. In comparison to the Belgian population the age-at-death distributions are more stratified, particularly between the lowest educated group and the others.

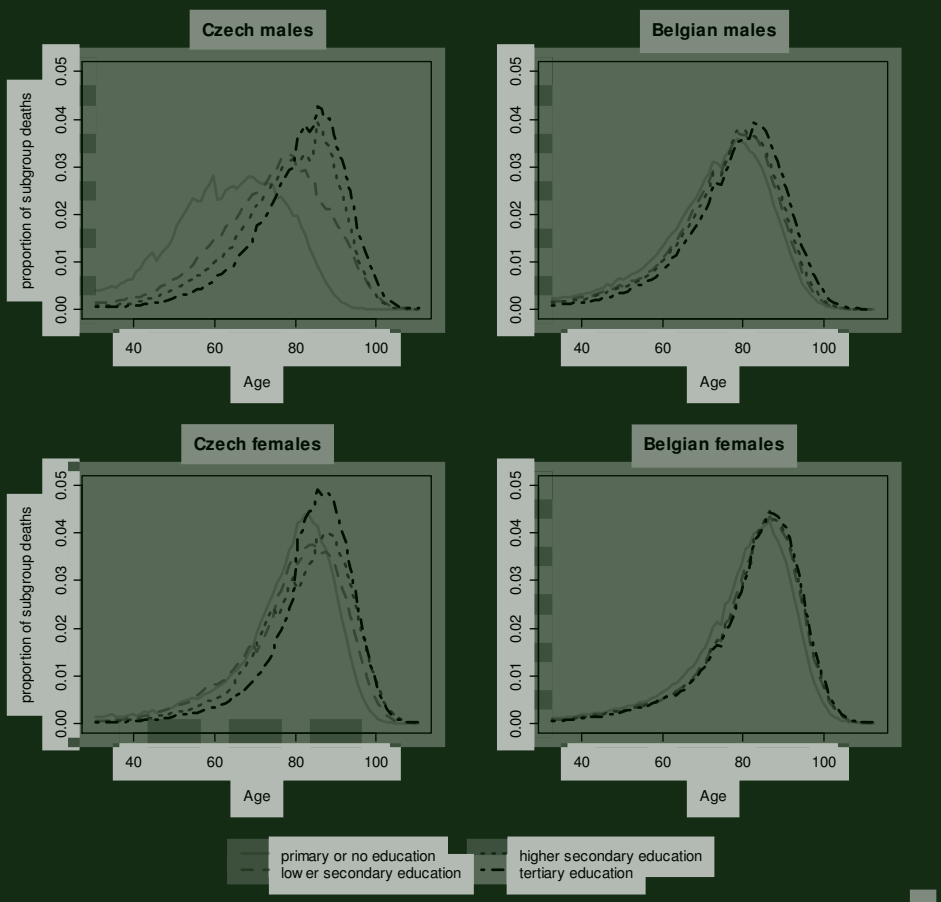

Figure 1: Age at death distributions for males and females in Belgian and the Czech Republic by level of education.

Discussion

Summary of results

Educational differences in age-at-death were substantial in all European countries, but contributed only a small fraction to the total individual lifespan variation: 0.6-2.8 percent in Western Europe, and 2-10 percent in Central and Eastern Europe. Lower educated groups not only had lower mean lifespans, but also had greater between-individual variation in lifespan. The gap in between-individual lifespan variation between Western Europe and Central and Eastern Europe was more evident among the lowest educated groups—the tertiary educated groups had more similar lifespan distributions in all countries.

Evaluation of data and methods

One concern is whether, given our limited number of subgroups, we are fully capturing the educational gradient in mortality. When possible we used four subgroups, but in some countries we were restricted to three subgroups, and in others (ex. Switzerland) the vast majority of the population fell into only two subgroups. This might have resulted in a lower than actual between-group component. To be sure that the different number of subgroups was not biasing our inter-country comparisons of the contribution of between-group inequality, we also ran the analysis for all countries with educational groups one and two combined. The reduction from four to three subgroups decreased the between-group component by 15 percent on average (results not shown). Using three subgroups altered the country rankings only slightly, with Slovenia and Norway trading places for females and Lithuania and the Czech Republic for males when it came to examining the overall contribution of between-group variation to the total variation in age-at-death. Although more subgroups would increase the BG component, so long as we are capturing most of the linear socioeconomic gradient in mortality, we do not expect this effect to be large. Even if the between-group component were to double, it would still only explain a small fraction of individual level lifespan variations.

Another concern is whether the nature of unlinked studies may introduce a numerator/denominator bias. Authorised informants may state a different educational status for the deceased than was recorded in the population census. If the deceased are reported as having a higher than attained educational level (“promoting

the dead") this would lead to overestimating mortality among the highest educated groups.[20] However, a record linkage study for Lithuania found that unlinked estimates overestimated mortality in lower educational groups and underestimated mortality in the best educated groups, particularly for females.[21] We were able to compare our unlinked estimates with these linked Lithuanian data[22] (see appendix). We found that the range in the average age-at-death between the highest and lowest educational groups was lower in the linked data by 23 percent for males and by 34 percent for females. This had the effect of substantially decreasing the between-group component. As a result, the contribution of between-group variations in age-at-death (using Theil's index) decreased from 8.3 to 5.0 percent for males and from 7.2 to 2.7 percent for females. While the overestimation is certainly substantial, the results from the linked data confirm a larger between-group contribution in Lithuania as compared to most Western European countries. Moreover we expect that this overestimation would be smaller in Czech Republic, Hungary and Estonia, because of better correspondence between educational categories on the census and death certificates.

Finally, there could be problems of comparability between countries given the different study years. The unlinked studies of Central and Eastern Europe take place around the year 2000, which is on average 5 years later than the longitudinal census-linked studies that followed subjects for the 10 year period between the 1990 and 2000 round of censuses. Alongside changing educational compositions in the population, during this period relative inequalities in mortality between educational groups increased throughout Europe.[23,24] Some studies found that the magnitude of this widening was even greater in Central and Eastern European countries.[25,26] Thus if we had had data for these countries for periods comparable to the longitudinal studies, we might have found smaller differences in the between-group inequality component between Eastern and Western European countries.

Taking these limitations together, we can reasonably conclude that educational inequalities explain a small portion of lifespan variation. The high quality of the longitudinal census-linked data gives us confidence in these results and rankings. We assume the between-group contribution to be higher in Hungary, the Czech Republic, Estonia and Lithuania than in Western Europe but caution that data concerns are likely to have overestimated the figures presented in Table 4.

Comparisons to other studies

To the best of our knowledge, we are the first to decompose individual-level variation in age-at-death into its between- and within-group components using Theil's index. The contribution of the between-group component that we observed is similar to American estimates made by Tuljapurkar,[27] calculated by approximating the variance (*V*) decomposition that we presented in Appendix B. Morbidity researchers have decomposed the Gini coefficient or the related Health Concentration Index to determine the degree to which subgroup variation in age-standardised levels of health could be explained by socioeconomic status, a different but related question.[28-30] In these studies they found a much higher contribution from the socioeconomic component than we did. Yet it is difficult to make a direct comparison here: the distribution of age-standardised levels of health, where many individuals self-report perfect health, differs considerably from the distribution of ages-at-death.

Interpretation

Should a 1-10 percent contribution from between-group differences to the total between-individual variation in age-at-death be considered a large or a small amount? It is important to recognize that between-individual variation arises from many different sources, including genetic, behavioural factors, environmental conditions and chance. These factors may in part be associated with educational level and thus vary between educational groups, but there is likely to be even more variation on many of these factors within educational groups.

We are not the first to point out that between-group differences in life expectancy account for little of the total between-individual variation. Doblhammer [31] found that a lifespan difference of nearly half a year by month of birth explained just over one-hundredth of a percent of the total variation in age-at-death. In an additional analysis, we applied Theil's decomposition method to calculate the contribution of between-sex differences to total variation in age-at-death, using data from all countries of the Human Mortality Database for the year 2005. We found that the between-group component explained between 1.6 percent (England and Wales) and 9.9 percent (Russia) of total lifespan variation (results not shown). It would be interesting to run this type of analysis for risk factors such as smoking. We expect a relatively small contribution from the smoking-related between-group

component despite a ten year difference in life expectancy between smokers and non-smokers.[32]

Hence it is not that between-group educational differences in mortality are small, it is more that the magnitude of all inter-individual lifespan variation is tremendous. Even the large 5-year advantage in life expectancy held by the highest educated Belgian males over their lowest educated counterparts acted mostly to shift the whole death distribution to higher ages (Figure 1). It did not alter the shape of the two distributions, which remained largely overlapped, owing to the much greater within-group variation.

In addition to putting inequalities in mortality between socioeconomic groups within a broader perspective, our analysis leads to some new insights into the nature of these inequalities. Educational subgroups differ not only in their mean length of life but also in the spread around that mean: the lower life expectancy of lower educated groups concurs with a much greater variation in age-at-death as compared to higher educated groups. Also, the larger educational inequalities in mortality in some Central and Eastern European countries can be seen to arise from the larger between-individual variation in age-at-death within their lower educated groups. This suggests that reduction of socioeconomic inequalities in mortality might primarily require a reduction of variability in age-at-death. This may require better protection of people with higher vulnerability, e.g. because of smaller personal resources or less favourable living conditions. The results of our analysis support the idea that a main function of modern welfare states is to provide such protection against the vicissitudes of life.[33]

Implications

Returning to the debate introduced in the introduction of this paper, it seems that individual-level variations and group-level inequalities should not be seen as competing perspectives, but as interrelated phenomena. The one is embedded in the other. Our analysis illustrates the suggestion by Gakidou et al.[34] that within-group differences are themselves interesting and substantial, and a necessary complement to research into between-group inequalities. But simply measuring the sum of between-group and within-group differences, which was proposed by the WHO report as an alternative measure of health inequalities, cannot replace a specific focus on measuring inequality along socioeconomic lines or any other grouping of interest such as gender, ethnicity, regions, or life style.

Although socioeconomic differences in mortality are but one of many factors determining when individuals die, they are often seen to be among the most important and inequitable. This is because socioeconomic inequalities are at least partly avoidable, and because they follow from inequalities in the distribution of socioeconomic resources which themselves are often seen to be unjust.[35] Even if they contribute only a small fraction of all between-individual variations in lifespan, they are a legitimate concern for public health. What this study adds is that tackling inequalities in mortality between socioeconomic groups can also be approached through reducing variation in age-at-death among lower educated people, by providing protection to the vulnerable.

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CHAPTER 6

Appendix to Chapter 6

Part A: Calculating and decomposing inequality

Theil's index of inequality is a widely used measure for subgroup decomposition in the economics literature because of its property of being invariant to proportional changes in the distribution. Clearly this is a necessary precondition for cross-country comparisons of income inequality given large time and place fluctuations in currency. Lifespans, on the other hand, are almost exclusively expressed in years. The question of whether to use an absolute or a relative measure thus resides on whether the index should be sensitive according to proportional or additive changes in life expectancies. While it remains unclear which is the more important dimension to examine, we also calculate and decompose the variance in age-at-death, an absolute measure of dispersion.

