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**Certainty in Timing of Death:  
A New Analysis of Shifting Mortality and Life Span Disparity**

by

Sarah Marie Zureick

A dissertation submitted in partial satisfaction of the

requirements for the degree of

Doctor of Philosophy

in

Demography

in the

Graduate Division

of the

University of California, Berkeley

Committee in charge:

Professor Kenneth Wachter, Chair

Professor John Wilmoth

Professor S. Leonard Syme

Spring 2010

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Sarah Marie Zureick

## Abstract

Certainty in Timing of Death:  
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by

Sarah Marie Zureick

Doctor of Philosophy in Demography

University of California, Berkeley

Professor Kenneth Wachter, Chair

The human mortality experience has changed fundamentally as a result of the mortality transition. Not only are humans today living longer than their ancestors on average, they also experience greater certainty about the eventual timing of their death. This greater certainty is due to the considerable compression of the distribution of ages at death, which characterizes the mortality transition and results in lower life span disparity at the population level. In this dissertation, I investigate two key issues, which lie at the intersection of the mortality compression and mortality disparities literatures. First, I explore the recent transition from an era of mortality compression to a new era of mortality change, the shifting mortality era, in which life expectancy continues to increase but variability of age at death remains constant. Secondly, I examine differentials in life span disparity between the sexes and across countries over the course of the epidemiological transition. I adopt an age and cause-specific approach in analyzing trends in variability of age at death and differences in life span disparity across groups.

To account for age and cause-specific effects, I take advantage of recent methodological advances in demographic analysis and employ computationally based decomposition, perturbation, and simulation methods. Using an extensive collection of period life tables from the Human Mortality Database (HMD), I am able to explore aggregate level all-cause mortality change over the past one hundred twenty years in ten Western European countries. For some of my analyses, I take advantage of the full collection of historical mortality data available in the HMD, which includes populations in thirty-seven distinct geographic areas on five continents. Using a long series of cause-of-death data from France, which extends back to 1925, I am able to examine how sex-specific trends in variability of age at death were influenced by changes in age and cause-specific mortality during the epidemiological transition.

In documenting sex-specific trends in variability of age at death, I find that, similar to the emergence of the sex gap in life expectancy, a sex gap in variability of age at death

developed around the midpoint of the 20th century with females experiencing lower life span disparity in comparison to males. Using decomposition techniques, I demonstrate that the gap emerged because females experienced greater reductions in premature mortality in comparison to males. Cause-of-death decomposition results for France reveal that this is due to (1) declines in infectious disease, which young females had suffered disproportionately, and (2) male disadvantage in external cause mortality across the stages of the epidemiological transition. Examining cross-country differences in life span disparity between Sweden and the United States, I find that a gap in variability of age at death emerged between these two countries with Sweden exhibiting lower variability due to (1) Sweden gaining an advantage in premature mortality and (2) the United States gaining an advantage in old age mortality.

Using measures of variability of age at death, I also examine trends in mortality compression and possible explanations for the recent transition to an era of shifting mortality. To this end, I investigate how initial mortality conditions interact with the age pattern of mortality change to produce mortality compression, expansion, or shifting. I find that proportional mortality change that is fixed at rates unvarying across age does not necessarily lead to a parallel shift in the death distribution. I discover that certain initial mortality conditions are particularly primed for compression and that there is a significant change in the age-pattern of the sensitivity of measures of variability of age at death to proportional changes in age-specific mortality rates over the course of the mortality transition. I demonstrate that the potential for compression still exists in more developed countries despite the current shifting trends and that trends in cancer related mortality may play a particularly important role in determining future trends in variability of age at death.

For my grandmother, Evelyn Zureick, who inspired me to strive for academic excellence in all subjects, but most especially Latin.

*Mors certa, hora incerta.*

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# Chapter 1

## Introduction

Reviewing the historical mortality experience of human beings is a grim endeavor. For most of history, humans experienced relatively short average life times, with many not living past infancy, and high variability of age at death. Fortunately, focusing on human mortality in the modern era is not quite so bleak. While we are still bound by the chains of mortality, due to the considerable progress made against premature mortality in recent history, we now enjoy greater longevity and increased certainty about the timing of our deaths. In this dissertation, I focus on this latter advantage: greater certainty in timing of death.

With the progress against infectious disease that occurred during the epidemiological transition, many forms of premature mortality have been substantially reduced. Figure 1.1 depicts the death distributions corresponding to period life tables in Sweden for the total population at the beginning of the 20th century, in the middle of the 20th century, and at the beginning of the 21st century. Between the first two time points, the proportion of deaths occurring in infancy, childhood, and young adulthood decreased substantially. This led to both an increase in the mean age of death and reduced spread in the death distribution (i.e. lower variability of age at death). This phenomenon is referred to as mortality compression.

In this dissertation, I focus on compression in the adult death distribution thus focusing on the shape of the death distribution above age ten as illustrated in Figure 1.1 (n.b. the death distributions depicted include deaths at all ages but measures are based upon standardized death distributions that only includes deaths above age ten). The solid vertical lines in this figure document the mean age at death for members of the synthetic cohort dying above age ten,  $M_{10}$ , and the solid horizontal lines reflect the standard deviation of ages at death above age ten,  $S_{10}$ , on either side of the mean. As depicted in this figure, between 1900 and 1950,  $M_{10}$  increased substantially from 64 years to 74 years while  $S_{10}$  declined from 21 years to 14 years.

In 1980, Fries claimed that mortality compression would continue in the latter half of the twentieth century as continued progress against disease would move deaths into older ages but biological limits to life span would constrain movement in the upper tail of the death distribution (Fries, 1980). As Figure 1.1 demonstrates, Fries's predictions did not

come to pass. Between the middle of the 20th century and the beginning of the 21st century, the spread of the distribution of adult ages at death as measured by  $S_{10}$  remains relatively constant, around 13 to 14 years, while  $M_{10}$  continues to increase from 74 years to 81 years. Instead of exhibiting continued mortality compression between the middle and end of the twentieth century, the Swedish adult death distribution seems to “shift” to higher ages with the mean of the distribution increasing but no reduction in the spread of the distribution. Demographers describe this change as a transition from an era of mortality compression to an era of shifting mortality and take it as evidence that humans are not approaching a biological limit to life span (Wilmoth and Horiuchi, 1999; Cheung and Robine, 2007).

As illustrated in Figure 1.2, the transition from mortality compression to shifting mortality occurred in a number of more developed countries around the midpoint of the 20th century. At this point, life expectancy continues to increase but declines in variability of age at death, as measured by the standard deviation of ages at death above age 10 ( $S_{10}$ ), taper off. While this transition has been documented using a variety of measures of variability of age at death and the implications of this trend have been explored, the reasons for the both the development and continuation of the phenomenon have yet to be fully detailed. In this dissertation, I make a contribution to this literature by identifying the changes in age and cause-specific mortality responsible for the transition from mortality compression to shifting mortality.

I also tie my work into a more recent strain of this literature, in which measures of variability of age at death have been recast as measures of mortality disparity. In this context, measures of variability of age at death are referred to as life span disparity measures. Reductions in life span disparity are thought to be advantageous both because they lead to greater certainty in timing of death and a more equitable distribution of deaths (Edwards and Tuljapurkar, 2005). Using the same age and cause-specific approach that I use to study the transition from mortality compression to shifting mortality, I analyze the differentials in variability of age at death observed between the sexes and across countries. In particular, I demonstrate why certain groups experienced more rapid declines in variability of age at death during the era of mortality compression and thus experience lower variability of age at death in era of shifting mortality.

Understanding trends in variability of age at death and differentials in these measures across groups is more complicated than understanding trends and differentials in life expectancy. While mortality improvements at any age act to increase life expectancy, the effect of mortality change on measures variability of age at death varies by age. At the oldest ages, mortality improvements increase variability of age at death as deaths are pushed out further on the tail of the death distribution. On the other hand, mortality improvements at the youngest ages tend to decrease variability of age at death as they take deaths away from the left hand tail of the death distribution and concentrate deaths at later ages. Both of these scenarios are illustrated in Figure 1.3. For one measure of life span disparity,  $e^\dagger$ , Zhang and Vaupel have documented that there exists a single age, referred to as the crossover age, at which change in mortality has no effect on this measure (Zhang and Vaupel, 2009). As

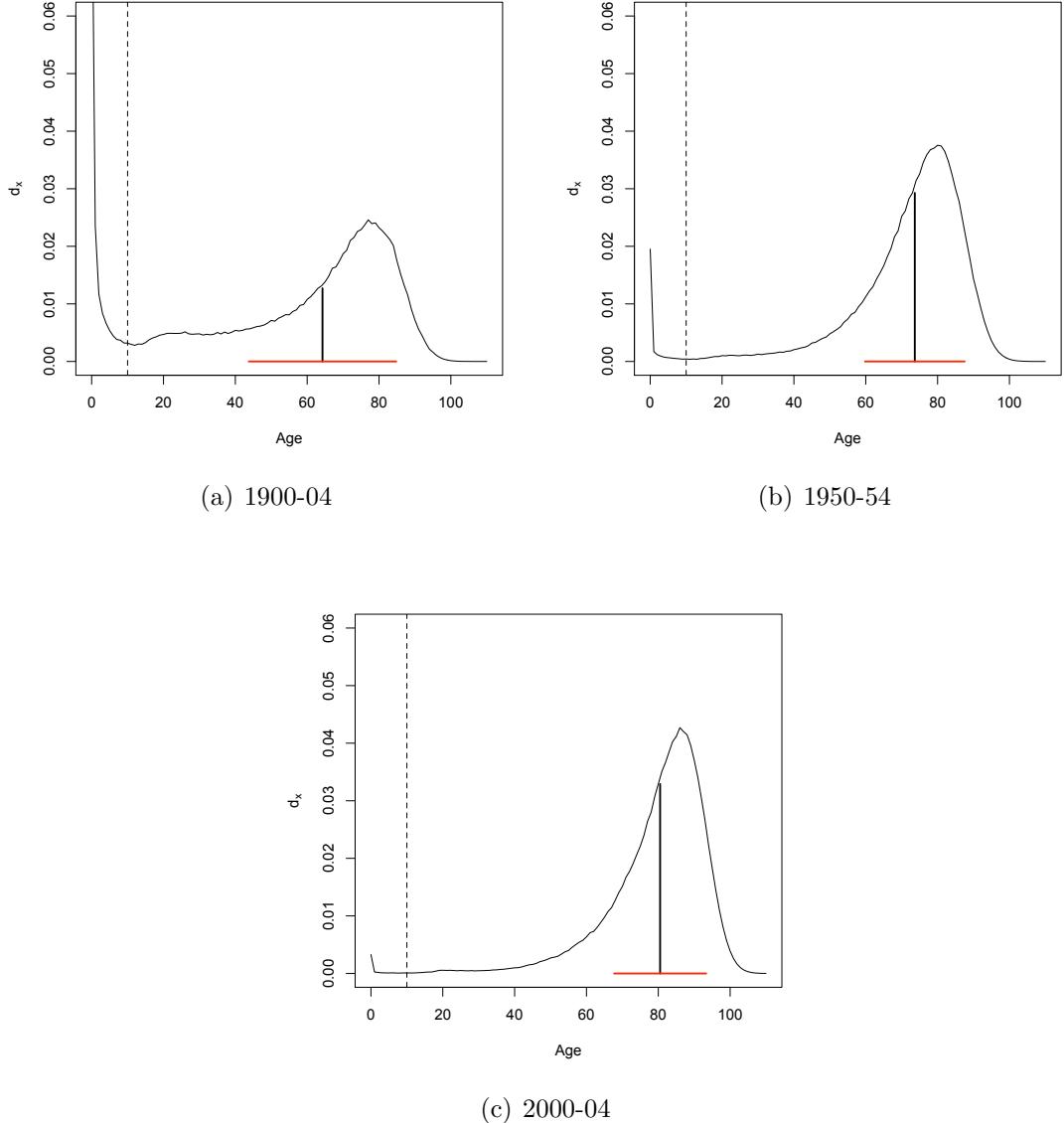


Figure 1.1: Swedish death distributions based on period life tables, total population. Solid vertical lines indicate mean age at death above age 10,  $M_{10}$ . Solid horizontal lines depict standard deviation of ages at death above age 10,  $S_{10}$ , on either side of the mean. Data source: Human Mortality Database (HMD, 2009).

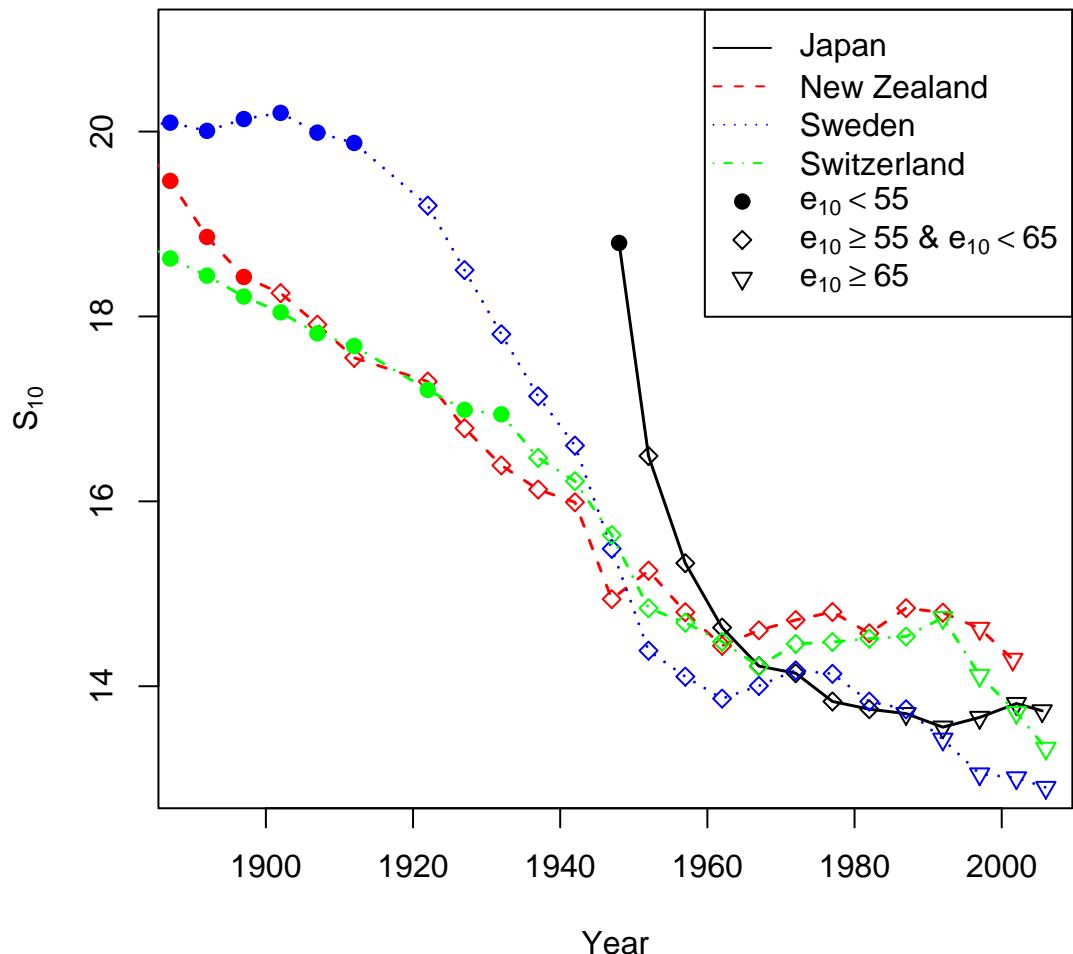


Figure 1.2: Relationship between  $e_{10}$  and  $S_{10}$ . Trends in  $S_{10}$  over time. Male population.  
Data source: Human Mortality Database (HMD, 2009).

will be illustrated in the decomposition analyses that I present in later chapters, a crossover age is often observed for the measure of variability of age at death, which I use,  $S_{10}$ .

Before presenting my own results, I offer a review of the literature in this area. In Chapter 2, I provide a description of the epidemiological transition and chronicle the study of mortality and morbidity compression. I also offer an overview of the more traditional studies of mortality disparities which examine differences in survival among groups stratified by some criteria (e.g. sex, race, socioeconomic status, etc.). I then review a comparatively smaller literature in which measures of variability of age at death or life span disparity have been used to quantify inter-individual mortality disparities cross nationally and across subpopulations stratified along typical social dimensions. A subset of these studies include attempts to quantify the contribution of social inequality to disparities in mortality observed at the population level.

In Chapter 3, I place the issue of trends and differentials in variability of age at death within a broader context by exploring the implications of mortality compression over the course of the mortality transition from a biological, economic, psychological, and demographic perspective. I give particular attention to how changes in certainty about timing of death impact another key demographic process: fertility.

I provide greater details on the data, measure, and methods that I use the dissertation in Chapter 4. The complications introduced by the existence of a crossover age necessitate examining trends and differentials in variability of age at death using an age-specific approach. In this dissertation, I take advantage of recent methodological advances in demographic analysis and employ decomposition and perturbation techniques in order to examine the effects of age and cause-specific mortality rates on measures of variability of age at death. Using an extensive collection of period life tables from the Human Mortality Database (HMD), I explore aggregate level all-cause mortality change over the past one hundred twenty years in ten Western European countries. For some of my analyses, I take advantage of the full collection of historical mortality data available in the HMD, which includes populations in thirty-seven distinct geographic areas on five continents. Using a long series of cause-of-death data from France, which extends back to 1925, I examine how sex-specific trends in variability of age at death were influenced by changes in age and cause-specific mortality over the course of the epidemiological transition.

In Chapter 5, I examine the relationship between the age pattern of mortality change and trends in variability of age at death in order to evaluate theoretically whether shifting mortality is an inevitable outcome of the mortality transition. Through simulation exercises, I test whether proportional change in mortality over time at rates unvarying across age necessarily leads to a shift in the death distribution. The results of the simulation exercises along with results of perturbation analysis lend insight of how initial mortality conditions and the age pattern of mortality change interact to produce mortality compression, expansion, or shifting. Importantly, the results of the perturbation analysis suggest that the potential for changes in age-specific mortality to affect variability of age at death varies substantially over the course of the epidemiological transition.

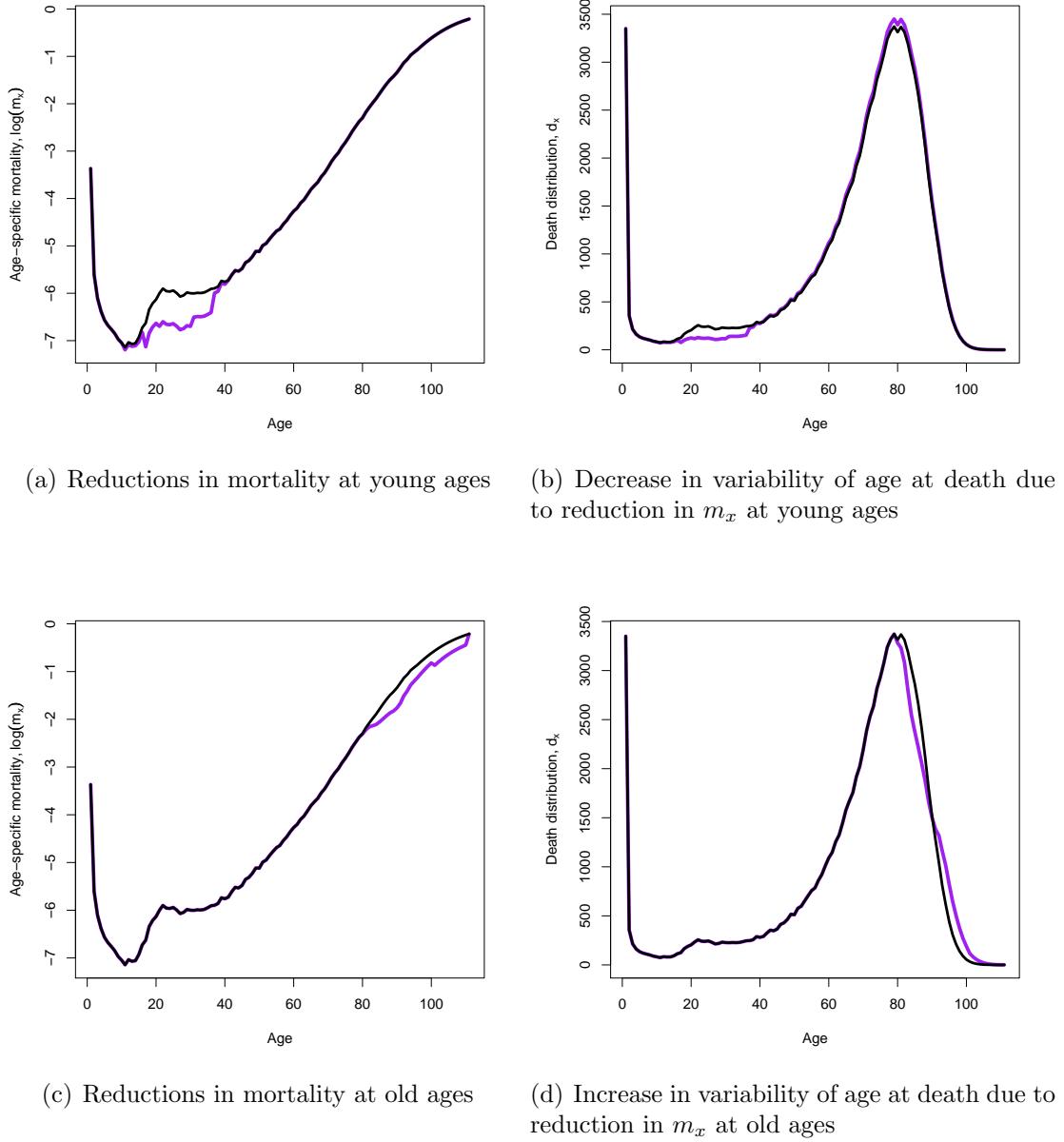


Figure 1.3: Hypothetical effects of changes in age-specific mortality on variability of age at death. Initial  $m_x$  and  $d_x$  from Sweden 1940-44, total population. Data source: Human Mortality Database (HMD, 2009).