Although precise calculation of both Theil's index (T) and the variance (V) require numeric integration of the survival curve, T has been reasonably estimated from single year life tables according to,[2]

$$T = \frac{1}{l_\theta}\sum_{x=\theta}^{\omega} d_x \left[\left(\frac{\bar{x}_x}{e_\theta}\right)\ln\left(\frac{\bar{x}_x}{e_\theta}\right)\right], \qquad (1)$$

while the V was estimated as,

$$V = \frac{1}{l_\theta}\sum_{x=\theta}^{\omega} d_x (\bar{x}_x - e_\theta)^2, \qquad (2)$$

where θ and ω are respectively the youngest and oldest age intervals taken from the life table, l_θ is the radix of the population (taken to be the initial subgroup population size), e_θ is the average age at death of the population, and d_x and $\bar{x}_x$ are respectively the life table number of deaths and the average age at death in the age interval x to $x+1$. The male and female all education groups combined populations were created by summing the life table deaths of all educational groups for each age interval. We calculated the lifespan measures conditional upon survival to age 35 as opposed to the measures at age 35. So in this case, rather than using the remaining life expectancy at age 35 (e_{35}), we used the average age at death conditional upon survival to age 35 ($e_{35} + 35$).

The indices were calculated for the male and female populations, then decomposed into their between- and within-group components. Calculating between-group inequality was be done by assuming that everyone in subgroup *i* had that group's mean age-at-death weighted by the subgroup's population share (w^i).

$$BG^T = \sum_{i=1}^{n}\left[w^i \left(\frac{e_\theta^i}{e_\theta^t}\right) \ln\left(\frac{e_\theta^i}{e_\theta^t}\right)\right] \quad (3)$$

$$BG^V = \frac{1}{l_\theta}\sum_{i=1}^{n} l_\theta^i (e_\theta^i - e_\theta^t)^2 \quad (4)$$

In this case n is the number of subgroups, e_θ^i refers to the average age-at-death conditional upon survival to age 35 for subgroup *i*, and e_θ^t is this average age for all education groups combined. Within-group inequality is a weighted average of the inequality levels present within each subgroup calculated by,

$$WG^T = \sum_{i=1}^{n}\left[w^i T^i \left(\frac{e_\theta^i}{e_\theta^t}\right)\right] \quad (5)$$

$$WG^V = \sum_{i=1}^{n}\left[w^i V^i\right] \quad (6)$$

where T^i and V^i are respectively the subgroup *i* Theil's index of inequality and variance in lifespan.

Part B: Decomposition of the variance measure of lifespan inequality into its between-group and within-group components by country and gender

	Variance		Within-group component		Between-group component		BG inequality as % of total	
	Male	Female	Male	Female	Male	Female	Male	Female
Sweden	154.3	149.9	151.4	147.2	2.9	2.6	1.88	1.77
Norway	152.8	144.4	149.5	142.5	3.3	1.9	2.19	1.28
Finland	191.2	155.1	186.3	153.3	4.9	1.8	2.56	1.17
Belgium	164.2	151.2	160.9	149.6	3.3	1.6	2.00	1.03
Switzerland	160.7	139.9	157.1	138.9	3.6	0.9	2.26	0.65
France	196.1	154.6	190.2	152.9	5.8	1.7	2.98	1.11
Slovenia	177.1	149.7	170.4	147.7	6.6	2.0	3.74	1.33
Czech Rep.	193.5	150.2	172.4	146.2	21.1	4.0	10.91	2.68
Poland	208.6	163.8	190.1	157.6	18.5	6.2	8.87	3.81
Estonia	241.5	208.4	222.4	197.2	19.0	11.2	7.89	5.37
Lithuania	269.4	255.7	244.2	229.8	25.2	25.9	9.34	10.12

Table S1: The country rankings were exactly the same in the contribution of *BG* inequality for the two inequality measures (*T* and *V*), albeit this contribution was higher for the *V* measure.

Part C: Comparing linked and unlinked datasets for Lithuania

	Primary & lower sec.		Higher sec.		Tertiary		Total Pop.		Between-Group		Within-Group		BG/T (%)	
	M	F	M	F	M	F	M	F	M	F	M	F	M	F
Average age at death conditional upon survival to age 30														
Unlinked	62.2	73.4	69.9	82.1	76.8	83.8	68.7	79.6	..	..	..	..	..	..
Linked	64.1	75.4	69.3	79.0	75.3	82.3	68.8	78.5	..	..	..	..	..	..
Theil's index x 100														
Unlinked	3.71	2.79	2.57	1.69	1.77	1.12	2.98	2.06	0.25	0.15	2.74	1.91	8.31	7.20
Linked	3.19	2.25	2.61	1.39	1.86	1.14	2.77	1.64	0.14	0.04	2.63	1.59	4.99	2.68
Variance														
Unlinked	281	253	238	210	196	145	268	239	23	19	244	220	8.74	7.76
Linked	276	236	237	160	197	142	249	185	13	5	235	179	5.28	2.92

Table S2: Comparison of linked and unlinked datasets for Lithuania; M refers to males and F to females, BG/T is the contribution of the between-group component to individual variation. The census-linked data cover the period 01.07.2001 – 31.12.2004, with the census having taken place on 06.04.2001. Details of the linkage procedure are described in Shkolnikov et al.[21] The unlinked dataset (with education groups one and two combined for better comparison) cover the period 2000–2002.

CHAPTER 7

The changing importance of educational inequalities to lifespan variation: Estonia and Lithuania examined over the 1990s

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submitted

CHAPTER 7

ABSTRACT

Background: By the end of the 1990s, educational between-group inequality was explaining a higher proportion of lifespan variation in Estonia and Lithuania than in most other European countries. It is unclear whether this was also the case prior to the mortality shocks of the 1990s.

Methods: We constructed life tables by sex, education and period (around 1990 and around 2000) for the two countries using census-based mortality data. Lifespan variation between the ages of 30 and 70 was estimated by the mean logarithmic deviation and decomposed into its between- and within-group components. We further decomposed changes in both components into contributions from direct and compositional changes to the educational subgroups.

Results: This analysis showed that temporary lifespan variation between the ages of 30 and 70 was higher at the end of the 1990s than it was in the beginning, and changes were proportionally greater for females. Increases in lifespan variation among lower and middle educated groups fuelled the overall increase in lifespan variation, although this was to some degree tempered, particularly in Lithuania, by a rising educational composition. The between-group inequality component rose and explained a greater part of the lifespan variation in the later period.

Conclusions: Lower educated groups suffered tremendously from the transitional shocks of the 1990s. Not only did their average lifespan fall, but they faced greater uncertainty in the timing of death. Diverging mortality experiences by educational subgroup led to greater stratification in the lifespan distributions, as well as increased inequalities in temporary life expectancy. The upward shift in the educational composition may be leading to the lower educated groups becoming more selected and vulnerable.

Introduction

Subpopulation mortality distributions can differ from one another because of differences in average lifespans (between-group inequalities) as well as differences in how lifespans are distributed within the group (within-group variation). In a previous study of 11 European countries (Chapter 6) we estimated that between educational-group inequalities explained about 0.6-10.9 percent of all individual level lifespan variation (ages 35-110+), depending on the measure, country and gender.

Of these countries Lithuania and Estonia had the largest lifespan variation and had among the highest contributions from the between-group component. Moreover, both countries experienced widening mortality trajectories by education during the 1990s—death rates continued to fall among the highly educated but actually worsened among those with lower levels of education.[1] Had it not been for an upward shift in the educational composition, remaining life expectancy in Estonia would have fallen an additional 0.96 years during this period.[2] As Lithuania also experienced substantial compositional changes during this period it is fair to assume that a similar study would probably have yielded similar results. Additionally, a quick calculation reveals that both countries also experienced widening lifespan variation during the 1990s.[i]

This leads to the question of whether the impact of socioeconomic variables on lifespan variation increased during this time. In this study we aim to determine how widening mortality differentials by level of education changed both the between- and within-group components of lifespan variation, using the mean logarithmic deviation and its associated additive decomposition. Additionally, to separately analyze direct versus compositional changes brought about by increasing education levels, we apply the dynamic decomposition technique of Mookherjee and Shorrocks[3] for the first time to the study of lifespan variation.

[i] Author's calculations based on period life tables of the Human Mortality Database, ages 0-110+, 1990-1999. This can be seen for both countries and genders. Lifespan variation was measured by the standard deviation in lifespans and the mean logarithmic deviation.

CHAPTER 7

Data and Methods

We used cross-sectional, census-based data assembled and harmonised as part of the Eurothine project. Both countries had two data files covering a few years surrounding two different census rounds: 1988-1990 and 2000-2002 for Lithuania, and 1987-1991 and 1998-2002 for Estonia. These contained sex-specific death counts and exposures by level of education, aggregated into 5-year age intervals from ages 30 to 85+ (70+ for the earlier period in Lithuania). Comparable educational levels had been created by regrouping national education schemes into three categories of the International System of Classification of Educations (ISCED): less than secondary education, completed secondary education, and tertiary education.

To improve the precision of the age-at-death distribution, the national population death and exposure counts reported by single year of age in the Human Mortality Database[4] (HMD) were proportioned out to each educational group, according to their corresponding shares derived from the Eurothine data for the equivalent time periods. The matching was done per 5-year age group for ages 30 to 69. Relative mortality risks were assumed to remain constant within the 5-year age categories. Ages above 70 were left in an open-ended interval to reduce known data problems at these ages.[5] Resulting from this matching were national death rates by single year of age (30-69, plus 70+), sex, and level of education (1-3) for each of the two periods.

Using these death rates, life tables were created for each sex and level of education, corresponding to each country and period. This allowed us to calculate temporary lifespan variation between the ages 30 and 70 ($e_{30|70}$) from the life table death density without confounding from the age structures of the educational subgroups.

Lifespan variation was measured using the mean logarithmic deviation (*MLD*) developed by Theil.[6] This is an entropy measure commonly used in economic research, shown in Chapter 2 to correlate closely with other inequality measures, albeit with higher sensitivity to changes in the left tail of the distribution. The primary advantages to this measure are that it is both additively decomposable such that total inequality is the sum of its between-group (*BG*) and within-group (*WG*) components, and it is decomposable over time into direct and compositional changes operating on both components.

From the life table death density the *MLD* of lifespan variation is calculated by:

$$MLD = \frac{1}{\ell_\theta} \sum_{x=\theta}^{\omega} d_x \left[\ln\left(\frac{e^0}{\bar{x}_x} \right) \right] \quad (1)$$

where d_x is the death density and $\bar{x}_x$ the average age at death in the age interval x to x+1. The initial population size is ℓ_θ and e^0 is the average lifespan, taken in this study to be the temporary life expectancy between ages 30 and 70 ($e_{30|70}$). The product is summed from initial age θ (30 in this study) to the oldest age ω (70+ in this study). The greater is the value of the index, the greater the variation in age-at-death. To ensure consistency between the educational subgroup populations and the aggregated national population, (temporary) life expectancy was calculated according to an alternative formulation of remaining life expectancy,

$$e^0 = \frac{1}{\ell_\theta} \sum_{x=\theta}^{\omega} d_x \bar{x}_x \quad (2)$$

Theil's additive decomposition of *MLD* as applied to lifespan variation becomes:

$$MLD = \sum_{i=1}^{n} w_i \ln \frac{e^0}{e_i^0} + \sum_{i=1}^{n} w_i MLD_i \quad (3)$$

where w_i is the population share and e_i^0 the lifespan of subgroup *i.* The first term of equation 3 measures the *BG* component by assuming that everyone in each subgroup dies at the subgroup's average age-at-death. In other words, it calculates the variation in subgroup average lifespans. The second term measures the *WG* component, and is the population-weighted average of the lifespan variation. The contribution of the *BG* component to total lifespan variation is simply the *BG* component divided by the *MLD* in lifespans of the whole population.