The theoretical analysis presented in Chapter 5 is complemented by an analysis of actual trends in variability of age at death using decomposition methods in Chapter 6. Using decomposition techniques, I quantify the contribution of changes in age-specific mortality to changes in variability of age at death in the eras of mortality compression and shifting mortality. Comparing my results to those obtained in the sensitivity analysis in Chapter 5 demonstrates how divergence in the age pattern of mortality change is playing a role in the current shifting scenario being observed in more developed countries. In addition to examining the total effect of changes in age-specific mortality rates on measures of variability of age at death, I also examine the two pathways through which changes in age-specific mortality,  $m_x$ , affect the shape of the death distribution ( $d_x = l_x m_x$ )-directly through the  $m_x$  term and through the survivorship function,  $l_x$ .

I take up the question of differentials in variability of age at death across groups in Chapter 7. In the first part of this chapter, I examine the emergence of the sex gap in variability of age at death during the course of the epidemiological transition. I identify the changes in all-cause age-specific mortality that produced greater decline in variability of age at death for females relative to males in the era of mortality compression. In the second part of the chapter, I investigate cross-country differences in variability of age at death. Specifically, I identify reasons why the United States currently exhibits higher levels of variability of age at death in comparison to the levels of variability observed in Sweden. The different nature of gender differentials in variability of age at death versus cross-country differentials between Sweden and the United States prompts a discussion of whether lower variability of age at death is necessarily advantageous.

In Chapter 8, I examine the transition from mortality compression to shifting mortality and gender differentials in variability of age at death from a cause-of-death perspective. I focus on sex-specific trends in variability of age at death in France and investigate the emergence of the gender gap in variability of age at death during the era of mortality compression and a more subtle widening of the gap in variability of age at death between males and females in the second half of the twentieth century during the era of shifting mortality.

Through my reviews of the current literature in Chapters 2 and 3, I provide justification for the relevance of life span disparity measures and explore the connection between social inequality in mortality and disparities in mortality observed among individuals at the population level. My goals in my own work, which I present in Chapters 5, 6, 7, 8, are to understand better the transition from mortality compression to shifting mortality and to explain differentials in life span disparity observed among groups.

# Chapter 2

## Literature Review

The human mortality experience has changed fundamentally as a result of the mortality transition. Not only are humans living longer, they are also much more certain about the eventual timing of their death. The major focus of this dissertation project is this latter advantage: greater certainty in timing of death. Traditionally, certainty in timing of death has been studied in the context of mortality compression; however, traditional measures of mortality compression have recently been recast as measures of life span disparity. These measures provide an indication of the level of mortality inequality in a particular population by measuring how spread out deaths are across age. In my dissertation research, I exploit the dual nature of measures of variability of age at death as an indicator of both mortality compression and mortality inequality. Thus, my review of the literature focuses broadly on these two topics.

I begin my review by defining terms that I will use throughout the dissertation and offering an overview of the multitude of measures available to assess mortality compression and mortality inequality. Then, I proceed to chronicle the study of mortality compression. The starting point for this section is evidence of the significant narrowing of the spread of ages at death at the population level throughout the late 19th and early 20th century in countries undergoing the demographic transition. While there is no doubt that mortality improvement over the course of the demographic transition has resulted in significant mortality compression, there are debates on whether these improvements in life expectancy have been accompanied by a compression or expansion of morbidity. It is also unclear whether improvements in mortality in more recent years have been accompanied by continued declines in variability of age at death or stability in these measures. The latter phenomenon, referred to as shifting mortality, has been heralded as the next stage of the mortality transition (Robine, 2001). A thorough review of the literature concerning mortality compression and shifting mortality is provided as context for Chapters 5, 6, and 8.

In the latter sections of this review, I focus on the literature concerning mortality inequality. I offer an overview of the more traditional studies of mortality inequality which examine differences in survival among groups pre-defined according to some criteria (e.g.

gender, race, socioeconomic status, etc.). I then review the comparatively smaller literature in which measures of life span disparity have been used to compare differences in inter-individual mortality inequality cross-nationally and across subpopulations stratified along typical social dimensions (i.e. race and educational attainment) (Edwards and Tuljapurkar, 2005; van Raalte et al., 2009; Shkolnikov et al., 2003; Smits and Monden, 2009; Weden, 2007). These comparative studies examine the disparities in life span disparity maintaining that greater life span disparity is detrimental because it indicates greater uncertainty about the eventual timing of death and a more inequitable distribution of deaths. This part of the review provides background information for Chapters 7 and 8, in which I examine differences in mortality inequality by sex and the emergence of the current gender gap in variability of age at death. I also review a few studies that have sought to quantify the contribution of social inequality to the inequality in mortality observed at the population level. These studies reveal that social inequality does not explain much of the total disparity in mortality observed among individuals within a population. In the next section, I define the terms that I will use throughout the dissertation and offer an overview of measures that have been developed to study mortality compression and mortality inequality.

## 2.1 Measures of mortality compression and indicators of mortality inequality

The *demographic transition* and related *epidemiological transition* motivate the study of mortality compression. Demographic transition theory describes the historical phenomenon wherein a population transitions from a demographic regime of high fertility and high mortality to a regime of low fertility and low mortality (Notestein, 1945; Davis, 1963). In the classical model of demographic transition, mortality decline precedes fertility decline, and this delayed response produces rapid population growth.

The term epidemiological transition was introduced by Omran in 1971, and it characterizes the changes in mortality observed over the course of the demographic transition (n.b. the terms epidemiological transition and *mortality transition* can be used interchangeably (Caldwell, 2001)). Specifically, epidemiological transition theory describes the changes in age and cause-specific mortality observed over the recent course of human history (Robine, 2003). Omran described three distinct mortality regimes that are observed during the transition: (1) *The Age of Pestilence and Famine* characterized by high and fluctuating mortality; (2) *The Age of Receding Pandemics* characterized by sustained and progressively steeper declines in mortality, which are less often disrupted by epidemics; (3) *The Age of Man-Made Diseases* characterized by an eventual stabilization of mortality rates at a low level with most deaths attributable to chronic and degenerative diseases (Omran, 1971). The first stage characterizes most of human history, and reflects the uncertainty associated with life time durations prior to the transition. During the second stage, the transition occurs, and

mortality becomes increasingly concentrated in later life as deaths due to infectious diseases are eliminated. Omran predicted that limited progress would be made against mortality following the containment of most infectious disease hence the third stage is characterized by a stabilization in mortality rates (Omran, 1971).

In contradiction to Omran's predictions, with improvements in data collection on mortality at the oldest ages, researchers have been able to document that mortality rates are decreasing even at the oldest ages, that the tail of the survival curve has been shifting upward to older ages, and that maximum life duration is increasing (Kannisto et al., 1994; Wilmoth and Lundström, 1996; Cheung and Robine, 2007). In response to increases in life expectancy mainly due to declines in cardiovascular mortality, Olshansky and Ault proposed a fourth stage of the epidemiological transition, *The Age of Delayed Degenerative Diseases* characterized by declines in mortality due to chronic and degenerative diseases at older ages (Olshansky and Ault, 1986; Robine, 2003). Based on his study of trends in mortality compression, which do not offer much evidence for Omran's third stage, Robine suggests adopting Omran's depictions of the first and second stages of the transition, but describing the third stage in a manner similar to Olshansky and Ault's fourth stage. Robine refers to this stage as *The Age of the Conquest of the Extent of Life*, and in this stage, mortality decline at older ages is more important than at younger ages for increases in life expectancy. Importantly, Robine suggests that during this stage further increases in life expectancy are not necessarily accompanied by continued mortality compression (Robine, 2001).

### 2.1.1 Mortality compression: definition and measures

In his review of measures of mortality compression, Kannisto defines *mortality compression* as occurring when a given proportion of deaths take place in a shorter age interval than before (Kannisto, 2000). With this definition, trends in mortality compression can most easily be observed by examining changes in standardized death distributions,  $d_x$ , using series of either cohort or period life tables. For example, Figure 2.1(a) displays changes in the death distribution for the Swedish population from 1900-2004. Clearly, according to Kannisto's definition, mortality compression occurs over this time period as deaths become increasingly concentrated around the *modal age at death* at older ages (i.e. the age at which the maximum number of deaths occur excluding deaths in infancy and childhood). An alternate way of viewing trends in mortality compression, which is entirely complimentary, is examining changes in the survivorship column,  $l_x$ , across cohort or period life tables. From this perspective, trends in mortality compression are assessed by measuring the degree of *rectangularization* of the survival curve. As seen in Figure 2.1(b), in Sweden, during the period 1900-2004, the survival curve becomes increasingly rectangular over time as more members of the (synthetic) cohort survive to older ages.

The mortality compression that occurred over the course of the mortality transition can easily be assessed graphically by examining trends in the distribution of deaths or the survival curve; however, in order to detect more subtle changes in the death distribution in

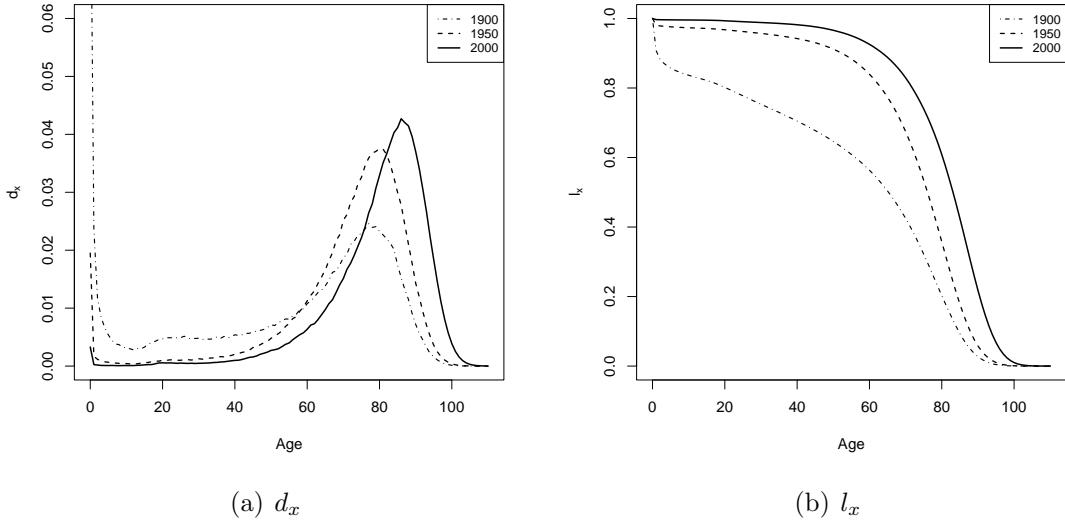


Figure 2.1: Changes in Swedish period death distributions,  $d_x$ , and survivorship,  $l_x$ , 1900-2004. Data source: Human Mortality Database (HMD, 2009).

more recent years it is helpful to utilize quantitative measures of compression. Both Wilmoth and Horiuchi as well as Cheung et al. have provided overviews of the measures available to assess mortality compression and rectangularization of the survival curve (Wilmoth and Horiuchi, 1999; Cheung et al., 2005). The measures which they have reviewed along with measures popularized since these reviews were conducted (including life expectancy lost due to premature death,  $e^\dagger$ ), are provided in Table 2.1.

In their review of measures used to assess the rectangularization of the survival curve, Cheung et al. consider three components of rectangularization: (1) horizontalization-“how long a cohort can live and how many cohort members survive before aging related deaths decrease the proportion of survivors”, (2) verticalization-“how concentrated aging-related deaths are around the modal age at death”, and (3) longevity extension-“how far the right-hand tail, representing the highest normal life durations, can exceed the modal age at death.” (Cheung et al., 2005, p. 246). The authors classify existing measures used to assess rectangularization into the following categories: central longevity indicators, horizontalization indicators, concentration and/or verticalization indicators, rectangularization indicators, maximum longevity indicators, mapping indicators, and other indicators (Cheung et al., 2005). The measures that I will utilize in my analysis largely fall into the concentration and/or verticalization category. The majority of measures included in this category capture some type of variability in the death distribution. Differences among these measures of variability of age at death arise because of differences in the age range covered by the measures, differences in the central indicator used in the calculation of dispersion (mean versus mode), and

whether or not the measurement is attached to fixed percentiles of the death distribution. In particular, I focus on the following measures. More detail on how these measures are calculated is provided in Chapter 4.2.

- $IQR$ -inter-quartile range, age span between the 25th and 75th percentiles of the cumulative (Wilmoth and Horiuchi, 1999).
- $C_{50}$ -age span corresponding to the most compressed 50 percent of deaths from the death distribution (Kannisto, 2000).
- $e^\dagger$ -person years lost in the life table due to early death (Zhang and Vaupel, 2009).
- $S_0$ -standard deviation of ages at death unconstrained by age (Edwards and Tuljapurkar, 2005).
- $S_{10}$ -standard deviation of ages at death above age ten (Edwards and Tuljapurkar, 2005).
- $SDM$ -root mean square deviation around the modal age at death (Canudas-Romo, 2008)
- $SD(M+)$ -root mean square above the modal age at death (Kannisto, 2000; Kannisto, 2001; Cheung and Robine, 2007)
- $\bar{H}$ -a measure often called “life table entropy” (Keyfitz and Caswell, 2005).

The impetus for the Cheung et al article is that although there has been much discussion about rectangularization of the survival curve within the literature in recent years there has not been any consensus on how to measure rectangularization (Cheung et al., 2005). In terms of criteria for evaluating measures of compression, Kannisto, who has written extensively on this subject, states that measures should not rely upon an age scale although measures could ignore infant and child mortality because the theory of compression is more focused on later life. Also, he suggests that measure can either utilize percentiles of the death distribution (e.g.  $IQR$ ) or float freely measuring compression wherever it is occurring in the death distribution (e.g.  $C$ -measures) (Kannisto, 2000). In their review of ten measures of mortality compression, Wilmoth and Horiuchi note the high degree of correlation among the measures that they review (Wilmoth and Horiuchi, 1999). Despite the high degree of correlation among different measures, as will be demonstrated in the review of the literature on mortality compression and shifting mortality, there has been much disagreement on trends in compression resulting from differences in measurement. Shkolnikov et al. have also demonstrated that different measures of mortality compression or mortality inequality can offer different interpretations of historical changes in mortality with measures even changing in opposite directions (Shkolnikov et al., 2003).

### 2.1.2 Mortality inequality: definition and measures

Inequality in mortality can be evaluated either by examining variation in mortality across individuals within a population or by focusing on differences in mortality across predefined groups within a population. The first approach is advocated as the ideal approach by Gakidou et al, whom define *health inequality* “to be variations in health status across individuals in a population” (Gakidou et al., 2000, p. 42); however, studies of mortality differentials tend to fall into the latter category.

There are a number of measures and methods that can be used to examine mortality differentials across subgroups. The standard demographic approach involves comparing either standardized or unstandardized age-specific mortality rates across groups classified along basic demographic dimensions such as sex and race/ethnicity. Beyond these basic demographic classifications, studies of mortality differentials focus on disparities along social dimensions such as socioeconomic status, marital status, religious affiliation, nativity, etc. (Hummer et al., 1998). Studies of differentials in mortality may also utilize a single composite measure of mortality such as life expectancy (either from birth or conditional on obtaining a certain age). For instance, recent cross-national comparisons of female mortality have focused differential trends in life expectancy above age 65 (Meslé and Vallin, 2006). Statistical methods such as hazard analysis can be used to quantify mortality differentials across groups while controlling for confounding variables (Rogers et al., 2000). Evidence from the literature supporting the ubiquity and degree of social disparities in mortality is presented in Section 2.4.

The methods of measuring mortality inequality, which I will focus on in my dissertation, fall into the category of measures, which quantify mortality inequality by examining differences in mortality among individuals rather than across predefined groups. I refer to these measures as inter-individual measures of mortality inequality. These measures seem to fit Gakidou et al’s criteria of an “ideal” measure because they quantify inequality by examining differences in mortality either among individuals or between the individual and the average outcome; however, these authors also advise that measures of health inequality should capture differences in risk rather than outcomes in order to remove variation due to chance (i.e. health expectancy rather than healthy life span) (Gakidou et al., 2000). Since it is not possible to observe mortality risk only mortality outcomes, Gakidou et al’s “ideal” measure can not be measured directly although efforts have been made to try to capture variation in risk using statistical models (Gakidou and King, 2002). Edwards and Tuljapurkar argue, however, that this underlying uncertainty is meaningful both because it a major contributor to the total variability in mortality outcomes and because this uncertainty differs across social groups (Edwards and Tuljapurkar, 2005).

In terms of using measures of variability of death to assess mortality inequality, the measures of variability of age at death discussed in the section on mortality compression can also be used to assess inequality in mortality along with mortality compression. Other measures, more traditionally associated with studies of income inequality, the Gini index

and the Theil index, can also be used to examine disparities in life span (Shkolnikov et al., 2003; Peltzman, 2009; van Raalte et al., 2009; Smits and Monden, 2009)

As a final note, throughout the dissertation, in my discussions of mortality inequality, either inter-individual or across groups, I use the term *inequality* to indicate differential outcomes. In some cases, these differential outcomes are the result of differential access to care and other structural inequalities; however, I do not distinguish inequality due to these factors from other processes that produce heterogeneity in survival such as biological differences.

## 2.2 Mortality compression and the rectangularization of the survival curve

Much attention has focused on documenting trends in mortality compression in response to Fries's proposal of the rectangularization hypothesis, which holds that human mortality and morbidity will become increasingly compressed as humans approach a fixed biological limit to life span (Fries, 1980). Fries based this claim on two assumptions: (1) maximum attainable longevity is fixed and death without disease is possible and (2) it is possible to delay morbidity and senescence. Fries cited a lack of centenarians as empirical evidence of a biological limit to human life span. While Fries is the most famous proponent of the rectangularization hypothesis, earlier authors speculated on this phenomenon (Comfort, 1979; Pearl, 1940; Upton, 1977).

In his 1980 paper, Fries predicted a maximum mean age at death of 85 years by extrapolating linear trends in life expectancy at different ages (i.e.  $e_{65}$ ,  $e_{25}$ , and  $e_0$ ) based on rates of progress in these measures during the the 20th century and examining the point of intersection of these trend lines. In extrapolating linear trends, Fries ignored entropy in the life table and, in extrapolating trends in  $e_{65}$ , likely failed to account for the more rapid improvements in mortality at older ages that would be necessary to sustain linear increase in  $e_0$  following substantial reductions in premature mortality. Fries described the ideal death distribution as a normal distribution with its mean centered at age 85 and with 66% of deaths occurring between 81 to 89 years old and 95% percent of deaths between ages 77 and 93. The calculation of percentiles is possible because the tail of distribution is assumed fixed with age 100 about 4 standard deviations away from the mean. The ideal survival curve is not completely rectangular due to some violent and traumatic deaths.

While Fries emphasized the plasticity of aging and the importance of preventative care for averting chronic disease, he saw these as a way to compress morbidity and mortality into an increasingly narrow age interval. Since Fries viewed human longevity as being subject to fixed biological limits, he thought investment in health care beyond the ideal age inadvisable. In his own words, "high-level medical technology applied at the end of natural life span epitomizes the absurd. The hospice becomes more attractive than the hospital" (Fries, 1980).

Fries' article inspired much research, and since the time of his writing, many scholars have obtained results contradicting both his predictions and assumptions. Robine et al. have demonstrated the fallacy of Fries's more narrow predictions of maximum average life span and related variability as Japanese females have achieved life expectancy higher than 85 years with corresponding variability of age at death much wider than what Fries predicted (Robine, 2008). Other researchers have challenged the central assumption on which the rectangularization hypothesis rests: that humans are fast approaching a biologically fixed limit to life span. For instance, Wilmoth and Lundström show that the upper tail of the death distribution for Sweden has been shifting steadily upward for over a century suggesting that the upper limit to human longevity is malleable (Wilmoth and Lundström, 1996). Kannisto et al. demonstrate that mortality rates have been declining at the oldest ages (above age 80) in developed countries over the course of the 20th century and that the pace of decline has increased in recent decades (Kannisto et al., 1994). In recent work on Japan, Cheung and Robine show that the maximum life duration has been increasing consistent with increases in maximum reported age at death and that these increases are not solely the result of increases in population, which make it more likely to observe an extreme age at death (Cheung and Robine, 2007).

In an early work, using data from the United States for the period 1900-1980, Myers and Manton investigated whether mortality trends indicated mortality compression consistent with Fries's hypothesis (Myers and Manton, 1984a). The authors acknowledged that survival curves appeared to be more rectangular over time, but they also cautioned that one can not distinguish between improvements at younger versus older ages when assessing rectangularization just by graphically assessing survival curves. Focusing on those who survive to age 60, the authors demonstrated that the standard deviation in ages at death above age 60 was increasing offering evidence that rectangularization was not occurring. In response to Fries's criticism that their method of truncating the death distribution by age was invalid, the authors carried out Fries's suggestion to look at a fixed percentile of the distribution (Myers and Manton, 1984b). Specifically, the authors looked at the last 25, 33.3, 50, 66.6, and 75 percent of deaths examining trends in the mean and standard deviation. The authors observed that while the mean increased steadily during their period of observation 1962-1979, the standard deviation did not decrease as much. For some fixed percentiles (66.6% for men, 25% for women), the decline in the standard deviation was negligible. The authors concluded from this analysis that the negligible changes in standard deviation did not indicate an approach to a biological limit to life span.

The debates between Fries and Myers and Manton inspired continued investigation of the effect of cutoff ages on measures of dispersion. Nusselder and Mackenbach demonstrate that different results can be obtained depending on the cutoff age used in the calculation (Nusselder and Mackenbach, 1996). In a detailed analysis using data from France covering the period 1890-94 to 1990-94, Robine shows that the relationship between change in the standard deviation over time and change in life expectancy depends on the cut off age used (Robine, 2001). If the cut off age is between 0 and 20, increasing life expectancy over time

appears to be accompanied by decreasing dispersion as the standard deviation decreases. On the other hand, if the cut off age is between 60 and 80, increase in life expectancy above these ages over time is also accompanied by increases in dispersion. There appear to be two equilibrium points on a graph of cut off age versus standard deviation where lines corresponding to periods are plotted. The equilibrium points occur around age 50 for males, age 60 for females and then around age 95 for both sexes. From his own analysis, Robine concludes that “only a calculation which includes all life spans or deaths is beyond criticism” (Robine, 2001).

Other evidence indicates continued mortality compression during the latter half of the 20th century. Examining 13 countries with good data included in the Kannisto-Thatcher oldest-old data base during the period 1960-1995, Kannisto finds that while  $e_{80}$  and  $M$  are generally increasing over this period  $SD(M+)$  and  $e(M)$  are declining indicating continued mortality compression (Kannisto, 2001). In addition, in an earlier paper, Kannisto demonstrates that, in contrast to Wilmoth and Horiuchi’s finding that trends in  $IQR$  stabilized in the latter half of the 20th century, trends in the measure  $C50$  were still showing decline (Kannisto, 2000). Debates about the possible transition to an era of shifting mortality, where variability of age at death remains constant even as life expectancy continues to increase, are the focus of the next section.

## 2.3 Shifting mortality

A number of researchers have documented the emergence of shifting mortality; however, the timing of the emergence of shifting mortality and the specific geographic areas where shifting mortality has been observed vary depending on the measure of mortality compression used in the analysis. Trends in the measures  $IQR$ ,  $C50$ ,  $S_{10}$ ,  $SDM$ , and  $SD(M+)$  over course the 20th century in France are shown in Figure 2.2. As can be seen in this figure, these different measures offer different interpretations of the history of mortality compression in France. Relative to the massive compression of mortality indicated by trends in  $IQR$  during the period 1900-1950, the trends in  $IQR$  in the second half of the century seem to indicate a transition to shifting mortality as Wilmoth and Horiuchi also observed in Sweden (Wilmoth and Horiuchi, 1999). Kannisto and Robine have argued that  $IQR$  over exaggerates the pace of mortality compression because it sometimes measures the space between the two modes in the death distribution rather than the concentration of deaths in one or the other. These authors have shown that documenting trends in variability of age at death using the measure  $C50$  rather than  $IQR$  reveals that compression was still occurring in a number of countries throughout the latter half of the twentieth century (Kannisto, 2000; Robine, 2001).

Consistent with the assessment of Edwards and Tuljapurkar of trends in  $S_{10}$  in industrialized countries, trends in this measure for France indicate stagnation since the 1950’s (Edwards and Tuljapurkar, 2005). In contrast to the trends in  $S_{10}$ , trends in  $SDM$ ,  $S_0$ , and  $e^\dagger$  indicate continued compression during the second half of the twentieth century reflecting

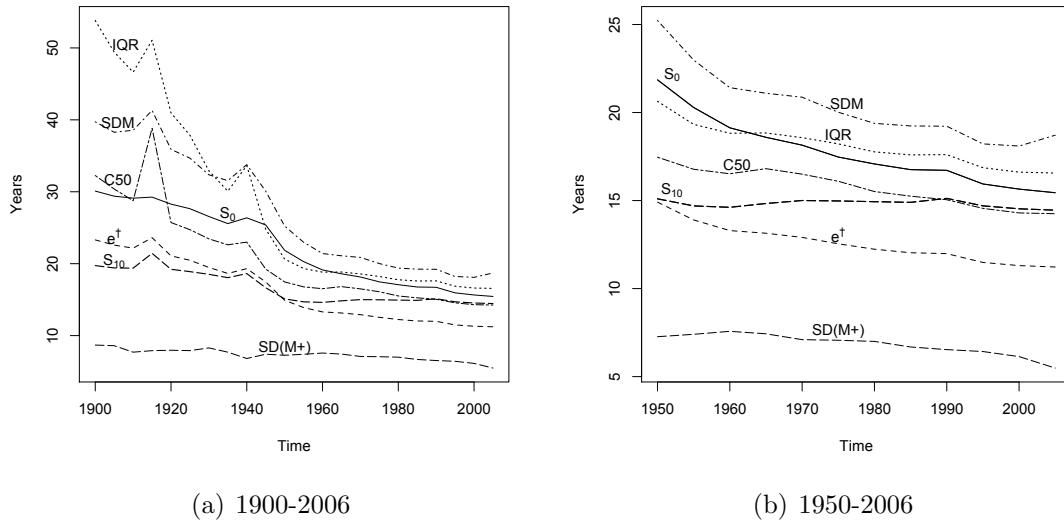


Figure 2.2: Trends in a variety of measures of mortality compression, France. Data source: Human Mortality Database (HMD, 2009).

gains made against infant mortality. Unlike the other measures,  $SD(M+)$  does not begin steadily declining in France until the latter half of the twentieth century.

While these different measures produce different interpretations of the geographic coverage and timing of the transition to shifting mortality, the authors utilizing these different measures to study trends in mortality compression cite common sources as the originators of the concept of shifting mortality. This concept was first introduced by Kannisto in his study of trends in old age mortality. One measures the “the age shift in mortality” introduced by Kannisto by finding the difference between the ages that correspond to the same mortality level between two time points. For instance, in the example given by Kannisto,  ${}_1q_{85}$  for Austrian females in the period 1950-1960 is 0.1721. To find the age shift in mortality from 1950-1960 to 1960-1970, one examines the probabilities of dying by age,  ${}_1q_x$ , in order to identify the age  $x$  where  ${}_1q_x = 0.1721$ . This occurs at  $x = 85.96$ . Thus, the age shift in mortality is 0.96 years (Kannisto, 1996).

As will be illustrated in Chapter 5, even if all age-specific mortality rates shifted across age .96 years between these two time periods, the result would not necessarily imply shifting mortality when using the measures of mortality compression described above. The translation of a shift in mortality rates to a shift in the death distribution requires special initial mortality conditions. For instance, if the age-specific mortality curve can be described as a Gompertz curve, a proportional decrease in mortality rates fixed across age and time corresponds exactly to a shift in mortality rates across age. Under these circumstances, the death distribution retains its shape while shifting to higher ages, and thus the measures of mor-

tality compression described above all exhibit constant trends. This ideal shifting mortality scenario is illustrated in Figure 2.3.

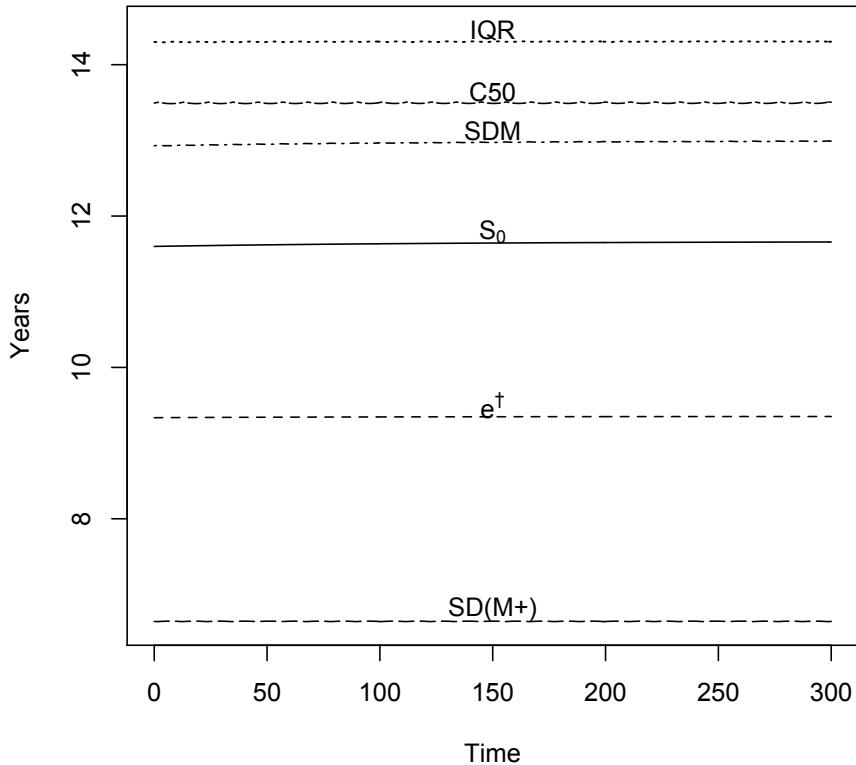


Figure 2.3: Trends in measures of mortality compression observed under the Gompertz mortality change model,  $m_x = e^{\alpha+\beta x-\rho t}$ , with  $\alpha = -10.5$ ,  $\beta = 0.11$ , and  $\rho = 0.01$ . These initial conditions correspond to those found in (Canudas-Romo, 2008).

Bongaarts proposal of the shifting logistic model provided further foundation for the concept of shifting mortality (Bongaarts, 2005). In fitting the following logistic model to annual mortality rates for ages 25+ from a variety of countries included in the Human Mortality Database, Bongaarts notes that the slope parameter,  $\beta$ , has been relatively stable in the period 1950-2000:

$$\mu(x, t) = \frac{\alpha(t)e^{\beta(t)x}}{1 + \alpha(t)e^{\beta(t)x}} + \gamma(t) \quad (2.1)$$

The relative stability in the  $\beta$  parameter allows a new interpretation of recent changes in

senescent mortality rates. As Bongaarts states, “instead of interpreting mortality as rising or falling, the schedule of the force of senescent mortality can be viewed as shifting to higher or lower ages over time” (Bongaarts, 2005, p. 29). In adapting the model for forecasting, Bongaarts allows the  $\alpha$  and  $\gamma$  parameters to vary with time while the  $\beta$  parameter is fixed (Bongaarts, 2005).

Bongaarts bases his model of shifting mortality on data from the second half of the twentieth century. Therefore, one might ask whether the period of observation covered by Bongaarts data should be considered the shifting mortality era. As can be seen in Figure 2.2(b), only trends in  $S_{10}$  indicate a transition to shifting mortality as early as 1950 (i.e. all other measures exhibit decline indicating continued compression while trends in  $S_{10}$  are flat). Of course, Bongaarts only describes senescent mortality as shifting, not all-cause mortality. A continued decline in the background mortality, as described by the background parameter,  $\gamma(t)$ , might explain why measures of mortality compression that cover most of the adult age range such as  $C50$ ,  $e^\dagger$ ,  $IQR$ , and  $SDM$  still exhibit compression during the 1950s. What appears contradictory, however, are the continued declines in  $SD(M+)$  observed since the 1950s in France, as shown in Figure 2.2(b), as well as many of other countries included in Bongaarts’ analysis (Kannisto, 2000; Kannisto, 2001; Cheung and Robine, 2007; Robine, 2008; Thatcher et al., 2008) .

$SD(M+)$  should largely reflect trends in senescent mortality, which overwhelms background mortality above the modal age. Consider the senescent component of the Bongaarts shifting logistic model, which is the first term of equation 2.1. As Thatcher et al. have demonstrated, the death rates and modal age at death implied by this model depend on both the  $\alpha$  and  $\beta$  parameter; however, compression around the modal age is determined solely by the  $\beta$  parameter. Therefore, trends in the measure  $SD(M+)$ , can be determined just by looking at trends in the  $\beta$  parameter (Thatcher et al., 2008). As noted above, Bongaarts finds trends in the  $\beta$  parameter to be remarkably consistent across his period of observation. This discrepancy between consistency in the  $\beta$  parameter, which implies shifting mortality, and observed declines in  $SD(M+)$ , which likely reflects compression in senescent mortality at older ages is troubling.

While Kannisto and Bongaarts described shifting mortality as a shift in age-specific mortality rates across age, those documenting the transition to the shifting mortality era describe shifting mortality as occurring when the death distribution shifts to higher ages while retaining the same shape (i.e. in the case of  $SD(M+)$ , the modal age at death rises while retaining the same variability of age at death above the mode as measured by  $SD(M+)$  (Cheung and Robine, 2007)). For the shifting logistic model proposed by Bongaarts, the shift in senescent mortality across age should result in a shift in the senescent death distribution. It is not true generally, though, that a shift in mortality across age leads to a shift in the death distribution.

Researchers have made efforts to understand exactly how underlying changes in age-specific mortality produce shifting mortality. In an attempt to understand whether an increase in the modal age at death always implies mortality compression, Thatcher et al. fit a

logistic model to death rates at age 70 and 90 (Thatcher et al., 2008). This model is similar to Bongaarts' shifting logistic model, but it does not include the parameter for background mortality,  $\gamma$ :

$$\mu(x) = \frac{\alpha e^{\beta x}}{1 + \alpha e^{\beta x}}$$

The authors find that the modal age at death depends on both the  $\alpha$  and  $\beta$  parameters while the measure of mortality compression around the mode depends solely on the  $\beta$  parameter (as mentioned above). Mortality compression occurs if the logit of the death rate at age 70 falls faster than the logit of the death rate at age 90. To achieve shifting mortality, where the modal age increases while variability of age at death above the mode remains constant, the logits of the death rates at age 70 and 90 must fall at the same rate so that the  $\beta$  parameter retains the same value over time. Fitting their historical model for six countries included in the Human Mortality Database, Thatcher et al. observe a recent trend towards a stable  $\beta$ , and thus shifting mortality, in French females, Italian males, and both males and females in Japan. An important lesson to take from this analysis is that if old-age mortality can be described accurately by a logistic model fit to mortality rates at 70 and 90, similar rates of decline in mortality at these two ages is a necessary condition for observing shifting mortality under this model. This suggests that the eventual transition to a shifting mortality era is dependent on similarity or dissimilarity in the age pattern of mortality decline.

Canudas-Romo explores the emergence of the shifting mortality era by simulating mortality change over time using a variety of mortality models and comparing the results to actual trends in the modal age at death, number of deaths at the modal age, and *SDM* for a number of countries included in the Human Mortality Database (Canudas-Romo, 2008). By extrapolating age-specific mortality rates using both the Gompertz and logistic models of mortality change as well as the more complex Siler model, Canudas-Romo shows that the number of deaths at the modal age and the standard deviation of the distribution of deaths around the modal age eventually converge toward an asymptote while the modal age continues to rise. These results are based on assuming a fixed relationship of mortality over adult ages as implied by the fixed  $\beta$  parameters in the Gompertz mortality, logistic model, and Siler mortality change models. The results of Canudas-Romo's simulation using the Siler mortality change model indicate that the pace of mortality compression slows after infant mortality has reached a relatively low level. This suggests that the potential for compression depends on current mortality conditions.

An explanation for shifting mortality related to divergence in the age pattern of mortality change appears in Wilmoth and Horiuchi's 1999 piece on trends in variability of age at death (Wilmoth and Horiuchi, 1999). Using historical mortality evidence from Sweden, the authors document that life expectancy rose steadily throughout the period of observation (1751-1995). In contrast, the *IQR* remained relatively stable from 1751-1875, declined rapidly during the period 1876-1955, and then remained relatively stable at its new lower level from 1955 onwards. The authors observe that during the period of rapid decline in *IQR*, the

average annual rate of proportional mortality decline varied greatly across age with much more rapid progress at younger ages. In contrast, during periods in which the *IQR* remained relatively stable, the average annual rate of mortality decline was similar across ages<sup>1</sup>. This observation along with some intuitive reasoning led the authors to propose that dissimilarity across the age pattern of mortality change was the main cause of the decrease in *IQR*.

The notion that divergent age patterns of mortality change (with younger age groups experiencing faster progress) lead to a decline in the variability of age at death is especially appealing if it also true that if the age pattern of change in mortality is similar across ages (especially the ages where deaths are concentrated), then variability of age at death will remain constant. The results of Thatcher et al. seem to suggest that similarity in the age-pattern of mortality change is a necessary condition for observing shifting mortality. Is it a sufficient condition? This line of thought leads to a simple and easily tested hypothesis: if mortality change is proportional across age (the pattern of mortality change is similar across age on a relative scale), then there will be no change in the variability of age at death. Instead of becoming more or less variable, the death distribution will just parallel shift in either direction. If this relationship holds true, similarities in the relative pace of change in mortality across age might be the underlying cause of the current shifting mortality scenario. In Chapter 5, I explore whether proportional change in mortality that is fixed across age always lead to a shift in the death distribution and find that this is not always the case. In the next section, I turn back to the topic of mortality inequality.

## 2.4 Mortality inequality among groups

In this section, I offer a review of current literature on mortality disparities among groups. These disparities exist along a number of dimensions. Here I offer a review of disparities by gender, socioeconomic status, race/ethnicity, nativity, geography, marital status, and religious participation. One of the major aims of mortality disparities research is to understand the underlying reasons for the observed differentials. In general, there are three types of explanations offered to understand health disparities across social groups. (1) It may be that the person's social status either directly or indirectly affects their risk of ill-health through some causal mechanism. This is generally thought to be the major explanatory factor; however, (2) it is possible to observe the same type of association between social grouping and health even if the direction of causality were reversed and some type of selection was involved (Goldman, 2001). For example, the relationship between SES and health has been shown to be bi-directional (Smith, 2004). Also, the selection pressures of migration might help to explain the "Hispanic paradox" wherein Hispanics and non-Hispanic whites have similar levels of infant mortality, low birth weight, and adult health and mortality (Palloni

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<sup>1</sup>In the most recent period, the pattern is not similar across all ages but it is in the ages where the majority of deaths are concentrated. The average annual rate of mortality decline is calculated using the formula  $-\log\left(\frac{m_x(t+n)}{m_x(t)}\right)/n$ .

and Morenoff, 2001). (3) Finally, observed disparities might be a statistical artifact if, for instance, measurement error occurs (Goldman, 2001). At one point, the mortality crossover in old-age for blacks and whites was thought to be the result of blacks being more likely to over report their age; however, recent studies using more reliable data have shown that this crossover may not be a statistical artifact but rather a real phenomenon (Hill et al., 2000; Rogers et al., 2000).