Age decomposition of $e_{30|70}$ and $MLD_{30|70}$ was done using step-wise decomposition[7] by modifying a VBA-program developed by Shkolnikov and Andreev.[8] Decomposing the change in $MLD_{30|70}$ over the two time periods into direct and compositional changes in the *BG* and *WG* components was performed using the dynamic decomposition of Mookherjee and Shorrocks.[3] [ii] In demography, this

[ii] This refers to their equations 14 *a* through *d*, which is an approximate decomposition. The approximation is widely used in the economics literature and found to be good. They also give an exact decomposition (eq. 13 substituted into 12). Due to the additional terms we found this equation more difficult to interpret.

decomposition analysis technique has been used to decompose whether inequalities in world life expectancy changed because life expectancies changed at different rates across countries or because populations grew faster in countries with unusually low or high life expectancies.[9] To our knowledge it has never been applied to examine changes in the death density. The decomposition results in four terms that can be interpreted respectively as the change in $MLD_{30|70}$ owing to:

1. the change in the *WG* component from changing death rates
2. the change in the *WG* component from the upward shift in the educational composition
3. the change in the *BG* component from the upward shift in the educational composition
4. the impact of *relative* changes in subgroup temporary life expectancies (i.e., if all subgroups experienced the same proportional increase in $e_{30|70}$, this term would be zero).

Given the small size of the population subgroups we also produced 95% confidence intervals around our estimates of temporary life expectancy and temporary lifespan variation. This was done through Monte Carlo simulation, assuming a binomial distribution of death counts. For each age interval the number of observations in each simulation round was based on the observed number of deaths, *Dx*, divided by the probability of dying, *qx*. The simulated death counts, dx^{sim}, divided by observed population exposures, *Nx*, gave us simulated death probabilities qx^{sim}. From these values we simulated 1000 life tables which we used to generate confidence intervals around our life expectancy and lifespan variation estimates. Similar methods have been applied to generate confidence intervals around life expectancy and healthy life expectancy for small populations.[10-13]

Results

Temporary life expectancy was lower for both males and females in Lithuania and Estonia at the end of the 1990s than it was in the beginning (Table 1). Alongside decreased longevity, temporary lifespan variation clearly increased in Estonia and stagnated in Lithuania (Table 2). Age decomposition of the changes to $e_{30|70}$ and $MLD_{30|70}$ illustrate the age contribution of these changes (Figure 1). The lower $e_{30|70}$

Males

	lower ed.	upper sec.	tertiary	Total
Estonia 1990*	31.9 *(31.7 32.1)*	34.1 *(34.0 34.3)*	36.1 *(35.9, 36.2)*	33.4
Estonia 2000†	29.0 *(28.7,29.3)*	32.6 *(32.5, 32.7)*	36.7 *(36.5, 36.8)*	32.2
Lithuania 1990*	31.8 *(31.6, 31.9)*	34.2 *(34.1, 34.3)*	36.6 *(36.4, 36.7)*	33.3
Lithuania 2000†	28.4 *(28.2, 28.7)*	33.6 *(33.5, 33.7)*	36.8 *(36.7, 37.0)*	32.6

Females

	lower ed.	upper sec.	tertiary	Total
Estonia 1990*	36.8 *(36.7, 37.0)*	37.6 *(37.5, 37.6)*	38.2 *(38.1, 38.3)*	**37.3**
Estonia 2000†	34.8 *(34.5, 35.1)*	37.3 *(37.3, 37.4)*	38.6 *(38.5, 38.7)*	**36.8**
Lithuania 1990*	36.5 *(36.4, 36.7)*	37.7 *(37.7, 37.8)*	38.2 *(38.1, 38.3)*	**37.2**
Lithuania 2000†	34.4 *(34.1, 34.7)*	37.8 *(37.7, 37.8)*	38.7 *(38.6,38.7)*	**36.8**

*the 1990 period corresponds to 1987-1991 in Estonia and 1988-1990 in Lithuania
†the 2000 period corresponds to 1998-2002 in Estonia and 2000-2002 in Lithuania

Table 1: Temporary life expectancy between the ages of 30 and 70 by country, gender and educational subgroup; 'Total' refers to all subgroups combined. Numbers in italics refer to 95 % confidence intervals based on Monte Carlo simulation.

Males	lower ed.	upper sec.	tertiary	Total
Estonia 1990*	12.2 *(11.5, 12.8)*	7.2 *(7.0, 7.5)*	4.5 *(4.1, 4.9)*	9.2
Estonia 2000†	16.2 *(15.2, 17.3)*	9.2 *(8.9, 9.5)*	3.7 *(3.3, 4.1)*	10.7
Lithuania 1990*	14.7 *(13.9, 15.5)*	7.8 *(7.6, 8.0)*	3.8 *(3.5, 4.1)*	10.8
Lithuania 2000†	17.4 *(16.6, 18.3)*	8.5 *(8.3, 8.8)*	3.8 *(3.4, 4.1)*	10.9

Females	lower ed.	upper sec.	tertiary	Total
Estonia 1990*	4.7 *(4.2, 5.3)*	2.7 *(2.5, 2.8)*	1.8 (1.6, 2.0)	3.5
Estonia 2000†	9.0 *(7.8, 10.2)*	3.2 *(3.0, 3.4)*	1.8 (1.5, 2.0)	4.7
Lithuania 1990*	6.5 *(5.7, 7.4)*	2.8 *(2.7, 3.0)*	2.1 (1.9, 2.3)	4.7
Lithuania 2000†	9.4 *(8.5, 10.3)*	2.9 *(2.8, 3.0)*	1.5 (1.3, 1.7)	4.8

*the 1990 period corresponds to 1987-1991 in Estonia and 1988-1990 in Lithuania
†the 2000 period corresponds to 1998-2002 in Estonia and 2000-2002 in Lithuania

Table 2: The temporary mean logarithmic deviation of lifespan variation between the ages of 30 and 70 (x 100) by country, gender and educational subgroup; 'Total' refers to all subgroups combined. Numbers in italics refer to 95 % confidence intervals based on Monte Carlo simulation

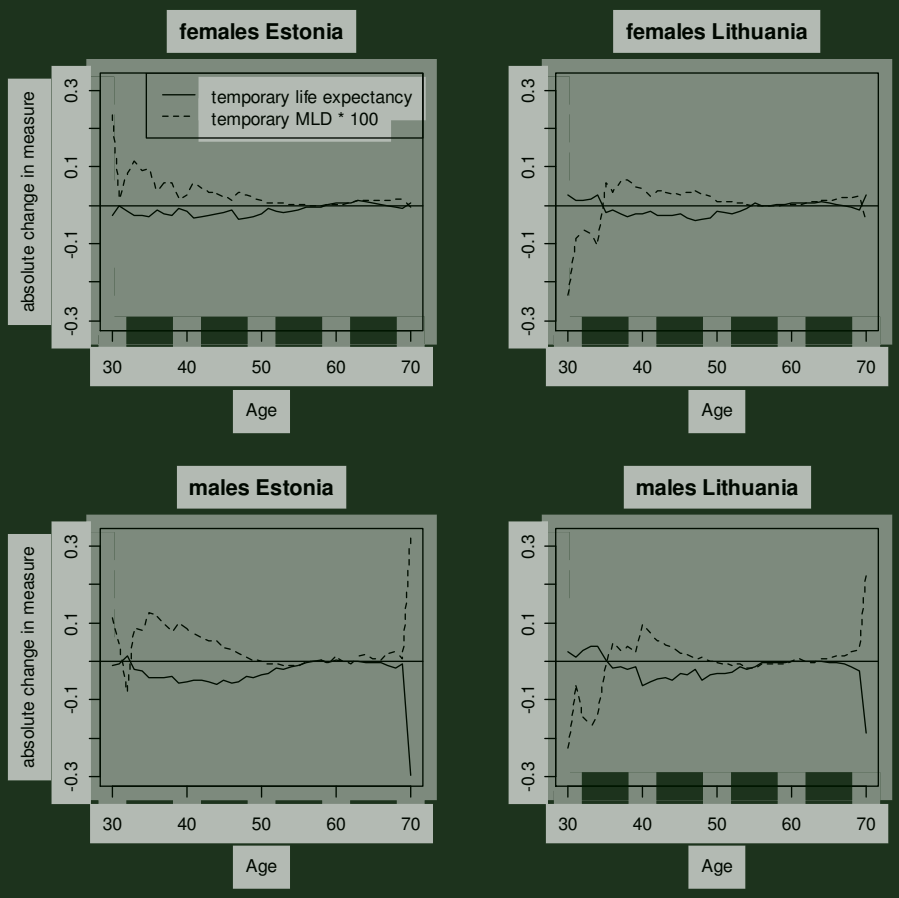

Figure 1: Age decomposition of changes to $e_{30|70}$ and $MLD_{30|70}$ *100 between the two periods

was mainly attributable to ages 35 to 55 for women, while the 70+ age category was additionally a large contributor for men. That Estonia experienced increased $MLD_{30|70}$ while Lithuania showed little change in the measure is explained primarily by the 30-40 age category. Lithuanians had lower death rates over these ages in the later period, which reduces lifespan variation, while Estonia had increased death rates. Proportionally, increases in $MLD_{30|70}$ were higher among females, although Estonian males experienced the largest absolute expansion in this measure.

Educational subgroups, however, fared rather differently from the national populations. As has been remarked upon elsewhere,[1 14] the tertiary educated improved their lifespans, while temporary life expectancy was actually lower for the less than secondary educated group in the second period than in the first. The female secondary educated groups experienced stagnation while the males had reduced longevity in the latter period. Differences by educational subgroup in lifespan variation showed similar patterns to trends in temporary life expectancy with the lowest educated evidently responsible for much of the rise in the $MLD_{30|70}$, and the tertiary educated experiencing mainly lower lifespan variation in the latter period.

As we would have expected from widening inequalities in temporary life expectancies, the *BG* inequality component increased substantially (Table 3). Proportionally increases were higher among females while males had larger absolute increases. The *WG* component, measuring the average subgroup lifespan dispersion levels, also rose for both female populations and for male Estonians. Despite increases in lifespan variation among both the less than secondary and upper secondary educated groups (Table 2), the male Lithuanians registered a decrease in their within-group component over the 1990s (Table 3). Thus the slight rise in lifespan variation in the total male population came entirely from the rise in the *BG* component.

In all cases, the contribution of *BG* inequality to total lifespan variation rose. In the early period females had levels comparable to *BG* contributions in western European countries in the middle of the 1990s (results not shown). However by the second period, the variation in temporary life expectancy among the educational subgroups was explaining 5-6 times more of the total lifespan variation over these age ranges. Male increases in the contribution of *BG* inequality were proportionally smaller.