I focus mostly on mortality disparities in the context of the United States; however, for gender disparities, I focus on evidence collected cross-nationally in order to give proper context for Chapter 7 where I examine gender gaps in variability of age at death using data representative of a variety of countries.

### **Gender inequalities in mortality**

Currently, females enjoy higher life expectancy in comparison to males in most countries of the world. In more developed and less developed countries alike, males generally experience higher mortality rates than females at all ages; however, this has not always been the case as females suffered higher mortality rates in childhood and early adulthood in the late 19th and early 20th century according to historical mortality records (Waldron, 2003). Of particular interest in the 20th century was the widening and subsequent narrowing of the gap in life expectancy between males and females in developed countries (Glei and Horiuchi, 2007). Initially, the gap in life expectancy widened due to male disadvantage in cardiovascular and neoplasm related mortality (Vallin, 1993).

During the earlier part of the 20th century, males suffered disproportionately from mortality due to lung cancer in comparison to females as a result of their earlier adoption of smoking (Waldron, 2003). By examining cohort smoking histories and subsequent mortality, Preston and Wang have shown that the widening and narrowing of the gender gap in life expectancy in the United States can largely be explained by the lag in females adopting smoking (Preston and Wang, 2006). Using data representative of 21 countries, Pampel arrives at a similar conclusion: the gender gap in life expectancy can be explained by the diffusion of cigarette use among each gender (Pampel, 2003). Looking beyond differences in smoking patterns, Glei and Horiuchi show that the narrowing of the gender gap in life expectancy is partly due to gender differences in variability of age at death. In particular, the smaller variance in the female death distribution results in smaller gains in life expectancy in comparison to males even with similar improvements in mortality rates (Glei and Horiuchi, 2007).

Biological differences are also thought to play a role in female mortality advantage. For example, female sex hormones are protective against cardiovascular disease. In addition, males experience a greater likelihood of accumulating abdominal fat, which puts them at higher risk for cardiovascular disease (Waldron, 2003). Very basic genetic differences even play a role in explaining female advantage. Christensen et al. have hypothesized that females having two X chromosomes in comparison to male's X and Y contributes to sex differences

in survival. The core of their reasoning is that both females and males have only one X chromosome active in each of their cells. Females can have either the X chromosome from their mother or their father active in each cell while males only have the X chromosome from their mother. This is why males are at greater risk of X linked diseases like color blindness and Duchenne's muscular dystrophy. Young women tend to have a 50-50 distribution of cell lines from their mother and their father while older women are more likely to have a skewed distribution. They hypothesize that the skewed distribution observed among older women is either due to random chance (since inactivation of one X occurs early in utero) or because there is some selection force operating. In order to test their hypotheses, the researchers utilize data on elderly monozygotic twins in the Danish twin study. The researchers find that the elderly twins have a similar distributions of X inactivation in comparison to their elderly female singleton counterparts, which suggests that selection is at work (Christensen et al., 2001).

In addition to differences in smoking and biological factors, other factors have been identified as contributing to gender disparities in mortality. With regards to females' mortality disadvantage in childhood and young adulthood in the earlier part of the 20th century, Vallin cites females' lower social status as a potential contributing factor as it made females more vulnerable to death due to infectious disease (Vallin, 1993). Males tend to engage in more risk taking and dangerous behaviors, which increases their risk of deaths due to external causes (e.g. accidents and homicide) relative to females (Waldron, 2003). As will be shown in Chapter 8, the elimination of infectious disease and male disadvantage in external cause mortality are a major explanatory factors of the emergence in the gender gap in variability of age at death.

#### **2.4.1 Socioeconomic disparities in mortality**

Socioeconomic disparities are of much interest in the United States because these disparities have persisted over time and even widened in recent years (Rogers et al., 2000). Also, interpretations of these disparities can vary because the notion of socioeconomic status is complex. Galobardes et al. offer this definition of socioeconomic position: "the socially derived economic factors that influence what positions individuals or groups hold within the multiple-stratified structure of a society" (p. 3) (Galobardes et al., 2007). Three key aspects of socioeconomic position have been identified in the health literature: material goods, social status, and work conditions and relations (Lawlor et al., 2005). Since socioeconomic position is defined on multiple dimensions and socioeconomic position can change throughout the life course, it is not possible to describe fully an individual's socioeconomic position with a single measure. Studies seeking to examine the influence of socioeconomic status on health and mortality often rely on many different measures of socioeconomic status such as income, wealth, education, and occupation. In addition, certain studies will incorporate multiple measures of socioeconomic status collected over the life course. In this section, I first review the literature on the relationship between adult socioeconomic status and mortality risk.

Realizing that socioeconomic status is a dynamic variable whose effects can accumulate over the life course, I offer a brief review of life course theory and studies which examine the influence of childhood socioeconomic status on adult mortality risk. Although of critical importance, I do not detail the many issues involved with measuring socioeconomic status. For a thorough treatment of these issues, see (Galobardes et al., 2007).

### **Adult socioeconomic status and mortality disparities**

In 1973, Kitagawa and Hauser published a landmark study concerning socioeconomic differentials in mortality in the United States (Kitagawa and Hauser, 1973). At that time and for many decades afterwards, this type of analysis was difficult to undertake because data on socioeconomic status was not collected in death records in the U.S. (Rogers et al., 2000). In order to compare mortality risk across socioeconomic classes, Kitagawa and Hauser matched death certificates from 1960 to census records collected in the same year. Using data on both income and education, the authors showed that those with less income and/or lower educational attainment suffered higher mortality risk. The relationship between socioeconomic status and mortality was stronger for adults (ages 25-64) in comparison to the elderly (ages 65+) (Kitagawa and Hauser, 1973; Rogers et al., 2000).

In a follow-up analysis using similar methods and data from 1986, Pappas et al. find that among whites all educational groups had experienced mortality declines between 1960 and 1986 yet educational and income inequalities in mortality had increased for both males and females during this period (Pappas et al., 1993). Preston and Elo find that the education gradient in mortality is stronger for men than for women and that it is observable even when controls are added for income and current residence as well as other household/demographic factors (e.g. household size, age, race) (Preston and Elo, 1995). In a more recent study using data representing the years 1991-1995, Rogers et al. continue to find socioeconomic gradients in mortality when socioeconomic status is measured by education, income, or occupational status. The effect of income on mortality risk can be explained by controlling for education, employment status and health status; however, educational attainment and employment status both have independent effects that are observable even when controlling for the other factor, income, health status, and other control variables (Rogers et al., 2000). Another recent study, which will be discussed more in the section on geographic inequalities in mortality, demonstrates that socioeconomic differentials in mortality have increased between 1980 and 2000 by showing that life expectancies have diverged in the less-deprived counties in the U.S. compared to the most-deprived (as indicated by education, wealth, occupation, income, employment status, poverty status, and housing quality of county residents) (Singh and Siahpush, 2006). The results outlined here suggest that socioeconomic disparities in adult mortality in the United States are noteworthy and have been increasing in recent decades.

## Life course models

Researchers who adopt a life course approach when studying chronic diseases investigate the effects of social and physical exposures that occur in different periods of the life course on risk for disease. Specifically, researchers focus on the behavioral, biological, and psychosocial pathways that lead to the onset of the disease (Ben-Shlomo and Kuh, 2002). In their 2002 article “A life course approach to chronic disease epidemiology: conceptual models, empirical challenges and interdisciplinary perspectives,” Ben-Schlomo and Kuh outline two life course models. The first model, the *critical period or latency model*, postulates that disease risk might be influenced by events that occur during particular periods in the life course and whose effects are not mediated by later life events (Ben-Shlomo and Kuh, 2002). Barker’s fetal origins hypothesis is an example of the critical period model (Barker, 1995). The other model that Ben-Schlomo and Kuh review is the *accumulation of risk model*. This model proposes that risk for chronic disease is affected by a number of factors that accrue over the life course (Ben-Shlomo and Kuh, 2002). The weathering hypothesis proposed by Geronimus, which will be discussed in the section on race/ethnic inequalities in mortality, is a particularly relevant example of the accumulation model (Geronimus et al., 2006).

In their systematic review of the relationship between life course socioeconomic factors and cardiovascular disease, Pollitt et al. outline two more life course models: the pathway model and the social mobility model. In *the pathway model*, early life events are pivotal in determining behaviors and opportunities later in life, which are associated with health risks (Pollitt et al., 2005). An important distinction between the pathway model and the accumulation of risk model is that in the pathway model events during childhood do not have an effect on later disease risk independent of their influence on later life behaviors and opportunities. Transition between socioeconomic positions over the life course is the focus of the *social mobility model*. In this model, changes in social class across the life course result in differential health risks (Pollitt et al., 2005). For instance, Forsdahl hypothesized in the late 1970s that upward social mobility across the life course results in higher risk of cardiovascular mortality. Forsdahl attributes this increase in risk to higher cholesterol levels in persons who move from lower to higher social classes during their life course (Forsdahl, 1978).

## Childhood socioeconomic status and mortality disparities

As suggested in the discussion of life course theory above, conditions in childhood can potentially impact later life mortality risk through a variety of channels. For instance, many studies have shown that childhood health has an effect on mortality risk in adulthood (Elo and Preston, 1992). Socioeconomic circumstances in childhood influence health in childhood, and the intergenerational transmission of poverty is thought to operate through this channel because poor health in childhood limits the potential for socioeconomic achievement in adulthood (Case et al., 2002; Case et al., 2005). Indeed, studies of the influence of child-

hood socioeconomic status on risk of mortality in adulthood based on US data sets suggest that the relationship between these two variables can largely be explained by the effect of childhood socioeconomic status on adult socioeconomic status and health behaviors lending support to the pathway model outlined in the previous section (Mare, 1990; Elo and Preston, 1992; Hayward and Gorman, 2004). A study of the mortality of Scottish men shows that the relationship between childhood socioeconomic status and later mortality risk varies by cause-of-death. Davey Smith et al. find that childhood socioeconomic status directly effects risk of mortality due to stroke and stomach cancer in adulthood. Childhood socioeconomic status also directly influences risk of death due to respiratory disease and coronary heart disease but adult socioeconomic status also contributes to risk for these diseases lending support to the accumulation hypothesis. Finally, childhood socioeconomic status does not seem to directly impact risk of death due to cancer and accidents and violence but adult socioeconomic status is an important risk factor for these causes, which fits the pathway model of life course effects (Smith et al., 1998).

#### **2.4.2 Race/ethnic inequalities in mortality**

Much attention has focused on differences in mortality across race/ethnic groups in the United States because of the diversity of the population and the persistent disparities observed across groups (especially with regard to black-white differences). In general, Asian Americans exhibit the lowest mortality and black Americans exhibit the highest mortality with white Americans and Americans of Hispanic origin falling somewhere in between. Using national health survey data collected between 1989 to 1994 and linked mortality files, controlling for age and sex, Rogers et al. find that African Americans have 41% higher odds of mortality and Asian Americans have 31% lower odds of mortality in comparison to Caucasian Americans. Breaking down the Hispanic category into more refined groupings, Rogers et al find that Puerto Ricans have 33% higher odds on mortality in comparison to Caucasian Americans while Mexican Americans and Cuban Americans have similar levels of mortality and other Hispanics have 17% lower odds (Rogers et al., 2000). The differences between African Americans and Caucasians as well as Puerto Ricans and Caucasians are attenuated somewhat when socioeconomic variables and marital status are included in the model (17% higher odds for African Americans and 23% higher odds for Puerto Ricans in comparison to Caucasian Americans), but disparities persisted consistent with evidence from other studies of the black-white mortality gap (Rogers et al., 2000; Preston and Elo, 1995),

While black Americans have higher mortality risks throughout most of the age range and disparities are especially marked in infancy, childhood, and young adulthood, there is evidence that black and white mortality risks converge at older ages and that the relationship possibly reverses at the oldest ages. In this literature, this is referred to as the “black-white mortality crossover,” and it is thought to be either the result of age misreporting or the result of differential selection (Nam, 1995). As data quality and methods have improved, the black-white crossover continues to be observed although at a later age than earlier studies

(around age 90-95) suggesting that the crossover may not be a statistical artifact but rather a real phenomenon (Hill et al., 2000; Rogers et al., 2000). Another subject of debate regarding race/ethnic mortality differentials, the “Hispanic paradox” will be discussed in the next section on mortality differentials by nativity.

Black-white differences in mortality are thought to be the result of a number of factors beyond socioeconomic differences including racism and discrimination (Williams, 1999). The weathering hypothesis proposed by Arline Geronimus is a particularly relevant example of the accumulation life course model. This hypothesis maintains that blacks on average suffer from health deterioration earlier than whites because blacks are more likely to be repeatedly exposed to social and economic difficulty as well as political marginalization. In their 2006 article, Geronimus et al. find that blacks have higher allostatic load scores compared to whites at all ages. These higher allostatic load scores reflect the cumulative effect of repeated stressors (Geronimus et al., 2006).

The black-white gap in life expectancy has been narrowing over the past century in the United States; however, as will be discussed in a later section, inter-individual measures of mortality inequality lend new insights into the changes in racial disparities in mortality during the 20th century that are masked when just considering changes in life expectancy (Weden, 2007).

### **2.4.3 Native versus non-native inequalities in mortality**

Race/ethnic disparities within the United States become even more complicated when one takes nativity into account. Evidence based on data from the US Census, the Current Population Survey, and the National Mortality Database indicates that overall natives have experienced higher mortality in comparison to non-natives since the 1980's and that these disparities have widened over time. In 1999-2001, the overall gap between non-native and native life expectancy was 3.4 years (80.0 vs 76.6 years respectively) in comparison to 2.3 years (76.2 vs 73.9 years) in 1979-1981. The differences between natives and non-natives tend to be greater for males in comparison to females. The difference in life expectancy between black male natives and black male non-natives is especially striking (67.5 years versus 75.6 years in 1999-2001) (Singh and Hiatt, 2006). Controlling for socioeconomic status and demographic background, non-native blacks experience 48% lower mortality in comparison to native born whites (Singh and Siahpush, 2002). Differences in health behaviors are thought to play a role in creating these mortality differentials. Non-natives are much less likely to smoke and to be overweight or obese in comparison to natives (Singh and Hiatt, 2006). In addition to differences in behavior, it is possible that selection plays a role as well in explaining differentials as the process of migration favors healthy individuals (Palloni and Morenoff, 2001). The non-native mortality advantage plays a key role in explaining the similarity in mortality levels observed between whites and Mexican Americans (often referred to as the “Hispanic paradox”) despite the latter group being of lower socioeconomic status on average (Hummer et al., 1999).

#### 2.4.4 Geographic inequalities in mortality

For the types of inequality that I review in this section, most groupings are based on individual-level characteristics; however, macro-level factors can also produce inequality in mortality outcomes (Hummer et al., 1998). Specifically, geographic context can have an important influence on an individual's life chances. One type of geographic disparity that has long been studied is urban-rural differences in mortality. While persons living in urban areas tend to be more highly educated and enjoy higher incomes, urban life also includes more hazardous elements such as environmental pollution and crime (Valkonen, 2003). In a recent study of US urban-rural mortality differentials, House et al. found that men who lived in urban areas exhibited much higher mortality risk in comparison to men living in rural areas (hazard ratio 2.25), but they did not find a significant difference for women. In terms of causes-of-death, men in urban areas were more likely to die due to tumors or infections in comparison to men in rural areas. The authors find that both increased smoking and drinking contribute to the urbanites mortality disadvantage (House et al., 2000).

Several studies recently have focused on trends in mortality inequality in the United States as a whole by examining differences in mortality across regions defined according to some criteria (Ezzati et al., 2008; Murray et al., 2006; Singh and Siahpush, 2006). The results of at least two of these studies suggest that there has been an increase in mortality disparity since the 1980s within the US. Singh and Siahpush classified counties into deciles based on socioeconomic indicators from Census data, and they find that the life expectancy gap between the best off counties and the worst off counties has increased from the 1980s to the present (Singh and Siahpush, 2006). In contrast, in a study by Murray et al. in which they divided the United States into "eight Americas" based on race, location, population density, income, and murder rates, the authors find no change in the absolute difference between advantaged and disadvantaged regions over the period 1982-2002. The widest gap in life expectancy of 15.4 years is observed between Asian males and high risk urban black males (Murray et al., 2006).

In a study examining the trend in the standard deviation of life expectancies across all counties in the United States, Ezzati et al. find a decreasing trend in the standard deviation from roughly 1961-1983 and an increasing trend subsequently. Comparing the counties within the top 2.5% of life expectancy to those in the bottom 2.5%, the gap in life expectancy between these extremes widened during the 1980s (for men it appears to be coming down recently) due to stagnation in improvements in mortality among those with the lowest life expectancy. Causes of death responsible for this stagnation include cardiovascular mortality, other chronic diseases, HIV and homicide (for men) (Ezzati et al., 2008).

Performing an analysis of trends in mortality inequality at the county level raises quite a few questions with the key among them being problems with selection. It is possible that healthy citizens in poorly performing counties are selected out of the population by way of education and outside job opportunities. Also, in the best performing counties, healthier citizens might be selected into the population. Ezzati et al. attempt to control for migration

in their analysis using simulation models and claim that migration can not explain the widening of the gap in life expectancy observed since 1983 (Ezzati et al., 2008). In addition to problems with selection, in their analysis, the authors fail to calibrate for the intrinsic spread extreme values. Therefore, it is unclear whether the widening of the gap is indicative of increasing social inequalities in health or random variation.

#### **2.4.5 Disparities in mortality by marital status**

Numerous studies have shown the beneficial effects of social ties in terms of lowering risk of mortality (Berkman and Syme, 1979; House et al., 1982; Seeman et al., 1987). In particular, many studies dating as far back as the 19th century have focused on the health benefit that marriage provides (Valkonen, 2003). Cross-national evidence indicates that married persons enjoy lower mortality risks in comparison to those who are single, widowed, or divorced (with divorced men displaying the highest mortality risks), and that the protective effect of marriage has been increasing over time (Hu and Goldman, 1990). Whether the marriage advantage is due to protective effects of marriage itself or due to selection effects (with healthy persons more likely to marry) has been a subject of much debate in the literature (Goldman, 1993). Research utilizing longitudinal data indicates both processes might be at work (Murray, 2000). For women, the protective effect of marriage has been shown to be associated with the greater financial security marriage provides (Lillard and Waite, 1995).

#### **2.4.6 Mortality disparities and religious participation**

Consistent with the literature on the benefit of social ties, affiliation with a religious organization has been found to be protective against mortality. These benefits are thought to be derived from both social contact as well as improved health behaviors associated with religious involvement (e.g. less drinking and smoking (Gottlieb and Green, 1984)). Rogers et al. find that, in comparison to those who attend religious services weekly, those who never attend services have 80% greater odds of death while those who attend somewhat regularly have 28% higher odds. These relationships are attenuated when controls are added for social participation, marital status, socioeconomic status, and smoking, but the relationship remains significant. They find that the relationship is stronger for younger persons (18-64) in comparison to the elderly, and that, in terms of cause-of-death, those who never attend church are at higher risk for death due to respiratory diseases and social pathologies (including accidents, cirrhosis of the liver, homicide, and suicide) (Rogers et al., 2000).

### **2.5 Mortality inequality among individuals**

In this section, I focus on studies that quantify mortality inequality by measuring differences in mortality among individuals (inter-individual mortality inequality). What distin-

guishes these studies from the studies reviewed in the last section is that in these studies inequality is measured across individuals at either the population or subpopulation level rather than across groups; however, what ultimately comes out of approaching the study of mortality inequality from this angle is that these measures provide a new way to understand mortality differentials across groups. Not only are these measures an indicator of disparity themselves, but the differences in these measures across groups (the disparity in disparities) are meaningful. I begin this review by examining studies which compare mortality inequality across countries. Then, I consider a few studies which look at differences in mortality inequality across population subgroups and how differences in mortality across subgroups contributes to total inequality in mortality.

Edwards and Tuljapurkar examine the convergence of mortality across developed countries including Canada, Denmark, France, Britain, Japan, Sweden, and find that while mortality patterns have largely converged, differences remain, and differences in variability in ages at death across countries explain much of the “lingering divergence” (Edwards and Tuljapurkar, 2005, p. 649). These authors use the measure  $S_{10}$  to quantify mortality inequality and find that the United States exhibits the highest inequality in mortality around the turn of the 21st century among the countries included in their study while Sweden experiences the lowest. Edwards and Tuljapurkar go through a series of possible explanations of the high level of mortality inequality observed in the United States, which will be discussed in greater detail below when I discuss the contribution of social inequalities to total life span disparity.

In a follow up study, Edwards examines variance in world life span. Using data from 180 countries in 1970 and 2000, Edwards examines differences in average world life span and the variance of world life span across these two time points. While unconditional variance in world life span has decreased during this period as a result of continued improvements in infant mortality, variance in world life span at adult ages (above age 10) has stagnated. Importantly, between these two time points, the contribution of differences in between-country variance in adult life span to total variance in adult life span has increased suggesting greater divergence in inequality measures across countries (Edwards, 2009b).

Two recent studies have utilized the Gini index to examine trends in inter-individual mortality inequality across countries (Shkolnikov et al., 2003; Peltzman, 2009). Examining trends over the second half of the twentieth century in the relationship between life expectancy and the Gini coefficient in Japan, Russia, Spain, the United Kingdom, and the United States, Shkolnikov et al. observe a great deal of convergence in life expectancy and Gini coefficients over the period of observation with all countries experiencing improvements in life expectancy and reductions in inequality except in Russia where mortality conditions deteriorated during the 1990s. For males in the US, the authors observe stagnation in the Gini coefficient in the 1980s and early 1990s. When the data is restricted to deaths above age 15, the Gini coefficient rises over this period. Using decomposition analysis, the authors show that this increase was due to increases in mortality at younger ages due to HIV/AIDS and violent deaths as well as continued improvements in mortality at older ages (Shkolnikov et al., 2003).

Peltzman's article on mortality inequality focuses on the dramatic improvement in mortality inequality observed over the past century (Peltzman, 2009). Improvements in mortality inequality outpace improvements in income inequality using the same measure, the Gini index. During the course of economic development, when a country transitions from low to high income, income inequality initially increases before decreasing. The relationship between income per capita and income inequality over the course of economic development is described by Kuznet's curve. In contrast to the relationship implied by Kuznet's curve, Peltzman notes that shifts from a high mortality regime to a low mortality regime are not accompanied by initial increases in mortality inequality (the Gini index decreases steadily as life expectancy increases). In the case of life span inequality, improvements in mortality inequality are largely the result of improvements in mortality at the youngest ages, which lead to both an increase in average longevity and a decrease in life span disparity. Peltzman also demonstrates that improvements in mortality inequality can not be accounted for entirely by improvements in income inequality, and that over the past twenty years in the US, income disparities have widened further than mortality disparities (Peltzman, 2009).

Smits and Monden calculate length of life inequality measures for the whole world using a data base that includes over 2000 life tables (Smits and Monden, 2009). Smits and Monden's analysis differs from others outlined here because, rather than using absolute measures of mortality inequality, the authors claim that these measures should be evaluated relative to life expectancy. They base this claim on the fact that what might be a relatively low level of inequality for a countries with life expectancy of 60 would be a relatively high level of inequality among countries with life expectancy of 75. They use the Gini coefficient and the Theil index to measure inequality using the death distribution over age 15, and then develop a measure of relative mortality inequality by standardizing the measure of inequality at each level of life expectancy. In their analysis, the authors attempt to test the "diffusion" and "forerunner" hypotheses. The forerunner hypothesis suggests that countries, which are leaders life expectancy, will experience lower levels of mortality inequality because they are successful in reducing premature mortality, which both lowers mortality inequality and increases life expectancy. In contrast, the diffusion hypothesis suggests that countries, which are not leaders in life expectancy, experience lower mortality inequality at a given level of life expectancy because they benefit from technological and medical innovations developed by the leading countries. Through statistical analysis of the effect of time on measures of relative mortality inequality, the authors find support for the diffusion hypothesis (i.e. the leading countries experienced higher levels of relative mortality inequality); however, it is not clear how the authors are accounting for the differential time periods covered in the data for each country.

Now, I turn my attention to studies comparing mortality inequality measures across population subgroups. In trying to explain the relatively high values of  $S_{10}$  observed in the United States, Edwards and Tuljapurkar examined differences in  $S_{10}$  across socioeconomic subgroups. Comparing the value  $S_{10}$  for those in upper 80th percentile of the income distribution in the US in 1981 to the bottom 20th percentile, they find that higher income is

associated with lower mortality inequality ( $S_{10}$  of 14.4 years versus 16.8 years). Comparing high school graduates to those with less than high school education in the same period, they observe a similar difference in  $S_{10}$ , 14.6 years for the more highly educated versus 16.7 years for those with less than a high school education. While these differences in  $S_{10}$  indicate greater uncertainty for those at the lower end of the social hierarchy in addition to shorter average life times, they do not explain why the US displays such high mortality inequality as indicated by  $S_{10}$  because even if the sample is restricted to high school graduates an  $S_{10}$  of 14.6 is still quite high in comparison to other more developed countries (Edwards and Tuljapurkar, 2005).

In a cross-national study of the contribution of educational differentials in mortality to total inter-individual mortality inequality, van Raalte et al. find that in Western European countries, differences in mortality among educational group only account for about 1% of the total inequality in mortality as measured by the Theil index, while in Eastern European countries it accounted for 2-8% of the differentials (van Raalte et al., 2009). This analysis, consistent with Edwards and Tuljapurkar's assessment of  $S_{10}$ , demonstrates that studies of inter-individual inequality can be interpreted as being distinct from studies of socioeconomic differentials in mortality (Edwards and Tuljapurkar, 2005; van Raalte et al., 2009). That is, results from these studies do not mirror results from studies of socioeconomic differentials because these measures are capturing mortality differentials that can not be explained by socioeconomic inequality alone. In fact, studies of inter-individual inequality provide new insights into socioeconomic differentials in mortality.

In a study of educational differentials in Gini coefficients among Russians in 1979 and 1989, Shkolnikov et al. demonstrate an important distinction between inter-individual inequality in mortality and mortality differentials across groups. Even though the gap in life expectancy between the best and least educated widened between 1979 and 1989, the Gini coefficient for the total population decreased over the period. Their analysis also demonstrates the value of using measures of inter-individual inequality to compare mortality differentials across groups. They find that differences in Gini coefficients across educational groups are more dramatic than differences in life expectancy (i.e. in 1989, Russians with university education had a life expectancy between ages 20 and 65 of 42.09 years and a Gini coefficient of 5.99 while those with less than a secondary education had a life expectancy of 37.04 and a Gini coefficient of 15.57) (Shkolnikov et al., 2003).

Measures of inter-individual inequality can also be used to gain new insight into mortality differentials by race. Edwards and Tuljapurkar show, similar to their demonstration for educational differentials, that even though African Americans experience higher levels of  $S_{10}$  relative to whites in the U.S. (2-3 years higher) these disparities do not explain the high levels of  $S_{10}$  observed in the US in comparison to other countries (Edwards and Tuljapurkar, 2005). Weden examines trends in  $IQR$  for whites and non-whites in the U.S. over the period 1900-2002. While trends in white and non-white life expectancy convergence over this time period, the race-specific trends in  $IQR$  first diverge and then later converge. By decomposing the change in  $IQR$  over time into the contributions of changes in age-specific

mortality rates, Weden shows that the lag in decline in *IQR* for non-whites in the first fifteen years in the century is due to lack of improvement in mortality in the 0-4 age group for non-whites. Weden also notes that in the period 1930-1945 differential changes in age-specific mortality in the 45-64 year old age group contribute to a widening of the racial gap in *IQR* over this period. Changes in mortality in this age group produce mortality expansion (contribute positively to changes in *IQR*) over this time period for non-whites, while for whites changes in this age group produce mortality compression. Observing differential patterns of age-specific compression between whites and non-whites, Weden suggests that mortality declines for non-whites "have been later, slower, and have involved more irregular age-composition" in comparison to whites (Weden, 2007). While these differentials in mortality change are obscured when one just examines trends in life expectancy, examining trends in inter-individual inequality (using *IQR*) for whites and non-whites reveals these differentials. This study along with the other studies of racial and socioeconomic disparities in life span disparity documented here offer a new perspective on mortality disparities.

## 2.6 Summary

What have we learned in this review of the literature? With regards to matters of mortality compression and shifting mortality, it is clear that dramatic reductions in variability of age at death are a key feature of the early stages of the mortality transition. What is open for debate, however, is whether mortality compression continues in the more recent stages of the mortality transition or whether we have entered into an era of shifting mortality. As was evident in the review of the literature on this topic, the answer to this question varies depending on the measure of mortality compression used. My work on this topic, which is featured in this dissertation, focuses on trying to understand how measures of mortality compression respond to changes in age-specific mortality to produce mortality compression or a lack thereof.

We have also learned through this review that traditional measures of mortality compression have recently been recast as measures of mortality inequality providing new insight into mortality disparities. The traditional mortality disparities literature reviewed here suggests that disparities exist along a number of dimensions and that they have persisted across time. Importantly, the limited number of studies that examine the contribution of social disparities to total mortality inequality observed at the population level find that social disparities can not explain much of the total disparity. Indeed, the results of studies of inter-individual mortality inequality do not mirror more traditional studies of social disparities in mortality suggesting that measures of inter-individual inequality capture more than social injustices—perhaps also biological differences in mortality risk. Finally, this review has demonstrated that measures of inter-individual inequality can be used to compare mortality differentials across social groups in order to gain insights into mortality disparities that are obscured when only mean life span is taken into account. In my work on gender disparities in mortal-

ity inequality in Chapters 7 and 8, I find that the changes in age and cause-specific mortality that produce the female advantage in certainty in timing of death differ from the changes which produce female advantage in life expectancy.

One aspect of the study of mortality compression and mortality inequality that I have yet to address is the broader implications of changes in these measures. I take up this important matter in the next chapter.

Table 2.1: Measures used to assess rectangularity and variability of age at death. Adapted from (Wilmoth and Horiuchi, 1999; Cheung et al., 2005).

Measure	Classification
Life expectancy ( $e_0$ )	Central longevity indicator
Modal age at death ( $M$ )	.
Median age at death	.
C-family ( $C10$ , $C50$ , and $C90$ )	Concentration and/or verticalization indicator
${}_{10}C_{50}$	.
Degree of verticalization ( $\theta$ and $\theta^*$ )	.
$e^\dagger$	.
Entropy Keyfitz's $\bar{H}$	.
Fastest decline and/or highest proportion of deaths	.
Inter-quartile range ( $IQR$ )	.
Life expectancy at median life span and third quartile	.
Prolate index	.
Standard deviation of ages at death ( $S_x$ )	.
Standard deviation above the mode ( $SD(M+)$ )	.
Standard deviation above the third quartile	.
Degree of horizontalization ( $\beta$ )	Horizontalization indicator
Percentiles	Mapping indicator
Life endurancy	Maximum longevity indicators
Length of the outer tail of longevity	.
Maximum life span (MLS)	.
$M + 4SD(M+)$	.
Fastest decline	Rectangularization indicator
Fixed rectangle	.
Moving rectangle and/or index of rectangularity	.
Person-years differential (PD)	.
Person-years ratio (PR)	.
Sharpest corner	.
Quickest plateau	.
Coefficient of variation	Other indicator
Gini coefficient	.
Theil index	.

# Chapter 3

## Implications of changes in certainty of timing of death

“A population in which death is almost always deferred until the older ages is one that grows faster, is more willing to train individuals for the ‘long run’, can rely more heavily on the nuclear family for emotional gratification, and can dispense more readily with fatalistic or supernatural philosophies. Mortality declines in the last century have had a subtle but possibly decisive impact on a wide range of human activities.”

-Preston, Keyfitz, and Schoen (Preston et al., 1972)

Those who document the history of the human mortality experience, like Preston, Keyfitz, and Schoen in their book of historical life tables, often comment that the changes in mortality observed over the course of the demographic transition must have had an impact on other areas of human life besides death. In this chapter, I take up this question giving particular regard to the implications of changes in certainty of timing of death as indicated by trends in variability of age at death. I draw on literature from a variety of academic subjects and explore the implications of mortality compression over the course of the mortality transition with reference to a biological, economic, psychological, and demographic perspective, giving particular attention to how changes in certainty about timing of death might impact another key demographic process: fertility. Before exploring these implications, though, I investigate two key issues in assessing human response to changes in certainty of timing of death. First, to what degree are persons able to assess accurately their mortality risk and changes in mortality risk over time, and second, to what degree do people value certainty in timing of death.

### 3.1 Mortality risk perception

In arguing that human behavior is responsive to changes in certainty of timing of death, I assume that persons have some understanding of their own mortality risk. In this section, I examine evidence from a variety of studies on risk perception in order to answer two questions: (1) are individuals able to estimate accurately their own mortality risks and (2) are individuals able to perceive changes in mortality risk over time. Here I offer a mix of evidence gathered from both more developed and less developed countries. It is important to be cognizant not only of the vast differences in mortality conditions among these different geographical locations but also of the time scale over which mortality change takes place. While persons may not be able to assess accurately short term changes in mortality from one year to the next, a young person entering adulthood in the United States at the turn of the 21st century likely perceives their mortality prospects differently than a person of the same age at the beginning of the 20th century. The answers to these questions about whether humans can accurately assess mortality risk are especially relevant for a later discussion of the impact of changes in certainty of timing of death on fertility as many demographic theories of fertility assume parents are able to gauge the survival prospects of their children.

The evidence about whether individuals are able to estimate accurately their own mortality risk is mixed. Using data from the Health and Retirement Survey, Hurd and McGarry have shown that survey respondents' self-assessments of their own survival probabilities have predictive power. The self-assessed survival probabilities of those who survived between two waves this longitudinal survey were around 50% higher in comparison to those who died during the interval between the two surveys (Hurd and McGarry, 2002). Comparing individuals self-assessed survival expectations in the 1995 Aging, Status and the Sense of Control (ASOC) survey, a national telephone sample representative of households in the United States, to actuarial tables for the United States, Mirowsky finds that on average subjective and actual life expectancies agree; however, younger persons fail to account for expected improvements in survival over time so their subjective expectations agree better with current mortality conditions than projected mortality conditions for their cohort. Other inconsistencies that Mirowsky discovers include males tending to overestimate their life expectancy by about 3.5 years and blacks overestimating their life expectancy by 6 years based on comparison with current actuarial tables and controlling for socioeconomic and health status (Mirowsky, 1999).

Evidence from the literature indicates that there are a variety of factors which influence individuals' perceptions of their own survival chances. In the Hurd and McGarry study, individuals' self-assessed survival probabilities over time were influenced by death of a parent or spouse and declines in self-assessed health (Hurd and McGarry, 2002). Ross and Mirowsky, in another study using the Aging, Status and the Sense of Control (ASOC) survey, demonstrate that individuals who report higher levels of social support also have higher subjective life expectancy. While the main focus of their study was the causal mechanism linking social support and subjective life expectancy, the authors also find that subjective life expectancy

is associated in the expected direction with survival of parents and adult children, marital status (for men), current health status, and health behaviors such as smoking, drinking, and poor nutritional habits (Ross and Mirowsky, 2002).

Evidence from less developed countries indicates that adults make more accurate assessments of the level and direction of changes in infant and child mortality in comparison to their own mortality. Delavande, and Kohler find that adults in Malawi, a country challenged by the HIV/AIDS epidemic, generally overestimate their own mortality risks but make more accurate assessments of the level of infant mortality (Delavande and Kohler, 2007). Similarly, in a study in Bangladesh, survey respondents accurately perceived that both infant and child mortality were declining. In contrast, respondents believed that adult mortality rates were worsening even though the inverse was true. Respondents cited degradation in food quality as the reason for increases in mortality. They perceived the new food system that had been imported into the country as having negative implications for health (Amin and Basu, 2004).

Perceptions of the quality of traditional versus modern methods of food production, medical care, etc. influence the perception of mortality. Montgomery argues that one of many obstacles to perceiving mortality decline is that individuals have to accept the notion that modern medical care is good and see it as a better alternative to traditional methods. Also, he points out that acting on an assessment of lower mortality is risky. For instance, those who risk going against the status quo by having fewer children because they perceive mortality risks to be lower may be stigmatized by others in the community (especially if their children end up dying) (Montgomery, 2000).

The results in this section suggest that individuals can not necessarily make accurate assessments of their own mortality risks even though their assessments are correlated with mortality risk factors and predictive of their own mortality. In terms of the possible impact on fertility, however, these results suggest that potential parents would be more likely to perceive accurately both level and changes in infant and child mortality in comparison to their own mortality risks.

### **3.2 Value of certainty in timing of death**

One of the major difficulties in evaluating the value of certainty in timing of death is distinguishing preference for longer average life span from preference for lower variability of age at death. As was discussed in Chapter 2, during the mortality transition, life expectancy and variability of age at death are strongly negatively correlated with rapid improvements in mortality in childhood and young adulthood resulting in both higher life expectancy and reduced variability. Health behaviors that might be interpreted as improving certainty in timing of death (e.g. avoiding smoking, vaccinations, wearing seat-belts, etc.) could just as easily be attributed to desire to lengthen life span as the adoption of these behaviors should promote both greater longevity and greater certainty about the timing of death.

In a recent paper, Edwards attempts to disentangle the preference for greater certainty in timing of death from the desire for greater longevity (Edwards, 2009a). The theoretical model that he builds based on the ratio of the expected utilities from both allows one to determine the amount of life expectancy one would be willing to give up in order to be more certain about the timing of death. Given standard assumptions about time discounting, Edwards finds that an average person would generally be willing to give up a half a year of mean life span for a one year reduction in variability of age at death. Incorporating data from studies on individual response to mortality risk, which attempt to estimate the monetary value of a year of life, Edwards suggests that fetuses choosing between being born in Sweden or the United States would be willing to pay \$60,000 in order to be born in Sweden (\$40,000 for a roughly two year increase in mean age at death for those surviving to age 10, 77.7 years versus 80 years, and \$20,000 for a two year decrease in the standard deviation of ages at death above age 10, 15 years versus 13 years) (Edwards, 2009a).

While this numerical example is intended to demonstrate the monetary value of certainty in timing of death, the use of Sweden and the United States as case studies highlights one deficiency of Edwards's theoretical model, which is that he does not account for quality of life. While it is trivial that Edwards ignores that immigration data suggests that the United States is a more desirable place to live than Sweden, a more serious omission is that he does not account for morbidity in his model. The question of the value of longevity and certainty becomes much more complicated when one tries to account for whether these years of extra life will be lived in good health and what level of uncertainty accompanies the transition to ill-health. A variety of measures of health expectancy have been proposed, which take into account both life span and time spent living in ill-health, and it would be useful for Edwards and others working in this area to try to incorporate these measures when estimating the value of certainty in timing of death (Murray et al., 2002). In the next section, I touch on this subject again when I discuss whether mortality compression and increases in life expectancy have been accompanied by a compression or expansion of morbidity.

### 3.3 Biological implications

In Chapter 2, I presented a broad overview of the literature on the rectangularization of the survival curve (mortality compression) and the possible transition to an era of shifting mortality where life expectancy continues to increase while variability of death remains constant. The central biological assumption of the rectangularization hypothesis proposed by Fries is that humans are subject to a fixed biological limit to life span (Fries, 1980). While Fries believed that morbidity was malleable and could be compressed into an increasingly narrow range, he assumed that the human maximum attainable age could not be altered.

As discussed in the review, since Fries proposed his theory, many researchers have offered evidence against his hypothesis. With improvements in data collection on mortality at the oldest ages, researchers have been able to document that mortality rates are decreasing even

at the oldest ages, that the tail of the survival curve has been shifting upward to older ages, and that maximum life duration is increasing (Kannisto et al., 1994; Wilmoth and Lundström, 1996; Cheung and Robine, 2007). In terms of trends in variability of age at death, relative stability or even expansion in these measures has been observed over the past half century (Myers and Manton, 1984a; Myers and Manton, 1984b; Wilmoth and Horiuchi, 1999; Edwards and Tuljapurkar, 2005). Along with linear increase in the maximum observed life expectancy across countries, these results suggest that either the maximum age at death is increasing or humans are not yet approaching a fixed limit to life span (Oeppen and Vaupel, 2002).