Meanwhile, during this period both countries experienced upward shifts in their educational composition (Figure 2). Among the entire population, the proportion having at a minimum higher secondary education rose from 55 to 70 percent in Estonia and from 49 to 69 percent in Lithuania.

Changing importance of educational inequalities to lifespan variation

Turning to the results from the dynamic decomposition (Figure 3), it becomes apparent that direct changes in the *WG* component fuelled the increase in lifespan variation in Estonia. Had the educational composition in the later period been the same as in the earlier period, *WG* variation would have increased by over 50 percent for females in Estonia, and by about a quarter for males due to increased mortality at early adult ages (increasing the left tail of the distribution) and lower mortality at older ages. The rise in the educational composition of the population of course tempered these rises. In Lithuania, direct and compositional changes practically cancelled themselves out. This explains why Lithuanians experienced little change in the *WG* component, as fewer individuals were a part of the lower educated subgroup that experienced rising levels of lifespan variation. Changes in relative mean lifespans were also adding to the general increase in the *MLD* for all populations, leading to a higher overall *BG* component.

	Males				Females			
	$MLD_{30\|70}$	*WG*	*BG*	*BG/T*	$MLD_{30\|70}$	*WG*	*BG*	*BG/T*
Estonia 1990*	9.15	9.05	0.10	**1.14**	3.50	3.49	0.01	**0.25**
Estonia 2000†	10.73	10.42	0.31	**2.89**	4.74	4.68	0.07	**1.46**
Lithuania 1990*	10.83	10.71	0.12	**1.14**	4.69	4.67	0.02	**0.35**
Lithuania 2000†	10.85	10.44	0.41	**3.79**	4.83	4.72	0.11	**2.25**

*the 1990 period corresponds to 1987-1991 in Estonia and 1988-1990 in Lithuania
†the 2000 period corresponds to 1998-2002 in Estonia and 2000-2002 in Lithuania

Table 3: The temporary mean logarithmic deviation between the ages 30 and 70 (x100) ($MLD_{30|70}$) and its decomposition into within-group (WG) and between-group (BG) components; BG/T is the contribution of between-group inequalities to temporary lifespan variation

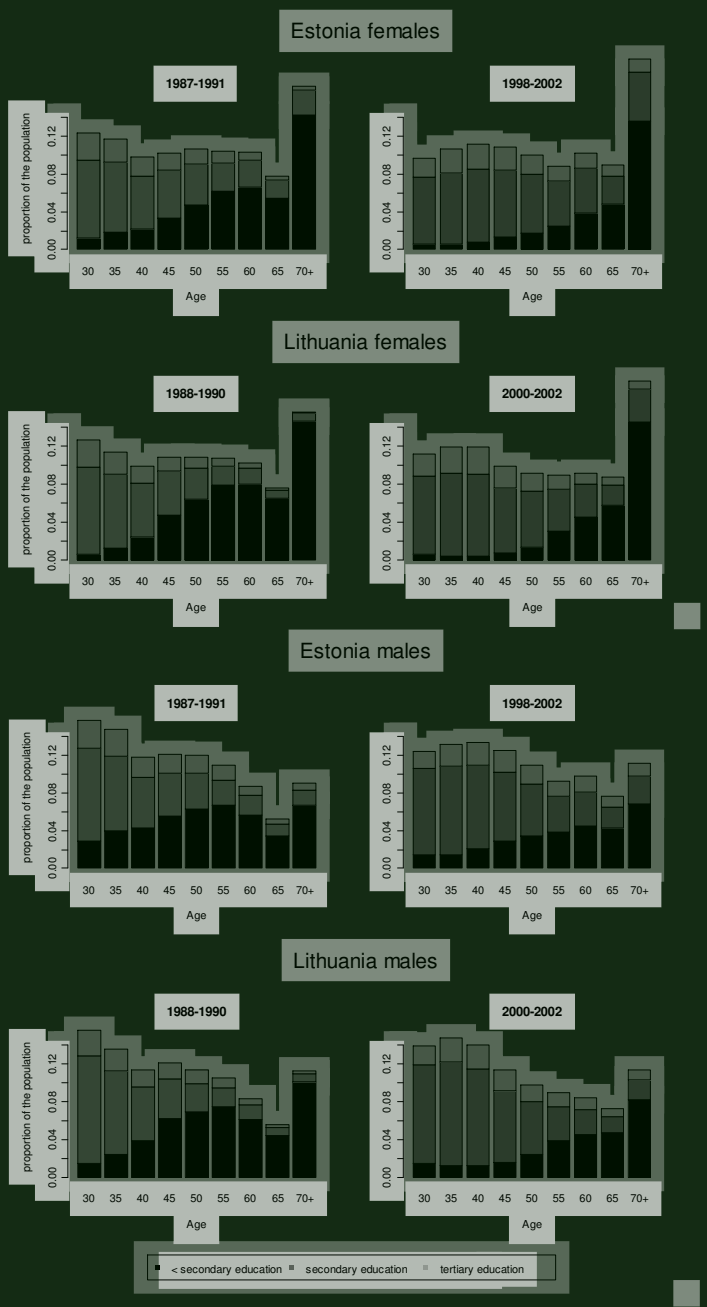

Figure 2: Population proportion by age, sex, country and educational level.

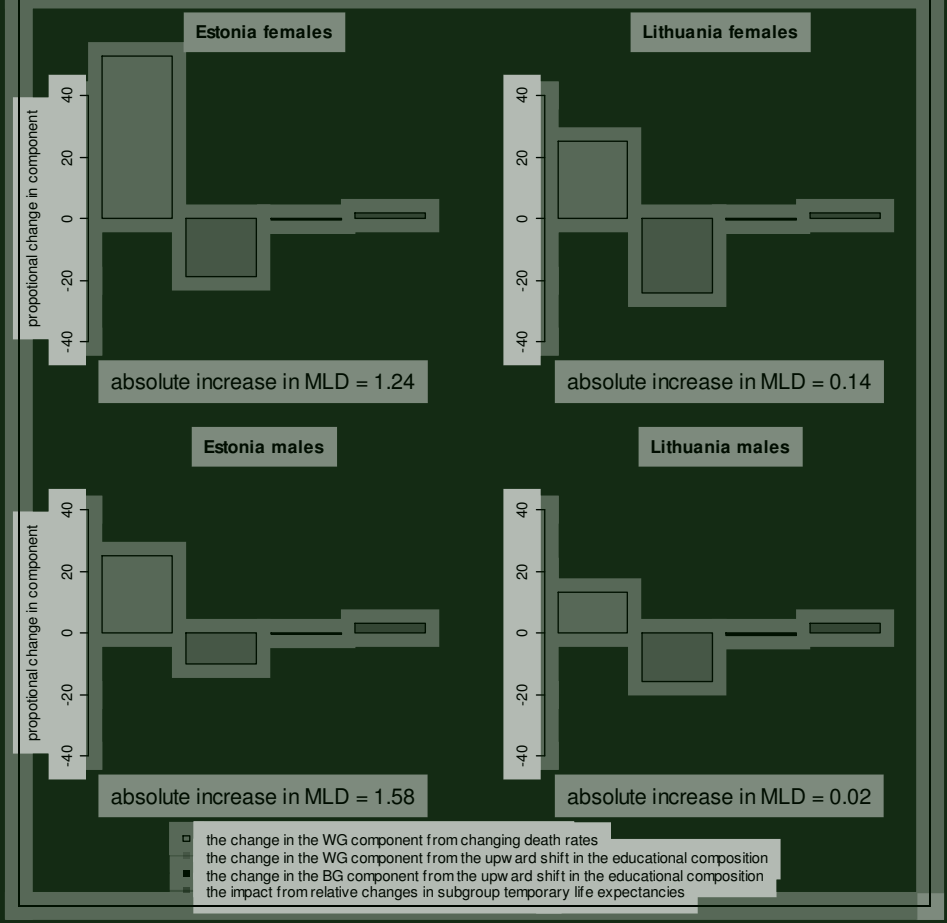

Figure 3: Changes in lifespan variation (MLD) attributable to direct and compositional changes in the within- and between-group components over the two time periods.

CHAPTER 7

Discussion

Summary of results

This analysis showed that temporary lifespan variation between the ages of 30 and 70 was higher at the end of the 1990s than it was in the beginning, and proportionally higher for females. This expansion was much greater in Estonia than in Lithuania, unlike the drop in temporary life expectancy, which was similar in both countries. This was owing to the differential mortality patterns over age. Estonians experienced higher mortality in the age category 30-40 during the later period, while these were ages where Lithuanians posited reduced mortality.

The diverging mortality experiences by educational subgroup led to greater stratification in the age-at-death distributions, as well as increased inequalities in temporary life expectancy. Consequently, the BG inequality component both rose and explained a greater part of the lifespan variation. Expansion of the age-at-death distribution in the lower and middle educated groups fuelled the overall increase in lifespan variation, although this was to some degree tempered, particularly in Lithuania, by a rising educational composition.

Evaluation of data and methods

The nature of unlinked studies introduces a numerator/denominator bias, as educational status was self-reported on the census and reported by next of kin on the death records. While in principle this bias could go either way,[15-17] the Lithuanian data for the later period we use here has been shown to overestimate educational inequalities in mortality, particularly for females, by overestimating mortality in the low educated groups and underestimating mortality among the high educated.[5 18 19] Although no linkage study has been performed in the earlier period for Lithuania, speculation is that the bias might have been smaller due to greater discipline in reporting information to authorities, more uniform educational systems across the Soviet Union and greater coherence among educational categories in the Soviet census and death records.[20] On the other hand misreporting was greater among those with less formal education which could also suggest that the situation might have been worse in the earlier period given the higher proportion of the population in the lower educated groups. Unfortunately no linkage study has been performed in Estonia over either period. Although the correspondence between census and death

records appears to have been better in Estonia,[1] we would nevertheless assume an overestimation of mortality inequalities there as well.

Given that no better data exists for Estonia or for the earlier period in Lithuania, we have taken steps to mitigate this bias. Misreporting by education was found to be strongest in the oldest age categories. Presumably this is because these individuals would have been educated in pre-Soviet times, categories which did not always correspond well with categories listed on the death records. Therefore, we limited our analysis to temporary lifespan variation between the ages of 30 and 70 as suggested by Shkolnikov et al.[5] Also, although we had data differentiated by primary and lower secondary education, we collapsed these two categories, assuming that much of the misreporting would be happening between these two groups.

The mortality shocks experienced by Estonia and Lithuania during the 1990s were most severe during the middle part of the decade (Figure 4). Our educational subgroup data is aggregated around a few years at the beginning and end of this decade. Thus the changes we observe reflect the changes in age-specific mortality at the beginning and end of the decade, and unfortunately cannot capture the changes in the middle part of the decade. Additionally, the study periods differ by a few years in Estonia than Lithuania, which might partially account for some of the observed differences between the two countries.

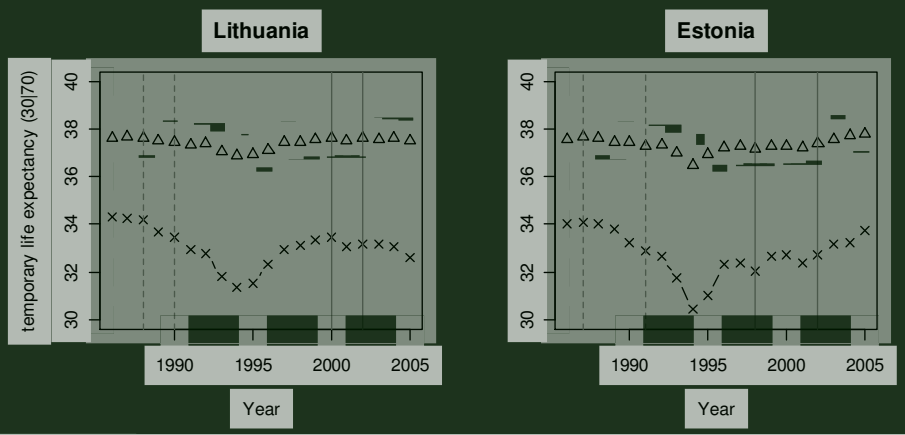

Figure 4: Trends in temporary life expectancy between the ages of 30 and 70, Lithuania and Estonia. The period in-between the two dotted grey bars corresponds to our earlier period, while the years in-between the solid grey bars are our later period. The measures were calculated based on period life tables from the Human Mortality Database, accessed 01/02/2010.

We used the mean logarithmic deviation to measure lifespan variation since it is the only additively decomposable measure whose between- and within-group components can be decomposed into direct and compositional effects. Usage of a different measure would have resulted in differences in the magnitude of change in lifespan variation over this time. The mean logarithmic deviation, though well correlated with other measures, is known to be more sensitive to changes in the left tail of the distribution. Given that mortality increases were concentrated precisely at these younger adult ages, the mean logarithmic deviation responded more to changes over this period than other measures of lifespan variation, although the general pattern remained similar (Figure 5).

Additionally, there could be concern about the statistical power of mortality estimates of the educational subgroups. Lithuania and Estonia are small populations. However, the confidence intervals obtained via Monte Carlo simulations showed that our point estimates for both temporary life expectancy and temporary lifespan variation were reasonable. Thus we do not expect our results to be heavily biased based on random fluctuations of vital events.

Finally there is a question about whether three subgroups are enough to capture the contribution of educational inequalities to lifespan variation. In Chapter 6 we found only a small reduction in the BG component by reducing the number of educational subgroups from four to three.[19] So long as we are capturing a linear gradient to mortality we do not expect this bias to be large.

Conclusion

This paper has demonstrated another dimension to the mortality shocks experienced by Estonia and Lithuania during the 1990s. In particular, it has shown that compositional changes to the populations tempered what would have been an even larger increase in lifespan variation. Lower educated groups especially faced much larger lifespan variation at the end of the 1990s than they did in the beginning. Also, we have shown that women experienced great changes to their age-at-death distribution, an aspect not well highlighted in the literature on transitional mortality experiences. Finally this study revealed that the contribution of educational differences in mortality on lifespan variation increased substantially in both Lithuania and Estonia during the tumultuous period.

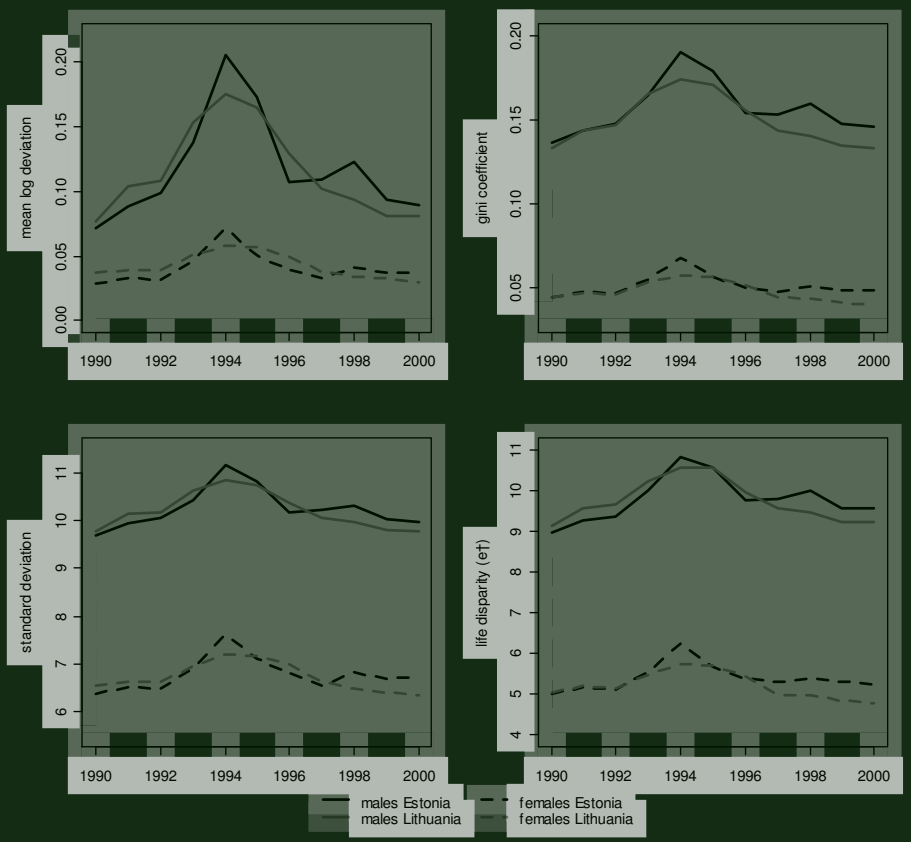

Figure 5: The time trends in lifespan variation between the ages 30 and 70 according to various measures. The measures were calculated based on period life tables from the Human Mortality Database, accessed 01/02/2010.

More generally, greater lifespan variation implies greater uncertainty in the timing of death. Cast in this way, it may be even more detrimental to life planning and well-being than a lower average lifespan. Since the 1960s, lifespan variation in over adult ages in developed countries has mostly stagnated, despite considerable improvements in life expectancy.[21-26] This paper ads to the literature arguing that both dimensions are important to examine separately, as trends in the average and the variation around this average can sometimes point in different directions.

While this study was focussed on the exceptional changes occurring in Lithuania and Estonia during the 1990s, the methods introduced here are general. By separately examining between-group average and within-group distributional changes in age-at-death we get a different, but complementary picture to traditional methods that focus on socioeconomic inequalities as being between-group differences. Moreover, decomposing lifespan variation into direct and compositional changes can help to determine the efficacy of public health policies in targeting vulnerable groups, who because of compositional changes may be becoming increasingly selected. Reducing socioeconomic inequality in mortality requires both raising the average length of life of disadvantaged groups as well as reducing the dispersion around this average.

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CHAPTER 8

Discussion

CHAPTER 8

Introduction

In this thesis I, together with invaluable help from co-authors, aimed to draw attention to the importance of summarizing the dispersion in lifespans among individuals, in addition to the current practice of summarizing average levels of mortality. More specifically I set out to answer the following research questions:

1. What is the most appropriate way to measure variation in age-at-death?
2. What is the relationship between lifespan variation and life expectancy?
3. How much are educational differences contributing to lifespan variation?

I will first turn to addressing these questions before opening a more general discussion.

Addressing the research questions

1. What is the most appropriate way to measure variation in age-at-death?

There is no one appropriate way to measure lifespan variation. This was the primary conclusion reached in chapter two of this thesis, and adopted in subsequent chapters. All measures examined in this thesis are highly correlated, and will accurately describe broad changes in the age-at-death distribution. Nevertheless different conclusions can be expected between populations with similar lifespan distributions, based on the measures' different underlying sensitivities to changes in mortality at different ages.

Researchers are advised to use the measure best suited to the research question. In some instances this choice will be dictated by the formal properties of the measure (e.g. decomposability, formal demographic relationships to other measures, ease of interpretation of the measure). At other times researchers may be flexible to use the measure best corresponding to the normative values placed on mortality reduction at different ages. The perturbation analysis methods to measure the sensitivity and elasticity of measures developed in chapter 2 allow this choice to be made explicit. When possible, I recommend using two or more measures with different underlying sensitivities before coming to any strong conclusions about the extent of lifespan variation in a population.

2. What is the relationship between lifespan variation and life expectancy?

Lifespan variation and life expectancy are highly negatively correlated. This correlation owes to progress at postponing premature mortality, defined in this thesis as deaths occurring at ages which, if postponed, would lead to compression in the age-at-death distribution (with a unique threshold age separating premature from older ages). Although only 38 percent of all deaths since 1840 have been premature using the life disparity measure, fully 84 percent of increase in life expectancy has come from progress in postponing premature deaths to higher ages. Lifespan variation up to the threshold age has fallen by more than half since 1840, while lifespan variation after the threshold age has over this time remained nearly constant.

Out of the 170 years from 1840 to 2007, 89 male and 86 female holders of record life expectancy also enjoyed the lowest life disparity. The countries that have been most successful in reducing premature mortality are the current life expectancy leaders. Japanese females are one such example. In addition to having the record life expectancy, Japanese females had the lowest life disparity from 1980 to 1995. Investigations into their age pattern of mortality decline show it as having been close to the most efficient pattern for increasing life expectancy. Recently Japanese females at older ages have begun contributing most gains to life expectancy, explaining the current stagnation in lifespan variation. Whether this is a pattern that will be replicated elsewhere remains to be seen. In chapter 5 some evidence for this was uncovered among the highly educated Swiss and Swedish female populations.

3. How much are educational differences contributing to lifespan variation?

For every population I examined, higher educated groups not only lived longer but also had lower lifespan variation than groups with fewer years of education. The gradient was steeper in Eastern Europe than in Western Europe, and among males. Differences in causes of death that pronounced themselves at younger ages had a larger impact on the discrepancies between educational groups in their lifespan variation than in their average age at death.

Differences in lifespans among individuals come from many sources, including genetics, lifestyle and chance. Educational between-group differences in

life expectancy explained about a percent of the total lifespan variation in Western Europe, and up to 5 times this amount in Eastern Europe. Educational inequalities explained more of the total lifespan variation ten years after transition in Estonia and Lithuania than around 1990, despite differences being tempered by upward shifts in the educational composition. Although educational inequalities explained only relatively small proportions of the total variation, differences in mortality arising from socioeconomic inequalities are perhaps among the most unfair sources of this variation.

Methodological considerations

When interpreting the results presented in Sections II and III of this thesis, a number of factors need be considered. These relate to the data itself, the measures used, the causality in the processes examined, and the validity of generalizing the results to regions outside of the areas under study.