If the maximum attainable age at death is increasing and this increase is not solely due an increase in population size (Cheung and Robine, 2007), this suggests that human beings are in a sense becoming more durable over time. In the biodemographic literature, there is support for humans being built to last longer than other species. In a comparison of human life span distributions with the distributions of five species of invertebrates, Horiuchi finds very striking differences between humans and invertebrates (Horiuchi, 2003). The life span distributions of the invertebrates are more symmetrical around the mode in comparison to the human distribution, which is skewed toward the left. In humans, a majority of the distribution is concentrated in a narrower interval in comparison to the distribution of invertebrates implying lower variability in timing of death for humans. Lower variability in human ages at death in comparison to invertebrates (even two genetically homogenous groups of invertebrates) indicates that humans are subject to greater quality control. Horiuchi also observes that mortality increase across age accelerates in humans during the younger old ages and decelerates at the older old ages while for invertebrates mortality increase just decelerates across age. In order to explain this phenomenon, Horiuchi proposes a repair-senescence hypothesis, which suggests that while highly effective repair mechanisms extend human life, the deterioration of these repair mechanisms as humans age results in the acceleration of mortality increase across age.

The notion that humans are being built to last longer is also consistent with the concept of technophysio evolution offered by Fogel to explain improvements in mortality from the 18th to 21st century (Fogel, 2004). According to Fogel, improvements in nutrition have allowed mothers to give birth to relatively larger babies, who are not only less likely to die in infancy but are also better protected against disease and death throughout their life course. Conditions in utero and fetal development are thought to be related to many health outcomes later in life (Barker, 1995). Fogel's theory of technophysio evolution coupled with Barker's fetal origins hypothesis suggest that as conditions in utero and in early life improve so should health in later life. Whether these improvements have in fact been able to delay the aging process is a central question facing researchers today.

Improvements in life expectancy coupled with mortality compression have had the combined effect of increasing the number of older persons in the population. As the elderly population experiences tremendous growth, questions arise as to how costly it will be to support this aging population (Fuchs, 1984). Of central importance to answering this ques-

tion is whether delays in the age of death have been matched by delays in the onset of disability. As life expectancy has improved, the time spent in ill-health or living with a disability has either compressed, expanded, or maintained a state of dynamic equilibrium (Nusselder, 2003).

Determining whether increases in life expectancy have been met with declines in the number of years spent in ill-health or with disability has been a major issue addressed by the Network on Health Expectancy (REVES). The internationally based studies of trends in disability-free life expectancy produced by this group indicate that the most severe forms of disability have declined, the less severe forms of disability have increased, and as a result there is stagnation in trends for overall disability (Robine et al., 2003). Trends in disability-free life expectancy are more consistent across countries when the disabilities included are restricted to the most severe forms. While the prevalence of chronic diseases and less severe forms of disability has increased as mortality rates have declined at older ages, the types of conditions individuals are suffering at older ages are less severe than those suffered by their predecessors indicating that increases in life expectancy have produced “a pandemic of light and moderate, but not of severe disabilities” (Robine et al., 2003, p. 119).

In a study of trends in disability-free life expectancy in the United States, Freedman et al. perform a careful analysis of trends in disability prevalence among the elderly in the United States in the 1980s and 1990s using five nationally representative data sets (Freedman et al., 2004). This analysis seeks to resolve discrepancies in trends reported for the US due to differences across studies in the period covered, the definition of disability utilized, the method of age standardization, and whether or not the study population includes the institutionalized population. The results of their analysis suggest a decline during the 1990s in the proportion of the elderly population (above age 70) reporting difficulty with daily activities and relying upon help for daily activities; however, the trends for elderly persons reporting either getting help or using equipment remained flat suggesting that the elderly are perhaps coping with their disabilities by substituting equipment for help from other persons.

In contrast to the studies above, which support either the expansion of (less severe) morbidity or dynamic equilibrium hypotheses, a recent cohort analysis of trends in disability in the United States by Manton et al. produced evidence for the compression of morbidity hypothesis as they found that the younger cohort in their study enjoyed both a longer life span and less time spent disabled in comparison to the older cohort (Manton et al., 2008). The mixed evidence presented here along with the many issues in defining disability and monitoring trends over time suggest that continued research is needed to determine whether increases in life expectancy and greater certainty in timing of death are accompanied by morbidity compression, expansion, or dynamic equilibrium.

## 3.4 Economic implications

In this section, I examine some of the economic implications of reductions of variability in age of death. The literature reviewed in this section suggests that uncertainty about the timing of death is costly.

### 3.4.1 Investment in education

As Preston, Keyfitz, and Schoen state in the introduction to their collection of life tables, a society where death is more likely to occur at older ages is one “that is more willing to train individuals for the ‘long-run’” (Preston et al., 1972). Indeed, as life expectancy increases and the variability of ages at death decreases, it becomes more profitable to invest in children’s human capital. Lee and Goldstein summarize this sentiment in their speculative piece on a possible rescaling of the life course due to increased longevity:

“From the point of view of a rational life cycle planner, an extended period of quasi-adulthood probably makes sense for those who can expect to live a long time. A long investment horizon makes it worthwhile to invest more in one’s own human capital and stay in school longer. Likewise, it makes experimentation less costly and potentially more rewarding.”

-Lee and Goldstein (Lee, 2003)

As the rate of return on investments in human capital increases, parents and society alike will increase their investments in education. The duration of education increases as the length of time it is profitable to invest in one’s education increases. While formal education used to begin and end in childhood if it began at all, it now extends into early adulthood. The encroachment of education into adulthood has delayed other processes which used to signal entry into adulthood like marriage and childbearing. Lee and Goldstein observe that individuals are finishing their education at older ages relative to the past and that both mean age at first marriage and mean age at first birth are also increasingly rapidly. They note that the pace of changes in these measures is actually faster than the pace of changes in life expectancy (Lee, 2003). The implications of increasing investment in education for fertility trends will be discussed in more depth in Section 3.6.2.

### 3.4.2 Retirement, savings, insurance, annuities, old age support

Edwards and Tuljapurkar review a number of economic implications of uncertainty in timing of death (Edwards and Tuljapurkar, 2005). They suggest that the impact of greater uncertainty about timing of death on age at retirement is unclear. Persons might retire earlier if life span is more uncertain if they view retirement as a benefit for years of work; however, they might also be inspired to work longer in order to accumulate savings because they are

more uncertain about the transition to ill-health. Likewise, it is difficult to determine how uncertainty about timing of death will affect saving behaviors because it is not clear whether bequests are intentional or circumstantial (Edwards, 2009a).

Uncertainty in timing of death leads to extra costs in the insurance and annuities markets. Insurance companies charge higher prices under conditions of greater uncertainty (and less equitable distribution of deaths) in order to avoid losing money in case only those who expect to live longer buy their policies (Edwards and Tuljapurkar, 2005). At the individual level, uncertainty is costly even when the individual has an actuarially fair annuity. While income from the annuity covers consumption costs, it cannot account for the utility risk associated with uncertainty in life span (Edwards, 2009a).

Greater inequality in mortality outcomes as implied by greater uncertainty in timing of death at the population level is also costly for a public pension system. The costs of supporting those who live longer are not balanced by earlier deaths because income is collected by the dependents of those who die early. Also, those who die earlier have contributed less to the system and those who live longer collect greater benefits as the poor have a survival disadvantage (Edwards and Tuljapurkar, 2005).

Because uncertainty about timing of death is costly, examining differences in uncertainty across groups (e.g. highly educated versus those with lower education attainment) represents a distinct way of measuring inequality across groups beyond what can be learned by evaluating differences in life expectancy or socioeconomic status (Edwards and Tuljapurkar, 2005).

### 3.5 Psychological implications

In a review of possible implications of declines in variability of age at death, Wilmoth and Horiuchi suggest that changes in certainty about timing of death have the potential to alter human attitudes towards death (Wilmoth and Horiuchi, 1999). On the one hand, it may be that people are less afraid of death in situations of greater certainty because death tends to occur in a more “natural” order (i.e. death is expected among elderly persons and their loss does not have as much of an effect on those who survive because their children have likely already transitioned to adulthood). On the other hand, greater certainty could heighten fear of death because deaths that do occur outside of the “natural” order are more traumatic. In support of the second line of reasoning, Wilmoth and Horiuchi cite a study of attitudes towards death among impoverished persons in Brazil suffering high rates of mortality in infancy and childhood, which shows that mothers are relatively indifferent to the deaths of their children (Scheper-Hughes, 1992; Wilmoth and Horiuchi, 1999).

Historical evidence from the United States suggests that maternal attitudes surrounding infant death changed markedly from the mid 18th to early 20th century (Dye and Smith, 1986). While in earlier eras, Americans held a deterministic attitude toward infant deaths (i.e. infant deaths were the result of God’s will), this gave way to a recognition of the role of

human agency during the Progressive Era, which in turn put pressure on mothers to ensure their children's survival. Until progress in medicine and public health improved infant and child survival outcomes around the turn of the 20th century, American mothers experienced a great deal of anxiety because they felt responsible for their children's survival but had few tools available to them to protect their children from early death (Dye and Smith, 1986).

## 3.6 Demographic implications

In this section, I consider the demographic implications of certainty in timing of death. I consider recent work on the implications of variability of age at death for modeling mortality differences among subgroups and mortality forecasting. Then, I draw on the rich literature surrounding demographic transition and fertility theory in order to understand how changes in certainty of timing of death might affect fertility behavior. I then explore the implications of increased survival and greater certainty in timing of death for population growth and family structure.

### 3.6.1 Mortality Modeling and Projections

As noted in my review of the literature in Chapter 2, in their 2005 article, Edwards and Tuljapurkar demonstrate that differences in variability of age at death are an important component of differences in death distributions among highly developed countries and that there are significant differences in certainty in timing of death among socioeconomic and racial subgroups in the United States (Edwards and Tuljapurkar, 2005). In a more recent paper, these authors examine the implications of variability of age at death for mortality modeling and mortality projection (Tuljapurkar and Edwards, 2009).

With regards to differences in mortality among subgroups, Tuljapurkar and Edwards point out that the method commonly utilized to model differences in mortality among subgroups, the Cox proportional hazard model, assumes that the mortality curves of different subgroups share the same slope. Assuming the age-specific mortality rates for each subgroup can be characterized by a Gompertz curve implies that the different subgroups experience the same variability in ages at death; however, as comparisons across socioeconomic and racial groups in the United States demonstrate, variability in age at death differs across subgroups thus this basic assumption is violated.

Tuljapurkar and Edwards also review how two mortality forecasting methods, the shifting mortality model (Bongaarts, 2005) and the Lee-Carter model (Lee and Carter, 1992), capture trends in variability of age at death (Tuljapurkar and Edwards, 2009). While they acknowledge that Bongaart's shift model accurately captures the mortality experience of industrialized countries in the latter half of the 20th century in terms of statistical fit, they caution against adopting the model for the long term because of the assumption that variability of age at death will continue to remain stable. While the Lee-Carter model does

well at reflecting the long term average trend in variability of age at death, it fails to capture the more “convoluted” path that trends in variability have taken in comparison to life expectancy for which trends are much smoother.

The evidence presented by Tuljapurkar and Edwards suggests that the mortality models currently being used by demographers are failing to capture accurately trends in variability of age at death and differences in variability of age at death across groups.

### 3.6.2 Fertility

When considering how mortality change might affect fertility behavior, it is important to recognize that there are different components of mortality change that might eventually impact reproductive behavior. In the context of classical demographic transition theory, the focus is often on changes in infant and child mortality; however, one could also consider how fertility might be impacted by trends towards greater certainty in timing of death of which improvements in infant and child mortality play a major role.

In order to examine the relationship between total fertility and variability of the adult death distribution (which is the main focus of the dissertation), I take advantage of the long historical series of fertility and mortality statistics available for Sweden through the Human Fertility Database and the Human Mortality Database (HFD, 2010; HMD, 2009). Figure 3.1 depicts the trends in period standard deviation of ages of death above age 10,  $S_{10}$ , and the period total fertility rate,  $TFR$ , for Swedish females from 1891 to 2007. These two measures follow the same course around the turn of the 20th century, stability followed by a period of sharp decline, but the trends diverge around the baby boom era as the  $TFR$  increases despite continued reductions in variability of age at death. In Figure 3.1(c), which shows the relationship between the two quantities over this time period, this divergence in trends is also apparent. Overall, though, the graph does imply that greater certainty in timing of death is associated with lower fertility. Below, I explore possible causal mechanisms for this relationship drawing on demographic theory.

Classical demographic transition theory holds that mortality declines first and then later fertility decline occurs. It is a matter of debate whether declines in fertility are a direct response to improvements in mortality (Davis, 1963) or whether declines in fertility like declines in mortality are the result of modernization but lag behind because of societal resistance to changes in fertility norms (Notestein, 1945). Since these alternate theories of demographic transition have been proposed, much research effort has been put forth to understand better the linkage between fertility and mortality over the course of the demographic transition. Decades of research have continued to reveal the complexity of this relationship as it has been discovered that fertility decline sometimes precedes mortality decline (as was the case in the United States) and that fertility decline occurs under a wide variety of social, economic, and mortality conditions (Montgomery and Cohen, 1998).

Preston proposes four possible direct mechanisms linking infant and child mortality and fertility behavior: (1) lactational disruption, (2) replacement strategy, (3) insurance function,

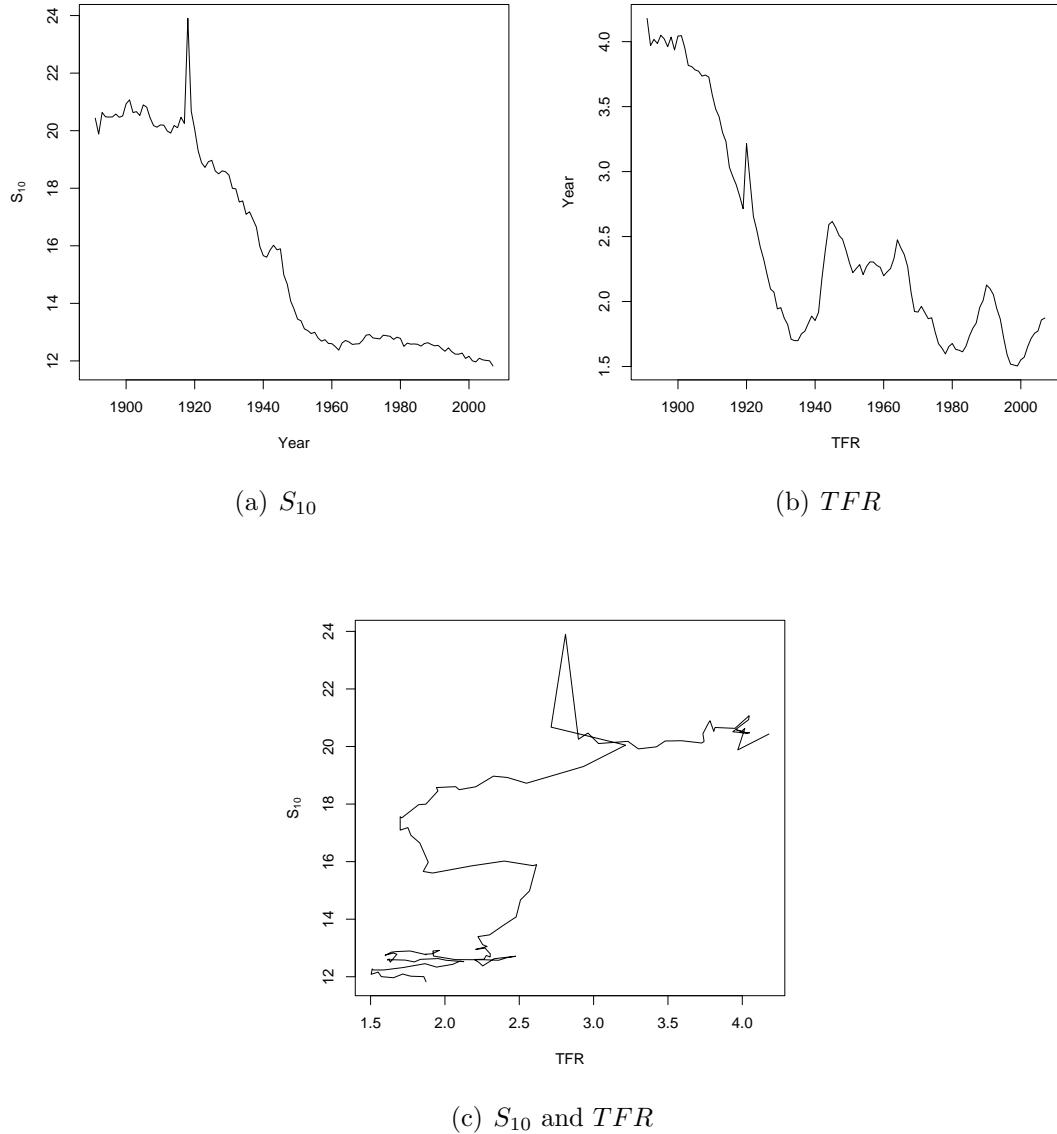


Figure 3.1: Trends in  $S_{10}$ , TFR, and the relationship between the two for Swedish females, period data, 1891-2007. Data source: Human Mortality Database and Human Fertility Database (HMD, 2009; HFD, 2010)

and (4) extra-familial (societal level) effects (Preston, 1978). Of particular relevance for the implications of certainty in timing of death on fertility is the insurance function since this mechanism assumes that parents take into account the likelihood of their children surviving when deciding how many kids to have; unfortunately, this is also the mechanism which is least well understood (Montgomery and Cohen, 1998; LeGrand et al., 2003).

The lactational disruption mechanism seems to be most important in pre-transitional societies where uncertainty about child survival is so high that parents are not able to insure adequately against losses if they adopt limiting behaviors (Lloyd and Ivanov, 1988; Montgomery and Cohen, 1998). In this case, higher mortality promotes higher fertility because birth intervals are reduced as lactational amenorrhea ceases when an infant dies. In contrast, the replacement mechanism seems to be most important in post transitional societies when the desired number of children is few and mortality conditions are favorable. In the intervening transitional period, where mortality conditions can be changing rapidly, the insurance function is thought to be most important mechanism (Preston, 1975; Lloyd and Ivanov, 1988; Montgomery and Cohen, 1998).

In a study examining fertility behaviors in Senegal and Zimbabwe, LeGrand et al. find evidence of parents employing insurance behaviors in the more developed regions within these countries while fertility regulation practices had not yet been adopted in more rural areas and among older cohorts consistent with the ordering outlined above (LeGrand et al., 2003). Although fertility behaviors have fundamentally changed in these contexts, residents of these countries do not associate these changes directly with improvements in child survival. LeGrand et al. maintain that the effect of insurance adopting behaviors on fertility is modest and that possible indirect effects are more important in explaining the relationship between child mortality and fertility.

There are many indirect pathways linking mortality and fertility. As discussed in Section 3.4.1, greater certainty in timing of death increases the returns to investments in education. Investment in education could affect fertility in a number of ways. For instance, education delays entry into marriage and childbearing, which decreases total life time fertility (Lee, 2003). Additionally, beyond timing effects, the traditional rational economic models of fertility would predict that greater investments in the education of women and greater opportunities for women in the marketplace will result in lower fertility because the opportunity cost of having children will increase (Soares, 2006). The quantity-quality model would also predict lower fertility as incomes and educational opportunities rise because parents will want to invest in the quality of each of their children rather than just maximizing quantity (Becker, 1981). LeGrand et al. find in their study of fertility in Zimbabwe and Senegal that parents adopt a mixed family building strategy, which involves limiting fertility in order to be able to make investments in quality of children but also having an extra child or two over their desired family size in order to insure against possible child death (LeGrand et al., 2003).

Assuming that parents have some sense of the likely survival probabilities of their children as the review of the literature in Section 3.1 suggests, the evidence presented here indicates

that there are many pathways through which increases in the certainty of timing of death coupled with increases in average longevity could reduce desired fertility. Indirectly, greater certainty in timing of death raises the returns to education. These investments in education and the potential returns to education lower fertility both by delaying entry into marriage and childbearing as well as encouraging smaller family size in order to produce children of higher quality (at least with respect to their educational level). In terms of a direct link, greater certainty about mortality allows parents to engage in more deliberate family building first by planning to have one or two extra kids (insurance effect) and later in the fertility transition to be able to only have another child after achieving desired family size if the death of another child necessitates it (replacement effect).