Data considerations

The data used in this thesis was cross-sectional, meaning that all age-at-death distributions were drawn from synthetic cohorts exposed to death rates pertaining to a specific time period. This has the known advantage of being able to reflect current mortality patterns without having to wait for all members of a cohort to die. Nevertheless these are hypothetical cohorts, with no actual cohort having been exposed to these death rates over their lifetime. Comparing the two, period life expectancy improvements tend to be slower than cohort life expectancy improvements,[1 2] while the period life expectancy at any given time approximates that of a cohort born 40 to 50 years earlier, though this lag is increasing over time.[2 3] Moreover, it is also claimed that period mortality is subject to tempo effects under changing mortality conditions.[4] These effects were estimated to have accounted for up to 3.3 years of the ascension in Japanese female life expectancy over the 1980-1995 period.[5] Whether these contested 'effects' are actually 'distortions' remains a controversial unresolved issue in demography (see, for instance, Wilmoth[1] and the articles in the Barbi et *al.* collection[6]). Since I interpret the results as reflecting period age patterns of mortality, without trying to infer any cohort patterns, I did not make any attempt to correct for tempo effects.

The data used in this thesis came from two sources: the Human Mortality Database and the Eurothine data collection. For data to be included in the HMD, there needs to be a 'well-founded belief that the coverage of their census and vital registration systems is relatively high'.[7] Nevertheless, in the background documentation, for some countries warnings are sounded over the quality of data pertaining to certain country and time periods. Most of these warnings relate to periods before 1950. While these problems could have affected some of our estimates in Chapter 3, I do not expect any impact on our general conclusions. Where possible, we used different colour schemes to distinguish historical from more recent events. As the HMD does not correct the raw data apart from egregious errors, age heaping and age exaggeration could also have caused some distortions in the data. In the methods that were used, age heaping would not make a large difference, as the over and under estimation of age would balance itself out. I also expect the impact from age exaggeration to be minor, both because exaggeration generally happens over ages where there are fewer events, and because measures are less sensitive to changes in age-specific mortality at older ages. In addition, above age 80 the HMD uses extinct cohort and survivor ratio methods to determine the exposure population, rather than relying on census counts which are thought to be less accurate, and they smooth life table death and exposure counts at older ages using the Kannisto method.[8]

Section III of this thesis used Eurothine data collected in two different formats: census-linked data which followed individuals for around 10 years over the 1990s and census-unlinked data which aggregated age-specific mortality rates over a few years. Comparing these two datasets may have introduced biases relating to the different time periods under study (the cross-sectional studies took place on average 5 years later) and biases from the data formats. Thus a degree of caution should be taken in comparing the results from the two types of study. Finally, the census-unlinked studies may have introduced numerator-denominator biases resulting from differences in educational achievement reported in the census (numerator) and death certificates (denominator). While in theory these biases could run either way,[9] I assumed based on a Lithuanian record-linkage study[10] that the census-unlinked results may have overestimated the magnitude of educational inequalities in mortality, although I expect this bias to have been small.

CHAPTER 8

Validity of our measurement of lifespan variation

The method to measure lifespan variation can at times lead to different conclusions depending on the sensitivity of the measure to changing mortality at different ages, as was shown in chapter 2. The considerations for choosing a measure in later chapters were mostly driven by the measure's formal demographic properties, particularly decomposability, and to a lesser extent the ease of interpretation. In all cases I checked whether having used a different measure would have changed the results. I found that the usage of a different measure could have moderated some of the results, though it would not have affected any of our broad conclusions. Specifically in Section III, all three measures used, the Theil's index, the Variance, and the Mean Logarithmic Deviation are sensitive to changes at early ages in the death distribution. The usage of the Gini coefficient or Life Disparity would have likely shown smaller differences in lifespan variation between educational subgroups, since the differences between groups were driven by different levels of premature mortality.

Validity of generalizing our results to other regions

The countries examined in this thesis were all medium to high income countries with good quality, comparable data. In chapter 3, which aimed at finding universal patterns in the relationship between life expectancy and lifespan variation, the analysis was limited to countries included in the Human Mortality Database. In chapters 4-6, the harmonized Eurothine data from 11 European countries was used to examine the role of socioeconomic inequality in shaping lifespan variation. Thus the patterns observed here were largely drawn from mortality patterns observed in Europe and English speaking western offshoots. Asian mortality analysis was limited to Japan and Taiwan, while South America was only represented by 24 years of Chilean data. Conspicuously absent from all analyses were China and India, together representing about a third of the world's population. It is unknown whether the patterns and relationships described in sections 2 and 3 of this thesis can be generalized to the rest of the world. Moreover, most high mortality populations included in the analyses were drawn from historical northern and western European records with a long history of data collection. These age patterns of mortality may not reflect the experiences of contemporary developing countries with similar life expectancies.

Comparisons to other findings

Compared to the wealth of studies describing trends in average levels, relatively few studies have examined the variability surrounding lifespans. Those that have can be divided into two groups. The first are studies that are concerned with the notion of compression of mortality at advanced adult ages. Initially these studies aimed at proving or disproving Fries' theory of mortality compression,[11] which centred upon reaching a fixed upper age limit to human lifespans.[12-14] More recent studies tended to examine mortality above the mode with the aim to see whether old-age mortality was being further compressed, was shifting to higher ages, or was becoming increasingly heterogeneous.[15-19] The second group of studies covered age-at-death variation over the entire lifespan,[13, 20-25] or the adult age at death distribution conditional upon surviving childhood.[26-28] These studies were especially concerned with whether lifespan variation was a separate dimension from life expectancy, and itself worth monitoring. What they tended to find was that although the two were highly correlated, differences between countries at the same level of life expectancy were substantial, and could not be easily explained. Moreover, Edwards and Tuljapurkar[26] found that differences between low mortality countries in the rate of convergence to the 2005 Swedish female age-at-death distribution increasingly came from differences in lifespan variation as opposed to differences in life expectancy.

The results from this thesis generally were in line with the conclusions of these previous studies. An exception was the study by Smits and Monden[27] on the relationship between life expectancy and lifespan variation. Using different methods than those presented here, they aimed to determine whether the timing of mortality reduction was related to the level of lifespan variation. What they found was that countries that achieved a level of life expectancy later in time did so with lower levels of lifespan variation than forerunner countries. This led them to conclude that "reducing inequality and gaining increases in life expectancy might be alternative goals that require different policy measures to be achieved". While I agreed with their results, albeit finding them to be weak and to only hold over a short time frame or life expectancy range, I found that their conclusion could not be justified. The results of Chapter 3 demonstrated that successful countries had achieved higher life expectancy by reducing premature deaths, thereby also reducing lifespan variation. In later chapters I showed that reducing socioeconomic inequality could be a means of reducing lifespan variation, while at the same time raising life expectancy.

The studies in Section III were the first large-scale comparative studies examining lifespan variation by socioeconomic subgroup. The results confirmed the

scattering of findings from Russia[21] and the United States[26 29] suggesting that as well as having shorter average lifespans, lower educated groups additionally faced greater lifespan variation. This had also been shown for African-Americans compared to White Americans.[26 30] These studies in addition to our own complement existing public health and demographic research examining average levels in mortality, giving greater insight into the age pattern of mortality shaping differences in mortality between groups, and between individuals. The magnitude of the socioeconomic gradient to lifespan variation followed similar geographic patterning in Europe to the socioeconomic gradient in life expectancy—larger inequalities in Eastern Europe and a smaller gradient in Nordic and Western Europe.[31-33] What this thesis adds is that these differences owe to a large extent to differences in the left tail in the age-at-death distribution. Higher mortality at younger adult ages is causing the lower educated groups to have both lower life expectancies and greater uncertainty in the timing of death.

Implications for policy

In this thesis I showed the strong relationship between high life expectancy and low lifespan variation. This was shown from birth at the macro level in Section II and from age 30 to 35 by educational groups in Section III. A substantial proportion of the differences between populations in lifespan variation were due to differences in premature mortality. It is striking that even at the same level of life expectancy differences between populations in the standard deviation of lifespans were in the neighbourhood of 2 years. Measured by life disparity, this roughly equated to a year and a half of remaining life expectancy between populations.

These differences imply that substantial room exists for policymakers to target mortality at ages that would reduce differences in lifespans between individuals. Such ages can readily be determined by the perturbation analysis techniques introduced in Section I. Generally, lifespan variation tends to be more sensitive to deaths at younger ages than life expectancy. Thus equalizing life chances would require more attention to causes of death striking individuals at earlier adult ages, such as external caused mortality, certain cancers and circulatory diseases. This might require social safety nets to protect high risk individuals and public health campaigns focussed on reducing alcohol abuse and lowering personal risks from injury. The contribution of smoking to lifespan variation, although it was not specifically examined in this thesis, is also likely to be substantial in that the

diseases that manifest themselves through years of smoking tend often to kill individuals at ages that I have identified here as being premature.[34]

Furthermore, this study found that lower educated groups have higher within-group levels of lifespan variation in all countries examined. This was caused by their higher levels of premature mortality, although the causes of death varied by country. Lifespan variation was especially high in some of the Eastern European countries we examined, and the contribution of educational inequalities in mortality to the total lifespan variation there were shown to have risen substantially over the transitional period of the 1990s. Policies should be designed to identify and protect vulnerable individuals, particularly through periods of stress and hardship. Reducing socioeconomic inequality requires both raising the average length of life of disadvantaged groups as well as reducing the dispersion around this average.

Open questions and directions for future research

For every question this thesis set out to answer, many new ones appeared. To better understand the implications of the results described in this thesis and to help design effective policy interventions, the following unanswered questions should be addressed.

Is reducing lifespan variation a valid policy objective?

First and foremost at times we almost took it as a given that reducing lifespan variation is desirable. Reducing lifespan variation benefits both individuals and society. An increased certainty in the timing of death increases the value of public and private investments in education and training, enhances the ability to smooth consumption over the life course, and can improve the public provisioning of pensions and medical care. Having a healthy workforce increases productivity and can be a prime driver of a country's income.[35] Meanwhile, if a high risk of premature death is perceived among individuals, this could conceivably lead to riskier behaviour and a general feeling of helplessness over the timing of life's events. Certainly the higher lifespan variation of disadvantaged groups brought about through elevated premature mortality suggests that there might be a moral imperative to equalizing the life chances across social groups. To the extent that such excess mortality is avoidable and unjust it is considered by many as inequitable.[36]

Nevertheless, whether reducing lifespan variation is a valid policy objective can be questioned. Certainly early deaths are tragic, robbing individuals of the opportunity to realize their hopes and ambitions. However, reducing lifespan variation could also be brought about by increasing the mortality levels at older ages—an obviously undesired result. In a more muted form, in Chapter 4 it was noted that the Japanese female population seemed to have reached a turning point, whereby reductions in old-age mortality were outpacing reductions in premature mortality, thereby leading to slowly increasing levels of lifespan variation. I also uncovered some evidence of this in Section III, amongst the highest educated female populations of Western Europe. This leads one naturally to wonder whether these patterns observed at the frontier levels of life expectancy are a trend that will trickle down to all populations. It also begs the question of whether increased dispersion of old age mortality should be cause for moral concern. This might be an especially difficult question to answer if, as was speculated in a recent study, some of the differences between the exceptionally long-lived populations and the rest of the advanced nations might have to do with attitudes toward the elderly and the willingness to perform surgeries on patients into their 80s and 90s.[37] On the other hand if lifespan variation is increasing at older ages because technologies or medical care are not being made equally available to all elderly members of society this would certainly be deemed inequitable.[38]

Even if we only consider policies to lower lifespan variation by reducing premature mortality, as I have argued in favour of here, certain difficult choices would have to be made. By definition reducing lifespan variation requires that reductions in premature mortality continue at a faster pace than reductions in old age mortality. With finite budgets, targeting premature mortality would imply a degree of age rationing in health priorities. Given a choice, would individuals rather public spending be directed to equalizing life chances or to improving survival probabilities at the oldest ages? Ethical analysis and measuring preferences of the population on this matter would dictate to a large extent how trends in survivorship should be monitored, and where interventions should be prioritized.