### **3.6.3 Population Growth and Family Structure**

As Preston, Keyfitz, and Schoen state, “a population in which death is almost always deferred until the older ages is one that grows faster” (Preston et al., 1972). As noted in this last section, increased survival prospects for children act to lower desired family size at an individual level; however, this does not mean that the number of births decreases at population level since more individuals are surviving to and through their childbearing ages as deaths are compressed into older ages. For example, in a synthetic cohort experiencing the period mortality rates of Swedish females in 1751-1754, around 37% of the cohort would die by age 15, the beginning of childbearing years. Another 28% of the synthetic cohort would die before the end of their childbearing years around age 50. In contrast, in a synthetic cohort experiencing the mortality rates of the Swedish female population in 2005-2006, less than 3% of the population dies between birth and age 50.

The figures on female survivorship from Sweden highlight another important aspect of compression of mortality into older ages. Because females (as well as males) are unlikely to die during their reproductive years, children are at considerably lower risk of being orphaned during childhood and married persons are much less likely to be widowed during their reproductive years in comparison to earlier generations. As a result, as Preston, Keyfitz, and Schoen suggest, in these low mortality settings, persons “can rely more heavily on the nuclear family for emotional gratification” (Preston et al., 1972). Just considering increased survival, however, obscures another phenomenon that is increasingly disrupting the nuclear family unit: divorce. This phenomenon may also be related to longer life span and increased certainty in timing of death. Persons unhappy in their marriage may be more likely to seek divorce if they expect their partner to live a long time and if their own survival prospects are such that they have many years to seek out a new relationship.

### 3.7 Conclusion

In this chapter, I have tried to highlight the many ways that certainty in timing of death impacts human life. Vast changes in the mortality landscape have taken place over the last century, and, in most areas of the world, humans now enjoy much more certainty about their survival prospects. While this may increase the trauma of early deaths for a decedent's social network, these changes have likely lessened anxiety overall about the possibility of dying and empowered humans with a greater sense of control over their own lives. As Preston, Keyfitz, and Schoen observe, a society in which death is delayed until later ages is one in which members "can dispense more readily with fatalistic or supernatural philosophies" (Preston et al., 1972). This greater sense of agency coupled with a greater certainty about survival prospects has allowed humans to take control of another key aspect of their demographic lives: their fertility behaviors. As a result of changes in the certainty and timing of these key demographic events, childbearing and death, the course of human life and family structure have been radically altered from evolutionary history.

# Chapter 4

## Data, measures, methods

In this chapter, I provide information on the data sets, mortality measures, and statistical methods that will feature heavily in my data analysis contained in later chapters of the dissertation.

### 4.1 Data

I utilize three data sets in my analysis of trends and differentials in variability of age at death: the Human Mortality Database (HMD), the World Health Organization's collection of 1,802 life tables (WHO1802), and the French Cause-of-Death Series (FCOD). I also draw on the Human Fertility Database (HFD) in my exploration of the relationship between greater certainty in timing of death and fertility decline. I describe each of these data sets below giving particular attention to how the specific features of the data set help or hinder my planned analysis.

#### 4.1.1 Human Mortality Database

The Human Mortality Database is the preeminent collection of high quality mortality data representing the historical mortality experience of a number of countries around the world (HMD, 2009). Data from the HMD can be downloaded from the following website: [www.mortality.org](http://www.mortality.org). The HMD was created in order to allow researchers to explore the longevity revolution of which greater certainty in timing of death is a key component. Using data from the HMD, I am able to explore the historical mortality patterns of populations in thirty-seven distinct geographic areas using life tables with single year age groups that extend up to age 110. I utilize both the series of single year life tables and the series of life tables divided into multi-year periods (typically five). While both cohort and period life tables are available in the HMD, I rely on the set of period life tables for my analysis. The period of observation covered in a life table series varies by area, but extends as far back as

1751 in the case of Sweden. Table 4.1 offers a overview of the countries and time periods represented in the Human Mortality Database. In total, the database reflects over 72 billion person-years of data (Wilmot et al., 2009).

While a number of countries are included in the HMD, the data base is largely representative of the experience of more developed countries because of the stringent standards for data quality required by the creators of the database. These standards include nearly complete vital statistics and census records for each country. Number of deaths by age for each period, taken from vital statistics records, are used to construct the numerators of the age-specific mortality rates. Problems with accurate counts of deaths by single year of age arise because the raw data is sometimes grouped into larger age categories (e.g. five year age groups). The creators of the HMD have developed sophisticated methods to distribute deaths proportionally over age categories in order to find death counts for single year age groups (Wilmot et al., 2007b).

Age misreporting, which is thought to be less prevalent in death records in comparison to census records, presents an issue when constructing the denominators for age-specific mortality rates from census records, especially at higher ages. Person years at risk, the denominator of the age-specific mortality rates, is estimated based on population size in a particular age group. In order to guard against the problem of age misreporting above age 80, estimation of population numbers for those above age 80 is achieved by one of three methods: (1) by the extinct generations methods for extinct cohorts, (2) by the survivor ratio method for non-extinct cohorts above age 90, (3) by inter-censal survival methods or another data source for non-extinct cohorts between ages 80 and 90 (Wilmot et al., 2007a).

Another important issue regarding mortality estimates at older ages is the tendency for age-specific mortality rates to be erratic because the population is sparse at the oldest ages. In cohort life tables included in the HMD, the age-specific mortality rates at the oldest ages are not smoothed because these rates reflect the real experience of the birth cohort. For the period life tables, on the other hand, age-specific mortality rates above age 80 are smoothed using the Kannisto model of old age mortality and estimating hazard rates assuming an asymptote equal to one (Thatcher et al., 1998). This model is also used to reconstruct age specific mortality rates for single years of age through age 110+ in cases where the open ended interval in the raw data starts at a younger age such as 80 or 90 (Wilmot et al., 2007b).

How do these adjustments to age-specific mortality rates potentially impact the analysis that I pursue in the dissertation? Smoothing the period age-specific mortality rates above age 80 likely produces a smoother distribution of age-specific deaths than would be observed in reality; however, since the erratic nature of mortality rates is largely due to random variation, the smoothing of these rates likely produces more stable estimates of variability in age at death. The measure of variability of age at death that I largely rely on for my analysis, the standard deviation of ages at death above age 10,  $S_{10}$ , incorporates data from most of the age range. Even though the standard deviation is sensitive to the tails of the distribution, the limited number of deaths that occur at the extremes likely means that the measure would

not be highly influenced by eccentricities in the data at the oldest ages. This issue is of more concern for those who study mortality compression by examining the standard deviation of the one tailed death distribution above the modal age at death ( $SD(M+)$ ) (Kannisto, 2001).

#### 4.1.2 World Health Organization 1,802 life tables

More geographic areas from less developed regions are included in the World Health Organization's collection of 1,802 life tables (Murray et al., 2003). In the dissertation, I use data from this collection to assess whether the historical trends in variability of age at death differ for countries and areas of less developed regions in comparison to more developed. A description of the life tables included in this collection that do not overlap with the life tables included in the Human Mortality Database are presented in Table 4.2. As can be seen in this table, for many of the non-overlapping geographical areas, data is only available for a single point of time.

The WHO1802 life tables were collected as part of an effort to develop a new model life table system based on empirical data reflecting the experience of a broad range of countries in the 20th century. As such, this collection only includes life tables estimated directly from data on deaths and population counts, that is, the collection does not include any life tables developed using model life tables (Murray et al., 2003). Unlike the life tables from the Human Mortality Database, the life tables in this collection feature five year rather than single year age groups. While it is still possible to calculate variability in age at death using this data, the estimates are less precise than estimates of variability calculated using one-year age groups.

Originally, the open ended interval for these life tables began at age 85, but the life tables were extended to age 110 using the Kannisto model (similar to the methods used for the HMD data set) (Thatcher et al., 1998)<sup>1</sup>. The life tables were extended in order to make a more accurate assessment of variability of age at death for the analyses of trends and differentials in variability of age at death pursued in the dissertation.

#### 4.1.3 French Cause-of-Death Series

In Chapter 8, I explore the reasons for the emergence of the gender gap in variability of age at death and the transition from mortality compression to shifting mortality by examining changes in age-sex-cause-specific mortality rates. The data used for this analysis comes from the French Cause-of-Death Series that covers the period 1925 to 1999. These data were compiled by France Meslé and Jacques Vallin and standardized to the 9th revision of the ICD (Vallin and Meslé, 1988; Meslé and Vallin, 1996; Vallin and Meslé, ). These authors provided me with the data directly for this project, but the data is available at <http://www-causfra.ined.fr>. For my analyses, I use the cause-of-death series to find the

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<sup>1</sup>The extension of the life tables was performed by John Wilmoth (Wilmoth et al., 2009).

proportion of deaths attributable to a particular class of causes for each sex by age in each period, and then apply these proportions to the total age-specific mortality rates from the HMD in order to obtain an age and cause-specific mortality rates for each sex by period.

The cause-of-death decompositions are carried out using the following classes of causes: infectious diseases, respiratory diseases, digestive diseases, neoplasm, cerebrovascular diseases and unspecified disorders of the circulatory system, heart diseases, external causes, maternal causes, and mental disorders. The remaining causes are grouped into the “other” category. Additionally, there is a category corresponding to deaths that have been attributed to ill-defined or unknown causes. I outline my reasons for choosing this particular classification system in Chapter 8.

Before standardizing the cause-of-death data to the 9th revision of the ICD, the cause-of-death series included data with deaths classified according to many different revisions of the ICD. In their article describing the construction of the French Cause-of-Death series, Meslé and Vallin caution that the data relating to periods prior to 1950 can not be linked as easily with the post-1950 data as the transitions between the 5th to 6th and 5th to 7th revisions of the ICD were extremely complex (Meslé and Vallin, 1996). The potential problem that this poses for my analysis is that the results I obtain might be due to misclassification of deaths rather than real changes in cause-specific mortality. Of special importance, the proportion of deaths classified as ill-defined or unknown changes over time. I discuss potential problems as it relates to my analysis in more detail in Chapter 8.

#### **4.1.4 Human Fertility Database**

In Chapter 3, I discuss the possible link between greater certainty in timing of death and declines in fertility. For this analysis, I utilize a series of Swedish period total fertility rates covering the period 1891-2007 from the Human Fertility Database (HFD, 2010). This data is available for download at the following web address: [www.humanfertility.org](http://www.humanfertility.org). Similar to the Human Mortality Database, the Human Fertility Database, a more recent project, has been designed to be the preeminent source of high quality series of national macro-level fertility data. The fertility measures featured in the HFD collection are based on complete or nearly complete vital registration data on births and accurate estimates of population counts in the range of female reproductive ages (in order to determine person years of exposure to risk of pregnancy).

## **4.2 Measures**

A broad overview of the measures available to assess mortality compression and mortality inequality is presented in Chapter 2. Here, I briefly review select measures. I also lay out my reasoning for adopting the standard deviation of age at death above age 10,  $S_{10}$ , as my measure of choice for the most of the analyses which I carry out in the dissertation.

The majority of measures that I utilize in my work capture some type of variability in the life table death distribution. Differences among these measures of variability of age at death arise because of differences in the age range covered by the measures, differences in the central indicator used in the calculation of dispersion (mean versus mode), and whether or not the measurement is attached to fixed percentiles of the death distribution. Below, I define the measures of variability of age at death, which I utilize, and Figure 4.1 visually depicts the differences among these measures. These measures were also listed in Chapter 2.1.1, but here I provide greater detail on how these measures are calculated.

In the following definitions,  $x$  indicates age,  $l_x$  indicates survivorship to age  $x$ ,  $e_x$  is life expectancy at age  $x$ ,  $d_x$  indicates the number of deaths that occur at age  $x$ ,  $a_x$  is the average time lived from age  $x$  to  $x+1$  for those dying in the interval,  $M_{10}$  is the average age at death for those who survive to age ten ( $e_{10} + 10$ ),  $\bar{M}$  is the modal age at death, and  $T$  indicates the oldest age group. With the exception of  $M_{10}$  and  $\bar{M}$ , these measures are taken directly from the period life tables contained in the HMD and the WHO collection of 1,802 life tables.

- $IQR$ -inter-quartile range, age span between the 25th and 75th percentiles of the cumulative death distribution (Wilmoth and Horiuchi, 1999).

$$IQR = x_2 - x_1$$

where  $l_{x_2} = .25$  and  $l_{x_1} = .75$ .

- $C50$ -age span corresponding to the most compressed 50 percent of deaths from the death distribution (Kannisto, 2000).

$$C50 = x_2 - x_1$$

where  $l_{x_1} - l_{x_2} = .5$  and where  $x_2 - x_1$  is minimized.

- $e^\dagger$ -person years lost in the life table due to early death (Zhang and Vaupel, 2009).

$$e^\dagger = - \int_0^\infty l_x \log(l_x) dx = \int_0^\infty e_x d_x dx$$

- $\bar{H}$ -a measure often called “life table entropy”

$$\bar{H} = \frac{- \int_0^\infty l_x \log(l_x) dx}{\int_0^\infty l_x dx}$$

(Keyfitz and Caswell, 2005). Note that life table entropy  $\bar{H}$  is related to  $e^\dagger$  through the following formula:  $H = \frac{e^\dagger}{e_0}$ .

- $S_0$ -standard deviation of ages at death unconstrained by age (Edwards and Tuljapurkar, 2005).

$$S_0 = \sqrt{\sum_{x=0}^T (x + a_x - e_0)^2 \times d_x}$$

- $S_{10}$ -standard deviation of ages at death above age ten (Edwards and Tuljapurkar, 2005).

$$S_{10} = \sqrt{\frac{\sum_{x=10}^T (x + a_x - M_{10})^2 \times d_x}{\sum_{x=10}^T d_x}}$$

- $SDM$ -root mean square deviation around the modal age at death (Canudas-Romo, 2008)

$$SDM = \sqrt{\sum_{x=0}^T (x + a_x - \bar{M})^2 \times d_x}$$

- $SD(M+)$ -root mean square deviation above the modal age at death (Kannisto, 2000; Kannisto, 2001; Cheung and Robine, 2007)

$$SD(M+) = \sqrt{\frac{\sum_{x=\bar{M}}^T (x + a_x - \bar{M})^2 \times d_x}{\sum_{x=\bar{M}}^T d_x}} \quad (4.1)$$

Aside from these measures of variability of age at death, I also utilize a new measure of life span disparity recently proposed by Zhang and Vaupel,  $e^\dagger$ , the life table years of life lost due to premature mortality ( $e^\dagger = \int_0^w e_x d_x$ , where  $e_x$  is life expectancy at age  $x$ ,  $d_x$  indicates the number of deaths that occur at age  $x$ , and  $w$  is the oldest age group). A visual depiction of how this measure is calculated is featured in Figure 4.2.

From the measures listed above,  $S_{10}$  is my measure of choice for most of the analyses that I carry out in the dissertation. This measure has been popularized by the work of Edwards and Tuljapurkar (Edwards and Tuljapurkar, 2005; Edwards, 2009b; Edwards, 2009a; Tuljapurkar and Edwards, 2009), and these authors provide a number of reasons for why one might choose to use this measure over the others listed above. Most important, Edwards and Tuljapurkar note that measures which take into account deaths over the entire age range (unconditional measures) tend to be heavily influenced by trends in infant and child mortality. Using  $S_{10}$  rather than the unconditional standard deviation,  $S_0$ , or other unconditional measures listed above including  $IQR$ ,  $C50$ , and  $SDM$  focuses attention on changes in the distribution of adult ages at death. Truncation also produces a distribution that is relatively normal making it unnecessary to employ percentile based measures like  $IQR$  and  $C50$ , which would be more advantageous if the distributions were heavily skewed (Edwards and Tuljapurkar, 2005).

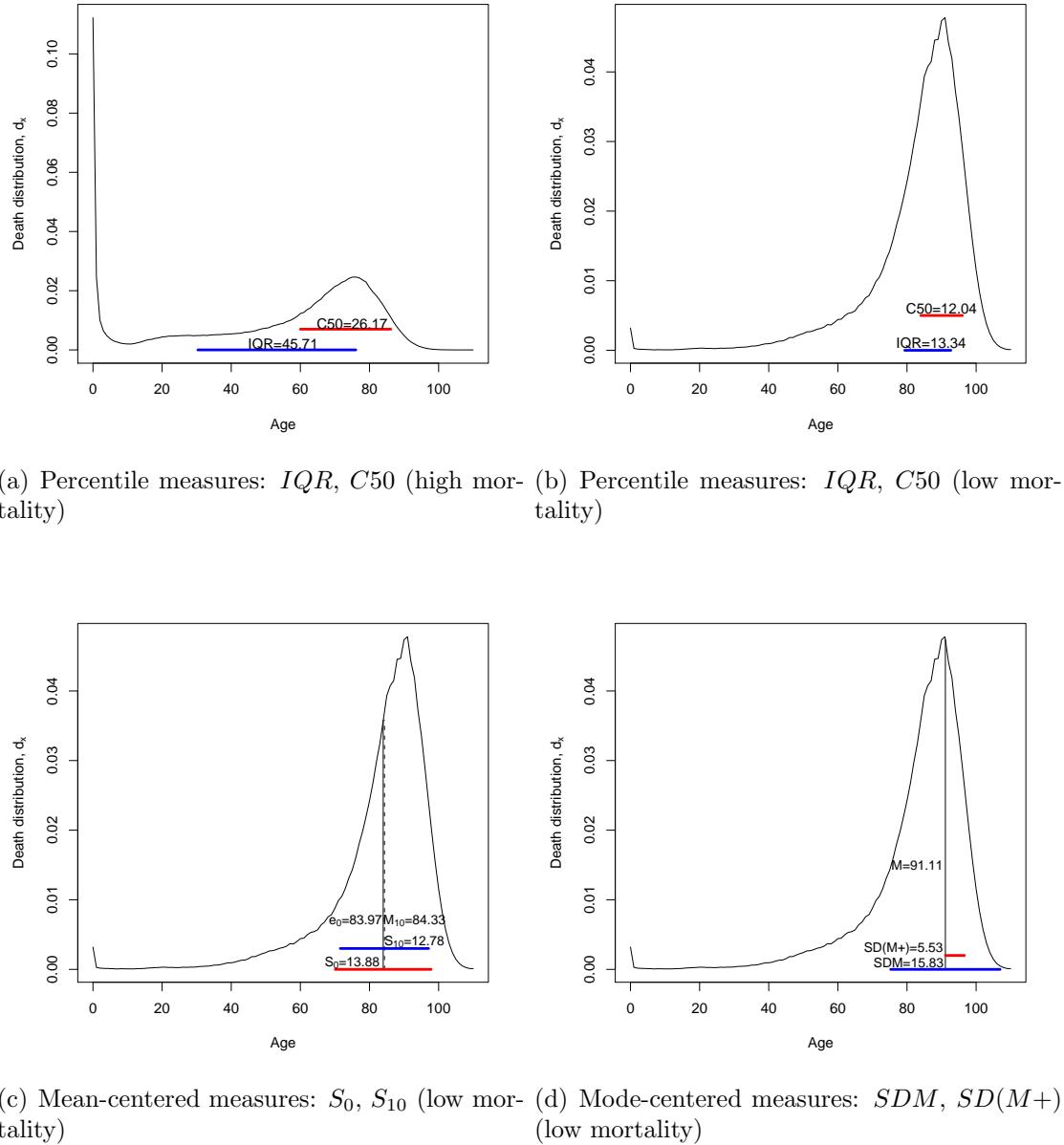


Figure 4.1: Comparison of different measures of variability of age at death. Death distribution corresponds to French females, 1910-14 (high mortality) and 2005-06 (low mortality). Data source: Human Mortality Database (HMD, 2009).