What are the determinants of lifespan variation?

In high income countries, individuals are dying with an average remaining life expectancy of 9 to 10 years (Chapter 3). What is driving these individual level differences in age at death? In Chapter 5 and 6 we found that socioeconomic inequality was explaining a small portion of this individual variation. Studies on

Danish twins have found that longevity is moderately heritable.[57 58] Certainly chance plays a role. Some individuals are simply frailer than others or more susceptible to certain diseases. Other potential pathways could include lifestyle and behavioural factors, material and living conditions, and environmental conditions. Macro-level factors, such as the ability and willingness to perform life extending procedures might also play a role. Tackling the discrepancies between individuals in their age at death can only be done through a comprehensive understanding of the determinants of lifespan variation.

Have differences in lifespan variation by socioeconomic group changed over time?

The relationship between the *average* length of life and the *variation* around this average is far from being settled. The results of Chapter 3 confirm the finding that high life expectancy is associated with low lifespan variation.[20 21] This relationship has persisted over time and, as was showed, is due to the progress in reducing premature mortality having been greater than the progress in reducing old age mortality. When infant mortality is excluded from the analysis, however, the picture becomes clouded. On the one hand a clear association between high remaining life expectancy at age 35, (e_{35}), and low lifespan variation at age 35, (S_{35}), was found at the educational subgroup level in Chapter 4. On the other hand, at the macro level decreases in lifespan variation conditional upon survival to ages broadly in the 10-40 age range has mostly stagnated since the 1960s, despite continued improvements in remaining life expectancy beyond this starting age.[20 23 24 26 28 39]

Hence future attention should be directed toward determining whether, in a period of increasing life expectancy, lifespan variation by level of education has persisted over time (a shifting scenario) or diminished with time (a reduction scenario). Each finding would have its own implications. A reduction scenario in lifespan variation by educational achievement would suggest that the highest educated acted as a vanguard group. This scenario could present itself if higher education provided a pathway to adopting better health habits or to taking earlier advantage of medical breakthroughs, behaviours that were eventually transmitted to lower educated individuals. While the level of lifespan variation would be the same for the subgroups at each level of life expectancy, at any given time differences between groups could persist as the high educated would have higher life expectancy and lower lifespan variation. A shifting scenario would imply that individuals with lower education faced greater uncertainty in the timing of death at

all levels of life expectancy. This could be owing to differences in environmental conditions, lifestyle and behaviour, or the psychosocial environment. In parallel, the role of common determinants of mortality inequalities including smoking patterns should be examined longitudinally, as a way of trying to understanding both macro and subgroup trends in their different levels of lifespan variation. Studies on time trends in socioeconomic differences in mortality might also shed some light on these hypotheses.

Does the timing of mortality reduction make a difference to the level of lifespan variation?

While I argued that the strong conclusions aired by Smits and Monden about the incompatibility of high life expectancy and low lifespan variation could not be justified, their results on the timing of mortality reduction and the pattern of lifespan variation do present an interesting puzzle. How can it be that on the one hand the closer a country was to the record life expectancy for any year, the closer that country was to having the lowest lifespan variation for that year (our study), while at the same time countries achieving a certain level of life expectancy later in time did so with higher levels of lifespan variation (their study)? One possible explanation could come from the differences in studying this question cross-sectionally versus over time. It was recently hinted by Shkolnikov et *al.,*[22] that between-country differences in lifespan variation at any given time came about for different reasons than these differences over time. Cross-sectionally, they found that countries with lower economic inequality also had lower levels of lifespan variation. However within a country, changes over time in economic inequality were not significant in explaining changing levels of lifespan variation. Likewise different causes of death were shaping between-country differences in lifespan variation over the cross-section in comparison to the causes of death shaping within-country differences over time. The Shkolnikov et *al.* study was limited to comparing the United States to England and Wales. An open question is whether these findings can be replicated in other countries, and could reconcile the findings of Smits and Monden with our own.

What is driving educational differences in lifespan variation?

Although I found in Section III that different levels of education translated into different age patterns of mortality, without additional covariates I could not infer

what it was about education that was driving these differences. For example, did higher educated individuals simply have better health-seeking behaviour? Did increased social capital allow them to become better informed about the best medical treatments available? Or did higher education translate to greater wealth and healthier living environments? Moreover it could be that the relationship did not run from education to health but rather from health to education, i.e. that sicker individuals were unable to complete the same years of education and died at earlier adult ages. Much has been learned already about the determinants of socioeconomic inequalities in mortality. For instance, we know that the higher mortality of lower socioeconomic groups is caused in part by behavioural differences (especially regarding cigarette smoking, diet, exercise and alcohol abuse),[40-46] material differences,[47-52] and psychosocial pathways.[53-56] Insofar as these determinants cause elevated levels of premature mortality, they can also be expected to drive differences in lifespan variation by socioeconomic group. As a first examination, the studies of this thesis provided descriptive results showing differences in lifespan variation by socioeconomic groupings. Further work needs to be done to understand the causes and consequences of these processes.

Are there similar differences in the distributions of healthy life expectancy?

In this thesis I limited the focus to mortality. However the methods I have used could be extended to morbidity research. Nationally representative surveys are routinely being conducted to monitor trends in disability, functional limitation and activity restrictions. This has led to a burgeoning number of studies examining trends and inequalities in healthy life expectancy, disability-free life expectancy and active life expectancy (see, for instance,[59-63]). As far as I am aware, no studies have taken the next logical step forward, which would be to examine inter-individual differences in healthy life expectancy. I would also expect substantial differences in the distribution of healthy life expectancy between populations, even at the same average level of morbidity. An interesting question would be to compare the magnitude of differences in the level of variation in healthy life expectancy as compared to lifespan variation across populations.

CHAPTER 8

Conclusion

Lifespan variation is a dimension of mortality that to date has not received the attention that it deserves in demographic and public health circles. This thesis aimed to get past some of methodological concerns preventing widespread adoption of measuring lifespan variation and to present powerful methods to allow policy-makers to monitor the distribution around average levels of mortality. Moreover the studies included in this thesis documented long-term trends in lifespan variation at the macro level, and differences between educational groups during the 1990s. As data is becoming more refined, more available and of a generally higher quality, sophisticated methods that examine the entire age-at-death distribution can improve our current understanding of mortality dynamics and patterns. While it is certainly important to track trends in average levels of mortality, understanding the dispersion around these average levels can help to better target vulnerable individuals and implement effective policy interventions.

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SAMENVATTING

Inleiding

De levensverwachting bij de geboorte is sinds 1840 in landen met een hoge welvaart en een succesvol sociaaleconomisch beleid bijna lineair toegenomen. In eerste instantie was de stijging van de levensverwachting primair het gevolg van een reductie van de sterfte bij pasgeborenen en jonge kinderen. In de loop van de tijd verschoof het leeftijdspatroon van de sterftereductie naar een steeds hogere leeftijd op. Als gevolg hiervan verschilt het huidige leeftijdspatroon van de sterfte kwalitatief sterk van dat in het verleden. De kindersterfte-'bult' is grotendeels verdwenen en de mortaliteit concentreert zich in toenemende mate rond de modale stervensleeftijd voor volwassenen. Er blijven niettemin verschillen bestaan tussen landen en sociaaleconomische groepen met betrekking tot de mate waarin deze mortaliteitscompressie heeft plaatsgevonden.

In demografisch en volksgezondheidsonderzoek wordt de gezondheid van een populatie vaak uitgedrukt als de levensverwachting bij de geboorte. De variatie rond dit gemiddelde wordt echter maar zelden aangegeven. Dit kan belangrijke verschillen in de vorm van de distributie van de levensduur maskeren. Onderdeel van het probleem zijn een gebrek aan consensus over de te gebruiken methode en verschillende normatieve gezichtspunten met betrekking tot het effect van risicomijdend gedrag op veranderingen in de sterfte op verschillende leeftijden.

In dit proefschrift stel ik mij ten doel een brede studie te ondernemen naar de variatie in menselijke levensduur. Meer specifiek probeer ik de volgende onderzoeksvragen te beantwoorden:

1. Wat is de beste manier om de variatie in leeftijd bij overlijden te meten?
2. Wat is de relatie tussen de variatie in levensduur en de levensverwachting?
3. In hoeverre dragen verschillen in opleiding bij aan de variatie in levensduur?

Gegevens

De gegevens die ten grondslag liggen aan dit proefschrift zijn afkomstig uit twee bronnen: de Human Mortality Database (HMD) en de Eurothine-dataset. De HMD is een open-source-dataset die gebruikmaakt van vergelijkende nationale sterftegegevens uit 36 landen en regio's. In sommige landen gaan de gegevens terug tot de 19^{e} eeuw of eerder, terwijl voor andere landen alleen gegevens beschikbaar

zijn uit de periode vanaf de tweede helft van de 20e eeuw. Alles bij elkaar bestond de HMD-dataset op het moment van raadpleging uit 6860 sterftetafels van zowel mannen als vrouwen. De Eurothine-dataset is in deel III gebruikt om vragen te beantwoorden met betrekking tot de relatie tussen sociaaleconomische ongelijkheid en variatie in levensduur. Deze dataset bevat geharmoniseerde, uit volkstellingen verkregen mortaliteitsgegevens uit elf Europese landen voor de leeftijdsgroep van 30-85+ jaar, in leeftijdsklassen van 5 jaar en gecategoriseerd op sekse en opleidingsniveau. Er is gebruikgemaakt van methoden om de Eurothine- en HMD-datasets met elkaar te vergelijken (deel III) om zo te kunnen komen tot een meer continue distributie van leeftijd bij overlijden.

Samenvatting deel I

In de laatste jaren zijn verschillende maatstaven toegepast om de variatie in levensduur te berekenen, elk met andere onderliggende eigenschappen. Hoewel wordt aangenomen dat deze maatstaven onderling uitwisselbaar zijn, is er nog weinig onderzoek gedaan naar de vraag onder welke omstandigheden deze aanname opgaat of om de responsen van de verschillende maatstaven op het onderliggende mortaliteitsschema met elkaar te vergelijken. In dit deel worden zeven meetmethoden voor variatie in levensduur vergeleken: ongelijkheid in levensduur ('life disparity'), de Gini-coëfficiënt, de standaardafwijking, de variantie, de Theil-index, de gemiddelde logaritmische afwijking en de interkwartielafstand. De sensitiviteit en elasticiteit van elke maatstaf werd afgeleid met behulp van de Markov-ketentheorie en matrixberekening. Op basis van empirische gegevens van Franse en Russische mannen werden de sensitiviteiten van de verschillende maatstaven voor sterfteverandering onder verschillende mortaliteitsregimes vergeleken. Zo kon worden getest onder welke omstandigheden de maatstaven verschillende conclusies opleveren over de omvang van de variatie in levensduur. Ten slotte hebben we laten zien hoe integratie van deze sensitiviteiten kan worden gebruikt als methode om te komen tot een decompositie van de totale levensduur. De resultaten van dit deel suggereren dat er niet één 'beste' manier is om de variatie in levensduur te meten en dat de verschillende maatstaven verschillen in de mate waarin ze gevoelig zijn voor verandering in leeftijdsspecifieke sterfte. Het resultaat van onze analyse is een eenvoudige methode om de eigenschappen te berekenen van deze belangrijke klasse van maatstaven voor de levensduur. Deze methode stelt onderzoekers beter in staat om hun keuze voor een bepaalde maatstaf af te stemmen op het effect van risicomijdend gedrag op veranderingen in de sterfte op

verschillende leeftijden binnen de bestudeerde groep. In het algemeen betogen we dat het altijd het veiligst is om twee of meer maatstaven met een verschillende sensitiviteit te gebruiken voor het meten van variatie.

Samenvatting deel II

Al twee eeuwen is de levensverwachting in welvarende landen gestaag toegenomen. De sterftereductie is sneller verlopen op jongere dan op oudere leeftijden, met een compressie van de distributie van de levensduur als gevolg. Oudere studies kwamen tot de conclusie dat de reductie van de variatie in levensduur enerzijds en de verhoging van de levensverwachting anderzijds verschillende doelen zijn die verschillende beleidsmaatregelen vereisen. In hoofdstuk 3 bepalen we het aandeel van de voortschrijdende reductie van vroegtijdige sterfte in de verhoging van de levensverwachting en de afname van de variatie in levensduur. De variatie in levensduur is gemeten door middel van 'life disparity', een maat voor de mate waarin de levensduur van verschillende individuen verschilt. We spreken van vroegtijdig overlijden als uitstel van het overlijden de ongelijkheid in levensduur zou hebben verminderd. Uit het onderzoek bleek dat de reductie van ongelijkheden in levensduur door het terugdringen van vroegtijdig overlijden het grootste effect had op de stijging van de levensverwachting. Sterfte op hogere leeftijd blijkt weinig effect te hebben op de variatie in levensduur en slechts een bescheiden bijdrage te leveren aan de stijging van de levensverwachting.

In die landen die er het best in zijn geslaagd om vroegtijdige mortaliteit terug te dringen, is de levensverwachting het hoogst en de ongelijkheid in individuele levensduur het geringst. Het record is op dit punt in handen van de Japanse vrouwen: ze hebben al sinds 1986 de hoogste levensverwachting. Bovendien was van 1980 tot 1995 de ongelijkheid in de individuele levensduur bij deze bevolkingsgroep het geringst. In hoofdstuk 4 laten we zien dat deze populatie een 'efficiënt' sterftereductiepatroon heeft gevolgd: die leeftijdsgroepen die het meest bijdragen aan de stijging in levensverwachting komen in hoge mate overeen met de leeftijdsgroepen die het meest gevoelig zijn voor veranderingen in leeftijdsspecifieke sterfte. Bovendien verliep de sterftereductie op jongere leeftijd sneller, waardoor er sprake was van compressie van de mortaliteit in een korter leeftijdsinterval. In deel II wordt derhalve aangetoond dat een grotere levensverwachting en een grotere gelijkheid in de levensduur van individuen geen onverenigbare doeleinden zijn. Beide doelen kunnen worden bereikt door een reductie van vroegtijdige mortaliteit.

Samenvatting deel III

Deel III bevat de eerste grootschalige vergelijkende studies van de relatie tussen levensverwachting en variatie in levensduur. Zowel voor mannen als vrouwen in alle elf onderzochte Europese landen gold dat lager opgeleiden niet alleen gemiddeld op jongere leeftijd overleden, maar ook te lijden hadden onder een hogere levensduurvariatie, mits de betreffende personen de volwassen levensfase bereikten. De relatie tussen verschillen in opleiding en de variatie in levensduur was met name sterk in Oost-Europa. Bovendien was de gradiënt groter bij mannen dan bij vrouwen. De decomposities in hoofdstuk 5 maken duidelijk dat dit het gevolg was van een hogere sterfte tijdens de vroege volwassenheid (30-65 jaar) als gevolg van doodsoorzaken die zich vroeg kunnen voordoen, zoals sterfte door externe oorzaken en bepaalde vormen van kanker. In hoofdstuk 6 constateren we dat ongelijkheden in sterfte tussen groepen met een verschillend opleidingsniveau ongeveer 1 procent van de totale variatie in levensduur verklaren in West-Europa, tegen 2-10 procent in Oost-Europa. Het relatief grote aandeel van de tweede groep kan samenhangen met de onderzochte periode: over het algemeen genomen de jaren onmiddellijk voor en na de ineenstorting van de communistisch regimes en de overgang naar een markteconomie. In de nadere analyse (hoofdstuk 7) zien we dat ongelijkheden in sterfte tussen groepen met verschillende opleidingsniveaus sterk stegen in Estland en Litouwen in de jaren '90, ondanks dat deze ontwikkeling werd gematigd door een stijging van het opleidingsniveau van de bevolking als geheel. De studies in dit deel wijzen zonder uitzondering op een hogere onzekerheid met betrekking tot de sterfteleeftijd in de lagere sociaaleconomische groepen. We pleiten daarom voor de implementatie van beleid en interventies gericht op kwetsbare groepen, met name in de vroege volwassenheid.

Conclusies

Tot nu zijn de meeste mortaliteitsonderzoeken gericht geweest op gemiddelden, terwijl de spreiding rond dit gemiddelde grotendeels werd genegeerd. Dit proefschrift laat zien dat populaties en subgroepen aanzienlijk kunnen verschillen in de variatie in levensduur, zelfs als de levensverwachting gelijk is. Een reductie in levensduurvariatie verhoogt de waarde van langetermijninvesteringen in onderwijs, spaargelden en menselijk kapitaal, zowel op individueel als maatschappelijk niveau. Op dit moment worden verschillen in levensduur tussen bevolkingen en subgroepen met een verschillend opleidingsniveau vooral veroorzaakt door verschillen in

vroegtijdig overlijden. Dit suggereert dat er een aanzienlijk rol is weggelegd voor een beleid dat erop is gericht om deze verschillen weg te nemen en de gelijkheid in levensduur tussen verschillende individuen te verhogen.

We zouden de gewoonte om de volksgezondheid uitsluitend te beoordelen op het gemiddelde, de mediaan of de modus zelfs enigszins arbitrair kunnen noemen. Want waarom zou het gemiddelde eigenlijk het meeste genoemde (of zelfs het enige), allesbepalende statistische criterium voor de volksgezondheid moeten zijn? Als we een gemiddeld gezondheidsniveau belangrijk achten, moeten we ook de spreiding rond dit gemiddelde in kaart blijven brengen. Of we een bevolking gezond noemen, wordt immers niet alleen bepaald door de hoogte van de leeftijd die de leden van die bevolking gemiddeld bereiken, maar ook door de mate waarin die leeftijd binnen het bereik ligt van *alle* leden van die bevolking.

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About the Author

Alyson van Raalte was born on the 16th December 1977 in Ottawa, Canada. She obtained a Bachelor of Commerce from Queen's University in Kingston, Canada in 2000. After graduation she spent 5 years backpacking throughout parts of Asia, Africa and Europe funded by living as a working-holidaymaker in Japan, Scotland and France. She returned to her studies in 2005/6 to pursue a Masters of Science in Population and Development at the London School of Economics. In 2006/7 she attended the European Doctoral School of Demography at the Max Planck Institute for Demographic Research in Rostock, where she ended up staying in the laboratory of Survival and Longevity to complete her doctoral studies. In 2008 she began her collaboration with the Public Health department at Erasmus MC. Since October 2010 she has been working in the Life Course Dynamics and Demographic Change independent research group at the MPIDR.

PhD Portfolio Summary

Name PhD student: Alyson A. van Raalte

Erasmus MC Department: Public Health (MGZ)

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PhD period:
28 August 2006 – 31 July 2007 (European Doctoral School of Demography, funded by the Netherlands Interdisciplinary Demographic Institute (NIDI));
1 August 2007 – 15 October 2010 (PhD student, Max Planck Institute for Demographic Research (MPIDR))
1 May 2008 – 15 October 2010 (collaborations with Erasmus MC, official enrolment date 27 July 2010)

Promotors: Prof. dr. Johan P. Mackenbach, Prof. dr. James W. Vaupel

	Year	Workload (Hrs)	ECTS
Courses			
European Doctoral School of Demography (EDSD), European Association for Population Studies, Rostock			
Prep courses*			
• Measures and models of demography	2006	45	1.6
• Basic mathematics for demographers	2006	135	4.7
• Basic statistics for demographers	2006	98	3.4
• Computer programming for demographers	2006	45	1.6
Core courses			
• Theories of demographic behaviour and change	2006/7	350	10
• Statistical demography – Event history analysis	2006/7	350	10
• Population data and summary measures	2007	230	8
• Modeling, simulation and forecasting	2007	230	8
• Consequences of demographic	2007	230	8
• Mathematical demography	2007	230	8
• Research seminar - soft skills training	2006/7	230	8

Courses continued			
International Max Planck Research School for Demography (IMPRSD), MPIDR, Rostock[*]			
• Demographic methods for public health research	2007/8	120	4.2
• Perturbation analysis of longevity	2009	68	2.4
Erasmus Summer Program, Erasmus MC, Rotterdam			
• Methods for public health research (J. Mackenbach)	2008	15	0.7
Presentations at international conferences[†]			
• "Inter-individual inequality in lifespans: an examination of four methods and five countries", Canadian Population Society conference, Vancouver BC	2008	24	0.8
• "Quantifying lifespan variation: which measure to choose?", British Society for Population Studies Conference, Manchester	2008	18	0.6
• "Life expectancy and disparity", Population Association of America conference, Detroit MI	2009	22	0.8
• "Decomposing lifespan inequality by subgroup, illustrated on sex differences in mortality", Population Association of America conference, Detroit MI [Poster]	2009	..	..
• "How much do socioecomic inequalities contribute to individual lifespan disparities?", Intenational Union for the Scientific Study of Population conference, Marrakech	2009	40	1.4
• "The changing importance of educational inequalities to lifespan variation: Estonia and Lithuania examined over the 1990s", Population Association of America conference, Dallas Tx	2010	22	0.8
• "The contribution of inter-provincial inequalities to lifespan variation", Canadian Population Society conference, Montreal QC	2010	24	0.8
• "Lifetime uncertainty and socioeconomic inequality", European population conference, Vienna [Poster]	2010	22	0.8
Teaching activities[†]			
• Teaching assistant to J. Vaupel for course "Mortality theories", European Doctoral School of Demography, Paris	2008	40	1.6

[*] IMPRSD and EDSD prep courses are designed on the formula that each course requires four hours of work outside of the classroom for each hour of instruction. Formally, no ECTS credits were awarded, but equivalent credits were calculated on the basis of 0.035 credits per hour of workload (instruction + course work).

[†] The workload corresponds to the length of the conference or length of course. ECTS equivalent credits were calculated on the basis of 0.04 credits per hour of workload.

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