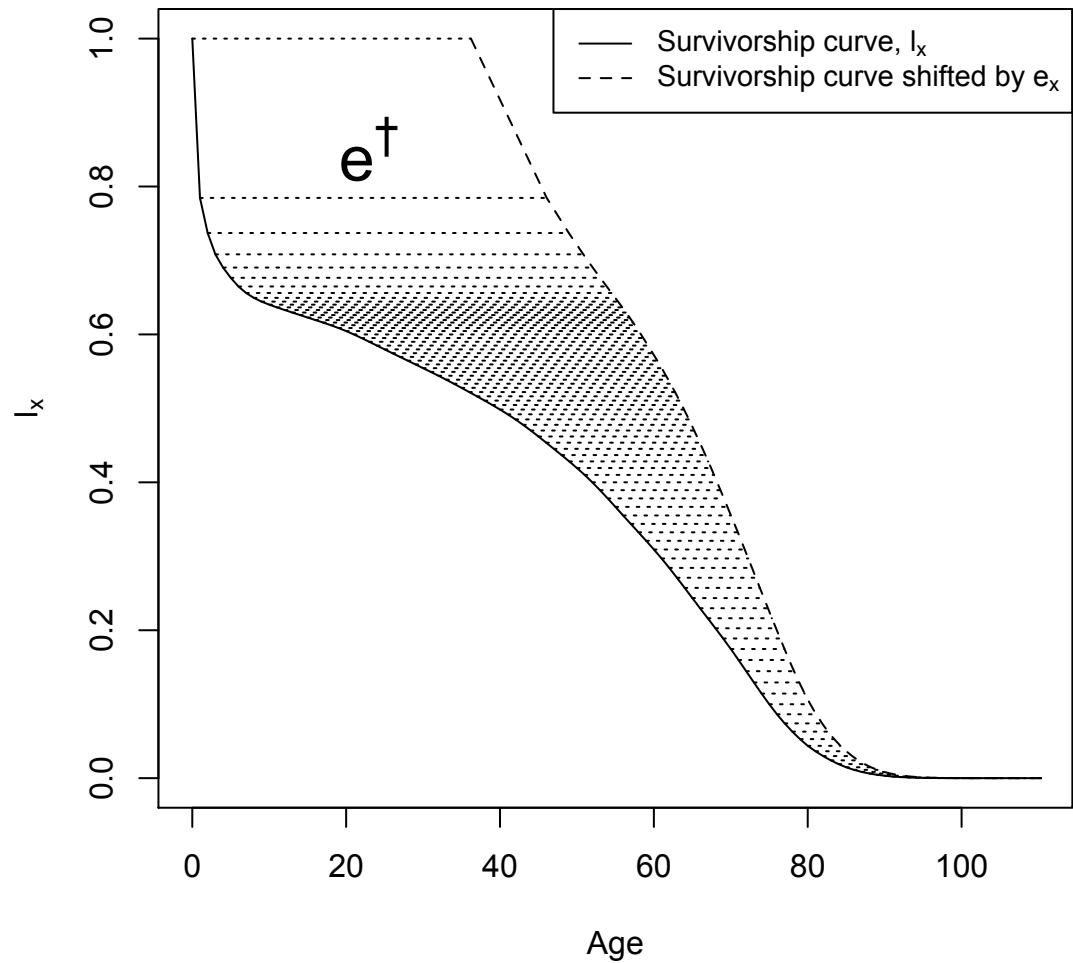


Figure 4.2: Representation of  $e^\dagger$ . The shaded area between the curves graphically represents the calculation of the measure  $e^\dagger$ . The survivorship curve,  $l_x$ , and life expectancy,  $e_x$  are representative of Swedish males in the period 1800-04. Data source: Human Mortality Database (HMD, 2009).

The standard deviation has other useful properties related to the fact that it is derived from the variance of the distribution. For instance, the variance is additively decomposable into components representing between and within subgroup inequality. Thus,  $S_{10}$  places an upper limit on the total amount of mortality inequality observed in adult ages at death for a particular population (Edwards and Tuljapurkar, 2005). Additionally, the standard deviation is invariant on an additive scale (Edwards, 2009b). Thus, if every death in a population is delayed by 10 years, the standard deviation remains the same as the death distribution just shifts to the right. In contrast, measures like the Gini or Theil index (introduced in Chapter 2), are invariant on a proportional scale, which means that a doubling of life span for every member of the population has no effect on these measures. Certainly, it makes sense to adopt a measure that is invariant to change on an additive scale to study trends in mortality compression. While some would argue that invariance on a proportional scale is preferable in a measure of inequality, especially in the case of income inequality, Edwards argues that demographers are more interested in additive differences in life span (e.g. the gender gap in life expectancy) thus a measure of inequality that is invariant on an additive scale is more desirable (Edwards, 2009b).

In a similar vein, by choosing to use  $S_{10}$  as my measure of mortality compression and mortality inequality, I am also arguing that an absolute measure of variability of age at death is more relevant than a relative measure. Smits and Monden argue that relative measures of mortality inequality are more appropriate than absolute measures. Since life expectancy and measures of variability of age at death are inversely related, these authors caution that what might be a relatively low level of mortality inequality for a country with life expectancy of sixty might be a relatively high level of mortality inequality among countries with life expectancy of seventy-five (Smits and Monden, 2009). Edwards defends absolute measures by arguing that the cost of uncertainty about timing of death does not necessarily decrease as life expectancy rises as a relative measure of mortality inequality would suggest (Edwards, 2009b). As I will demonstrate in Chapter 7, the choice of absolute versus relative measure of inequality matters when tracking trends in the gender gap in levels of mortality inequality.

Another issue in determining my choice of measure of variability of age at death is whether the mean of the death distribution or the mode is the most relevant measure of life span. Kannisto argues that mode is the more relevant measure of life span because it is not as sensitive to infant and child mortality and thus better captures the current stage of the mortality transition, the era of delayed aging, in comparison to life expectancy (Kannisto, 2001)<sup>2</sup>. Additionally, Kannisto prefers mode based measures of life span and variability like  $SD(M+)$  because they capture what Lexis referred to as normal life durations.

Kannisto cites work of Lexis in which he divided the area under the distribution of ages at death into three parts: (1) a J-curve in infancy capturing natural defects in human constitution, (2) a normal curve under the mode in late life which indicates normal life durations,

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<sup>2</sup>This argument is not so relevant for  $S_{10}$  since only life expectancy above age ten is used in the calculation of this measure

and (3) an area between the normal curve and the actual death distribution on the left hand side of the normal curve which represents premature deaths (Lexis, 1877; Kannisto, 2001). While  $SD(M+)$  captures changes in the component of the death distribution reflecting the normal life duration, it does not fully capture changes in premature deaths, the decline of which is the most important force compressing the distribution of adult deaths in earlier stages of the mortality transition. Therefore,  $S_{10}$  seems to be a preferable measure for examining mortality compression over the course of the mortality transition. In terms of using a measure of variability of age at death to compare differences in levels of adult mortality inequality across subgroups,  $S_{10}$  would also seem to be a better measure than  $SD(M+)$  since differences in premature mortality could potentially be an important contributing factor to mortality differentials.

Zhang and Vaupel have documented that  $S_{10}$  is highly correlated with other measures that have been used to study trends in mortality compression and life span disparity such as the inter-quartile range ( $IQR$ ), the Gini coefficient, and  $e^\dagger$  (Zhang and Vaupel, 2009, p. 726). Even though different measures of variability of age at death have been shown to be highly correlated, they are not necessarily completely interchangeable. The measure  $S_{10}$  represents a middle ground between measures which are highly influenced by trends in infant mortality because they are based on the entire death distribution (e.g.  $IQR$ ,  $e^\dagger$ , and  $SDM$ ) and measures that do not take into account premature mortality because they only measure dispersion above the modal age at death (e.g.  $SD(M+)$ ).

## 4.3 Methods

I provide more details on my particular methodological strategies in the substantive chapters of the dissertation. Here, I provide a broad overview of two statistical techniques on which I rely: decomposition analysis and perturbation analysis.

### 4.3.1 Decomposition Analysis

In the dissertation, I am interested in decomposing both trends in variability of age at death (as indicated by  $S_{10}$ ) over time and differences in variability of age at death among groups. The decomposition results quantify either the contribution of changes in age-specific mortality rates over time to change in  $S_{10}$  over time or the contribution of differences in age-specific mortality rates between groups to the difference in  $S_{10}$  between groups. I carry out these decompositions using the continuous-change method of decomposition developed by Horiuchi, Wilmoth, and Pletcher (Horiuchi et al., 2008). Throughout the dissertation, I refer to this decomposition method as the HWP method. This is a general method of decomposition that relies upon the assumptions that the covariates whose effects are being measured (in this case underlying mortality rates,  $m_x$ ) change gradually over time and that covariates change proportionally to one another over time. If these two assumptions are

met, the decomposition method allows one to estimate the contribution,  $c_i$ , of changes in a particular covariate to changes in the measure of interest. I am interested in determining the contribution of changes in  $m_x$  to the total change in  $S_{10}$ .

As a first step to carrying out this decomposition, it is necessary to express  $S_{10}$  in terms of  $m_x$ .

$$\begin{aligned}(S_{10})^2 &= \frac{\sum_{x=10}^T (x + a_x - M_{10})^2 \times d_x}{\sum_{x=10}^T d_x} \\(S_{10})^2 &= \frac{\sum_{x=10}^T (x + a_x - M_{10})^2 \times l_x m_x}{\sum_{x=10}^T l_x m_x} \\(S_{10})^2 &= \frac{\sum_{x=10}^T (x + a_x - \sum_{x=10}^T x(\exp(-\sum_0^x m_x))m_x)^2 \times (\exp(-\sum_0^x m_x))m_x}{\sum_{x=10}^T (\exp(-\sum_0^x m_x))m_x}\end{aligned}$$

In order to attribute the changes in  $S_{10}$  over time to the changes in  $m_x$  over the same time period, it is important to recognize that  $S_{10}$  and  $m_x$  are functions of time:

$$(S_{10}(t))^2 = \frac{\sum_{x=10}^T \left( x + a_x - \sum_{x=10}^T x(\exp(-\sum_0^x m_x(t)))m_x(t) \right)^2 \times (\exp(-\sum_0^x m_x(t)))m_x(t)}{\sum_{x=10}^T (\exp(-\sum_0^x m_x(t)))m_x(t)}$$

To use the HWP method, write  $S_{10}$  as a function of  $m_1 \dots m_T$ :

$$S_{10} = f(m_1, m_2, \dots, m_T)$$

The general decomposition method holds that for  $y = f(x_1, x_2, \dots, x_n)$ , the change in  $y$  from time 1 to time 2 can be expressed as:

$$y(2) - y(1) = \sum_{i=1}^n c_i, \text{ where } c_i = \int_{X_{i1}}^{X_{i2}} \frac{\partial y}{\partial x_i} \frac{dx_i}{dt} dt$$

The equation above indicates that the total change in the variable of interest,  $y$ , from time 1 to time 2 can be found by summing up the contributions,  $c_i$  of each covariate,  $x_i$ .  $X_{i,1}$  and  $X_{i,2}$  represent the value of  $x_i$  at the beginning ( $t = 1$ ) and end ( $t = 2$ ) of the interval respectively.

Computationally, estimating  $c_i$  involves looking at how changes in a particular value of  $x_i$  over a narrow interval change  $y$  while holding other values of  $x_i$  constant at values observed at the midpoint of the narrow interval and then summing the effects observed across all of the intervals between time 1 and 2.

For my analysis, I want to take partial derivatives of  $S_{10}$  with respect to  $m_i$  to find the contribution of changes in the age-specific mortality rate,  $m_i$ , to changes in  $S_{10}$ :

$$c_i = \int_{m_{i1}}^{m_{i2}} \frac{\partial S_{10}}{\partial m_i} \frac{dm_i}{dt} dt$$

For multiple cause-of-death decompositions, I rewrite equation  $S_{10}$  incorporating age and cause-specific mortality  $m_{x,i}$ . Consider causes  $i = 1, \dots, j$ :

$$(S_{10}(t))^2 = \frac{\sum_{x=10}^T \left( x + a_x - \sum_{x=10}^T x(\exp(-\sum_{a=0}^x \sum_{i=1}^j m_{a,i}(t))) \sum_{i=1}^j m_{x,i}(t) \right)^2 \times (\exp(-\sum_{a=0}^x \sum_{i=1}^j m_{a,i}(t))) \sum_{i=1}^j m_{x,i}(t)}{\sum_{x=10}^T (\exp(-\sum_{a=0}^x \sum_{i=1}^j m_{a,i}(t))) \sum_{i=1}^j m_{x,i}(t)}$$

Mathematically, the change in  $S_{10}$  observed between time 1 and 2 can be decomposed as follows:

$$S_{10}(2) - S_{10}(1) = \sum_{a=1}^T \sum_{z=1}^j c_{a,z}, \text{ where } c_{a,z} = \int_{m_{x,i,1}}^{m_{x,i,2}} \frac{\partial S_{10}}{\partial m_{x,i}} \frac{dm_{x,i}}{dt} dt$$

Computationally, the decomposition procedure involves dividing the range of each  $m_{x,i}$  observed between time 1 and 2 by  $N$ :

$$\Delta m_{x,i} = (m_{x,i,2} - m_{x,i,1})/N$$

Then, create  $N$  matrices  $\mathbf{A}_{k\bullet}$  whose elements are the values of  $m_{x,i}$  observed during the  $k$ th interval between time 1 and 2. The dimensions of an each matrix,  $\mathbf{A}_{k\bullet}$ , are determined by the number of age groups and the number of causes of death used in the analysis. The subscript  $k$  denotes the time interval and ranges from 1 to  $N$ . The  $\bullet$  index indicates that the values contained in the matrix were observed at the midpoint of interval  $k$ . In  $\mathbf{A}_{k\bullet}$ , the elements of the matrix,  $m_{x,i}$  are fixed at the values of these measures observed at the midpoint of time interval  $k$  (e.g. the  $x$ 'th row and  $i$ 'th column of  $(A_{5\bullet})_{x,i} = m_{x,i,1} + 4.5 * \Delta m_{x,i}$ ).

In order to find the contribution,  $c_{x,i}$ , of changes of a particular  $m_{x,i}$  contained in  $\mathbf{A}_{k\bullet}$ , the midpoint value of one particular  $m_{x,i}$  in each interval  $k$  is varied to the value observed at the beginning of the  $k$ th interval and then to the value observed at the end of the  $k$ th interval (these matrices can be denoted  $\mathbf{A}_{xi,k+}$  and  $\mathbf{A}_{xi,k-}$ ). These matrices are indexed with  $xi$  because only  $m_{x,i}$  is varied-all other elements of the matrices  $\mathbf{A}_{xi,k+}$  and  $\mathbf{A}_{xi,k-}$  are equivalent to the elements of  $\mathbf{A}_{k\bullet}$ .  $S_{10}$  is calculated for both variants and a difference is taken. This procedure is repeated for each of the  $N$  intervals and  $\hat{c}_{x,i}$ , is estimated as the sum of these  $N$  differences in  $S_{10}$ :

$$\hat{c}_{x,i} = \sum_{k=1}^N f(\mathbf{A}_{xi,k+}) - f(\mathbf{A}_{xi,k-})$$

### 4.3.2 Perturbation Analysis

I am able to quantify the potential effect of changes in age-specific mortality rates on measures of variability of age at death through perturbation analysis of a Markov chain model. These perturbation analysis methods, currently being developed by Hal Caswell, are extensions of the methods found in (Caswell, 2006; Caswell, 2009, in press).<sup>3</sup> The fundamental matrix for the Markov chain model can be derived from a square matrix whose sub-diagonal represents the probability of transitioning from one age group to the next at any particular age (all other elements are zero). The fundamental matrix can then be used to calculate a vector representing the expected longevity at each age (i.e. life expectancy) and a vector representing the variance of longevity at each age. By taking the derivative of the longevity vector and the variance of longevity vector with respect to the vector of underlying age-specific mortality rates, one can quantify the sensitivity of longevity and variance in longevity to changes in age-specific mortality. A more detailed description of how I use this method to calculate the sensitivities of  $S_{10}$  and  $e^\dagger$  is provided below:

#### Method for calculating the sensitivity of $S_{10}$

Age-specific mortality rates are the parameter of interest,  $\underline{\theta}$ :

$$\underline{\theta} = \begin{pmatrix} m_1 \\ m_2 \\ \dots \\ m_p \end{pmatrix}$$

The steps below outline how to carry out the perturbation analysis. Note the following:

- $\mathbf{X} \circ \mathbf{Y}$  calls for the component wise (element by element) product of these matrices.
  - I denote by  $\otimes$  the Kronecker product.
  - The operation  $\text{vec}(\mathbf{X})$  stacks the columns of the  $\mathbf{X}$  matrix in a single column.
  - The operation  $\text{diag}(X)$  indicates that a diagonal matrix is created with the elements of the vector X.
1. Start with a matrix  $\mathbf{U}$ , whose sub-diagonal elements represent the probability of surviving from one age group to the next. For an age-classified model with three ages, the elements of the matrix  $\mathbf{U}$  can be calculated using age-specific mortality rates as follows:

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<sup>3</sup>The perturbation analysis methods were presented in Hal Caswell's "Perturbation Analysis of Longevity" course offered March 30-April 17, 2009 at the IMPRSD in Rostock, Germany.

$$\mathbf{U} = \begin{pmatrix} 0 & 0 & 0 \\ P_1 & 0 & 0 \\ 0 & P_2 & 0 \end{pmatrix} = \begin{pmatrix} 0 & 0 & 0 \\ e^{-m_1} & 0 & 0 \\ 0 & e^{-m_2} & 0 \end{pmatrix}$$

Thus, the derivative of  $\mathbf{U}$  with respect to the age-specific mortality rate for the first age group is given by:

$$\frac{d\mathbf{U}}{d\underline{\theta}_1} = \begin{pmatrix} 0 & 0 & 0 \\ -P_1 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} = \begin{pmatrix} 0 & 0 & 0 \\ -e^{-m_1} & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

2. Calculate fundamental matrix of Markov chain process,  $\mathbf{N}$ .  $\mathbf{N} = (\mathbf{I} - \mathbf{U})^{-1}$
3. Calculate life expectancy at each age,

$$\mathbf{E}(\eta)^T = e^T \mathbf{N}$$

Where  $e$  is a column vector of 1's with number of rows equal to the dimensions of  $\mathbf{N}$ .

4. The variance of longevity for each starting state  $j$  is given by

$$\mathbf{V}(\eta)^T = e^T \mathbf{N}(2\mathbf{N} - \mathbf{I}) - \mathbf{E}(\eta) \circ \mathbf{E}(\eta)$$

5. To calculate the sensitivity of variance in longevity to our parameter of interest,  $\underline{\theta}$ , take the derivative of  $\mathbf{V}(\eta)^T$ . The rows of the matrix  $\frac{d\mathbf{V}(\eta)^T}{d\underline{\theta}^T}$  will indicate the sensitivity of variance of longevity at a particular age to changes in mortality in the age group represented by the column. To find the sensitivity of  $S_{10}$  to changes in age-specific mortality rates, one examines the 11th row of the matrix  $\frac{d\mathbf{S}(\eta)^T}{d\underline{\theta}^T}$ .

$$\begin{aligned} \mathbf{V}(\eta)^T &= \mathbf{e}^T \mathbf{N}(2\mathbf{N} - \mathbf{I}) - \mathbf{E}(\eta)^T \circ \mathbf{E}(\eta)^T \\ \mathbf{V}(\eta)^T &= \mathbf{e}^T 2\mathbf{N}^2 - \mathbf{e}^T \mathbf{N} - \mathbf{E}(\eta)^T \circ \mathbf{E}(\eta)^T \\ \frac{d\mathbf{V}(\eta)^T}{d\underline{\theta}^T} &= 2\mathbf{e}^T (d\mathbf{N})\mathbf{N} + 2\mathbf{e}^T \mathbf{N}(d\mathbf{N}) - \mathbf{e}^T d\mathbf{N} - 2d\mathbf{E}(\eta)^T \circ \mathbf{E}(\eta)^T \end{aligned}$$

apply **vec**

$$\begin{aligned} \frac{d\mathbf{V}(\eta)^T}{d\underline{\theta}^T} &= (2\mathbf{N}^T \otimes \mathbf{e}^T) d\text{vec}\mathbf{N} + (\mathbf{I} \otimes 2\mathbf{e}^T \mathbf{N}) d\text{vec}\mathbf{N} - (I \otimes \mathbf{e}^T) d\text{vec}\mathbf{N} - 2\text{diag}(\mathbf{E}(\eta)) d\mathbf{E}(\eta) \\ \frac{d\mathbf{V}(\eta)^T}{d\underline{\theta}^T} &= (2\mathbf{N}^T \otimes \mathbf{e}^T + \mathbf{I} \otimes 2\mathbf{e}^T \mathbf{N} - I \otimes \mathbf{e}^T) d\text{vec}\mathbf{N} - 2\text{diag}(\mathbf{E}(\eta)) d\mathbf{E}(\eta) \\ \frac{d\mathbf{V}(\eta)^T}{d\underline{\theta}^T} &= (2\mathbf{N}^T \otimes \mathbf{e}^T + \mathbf{I} \otimes 2\mathbf{e}^T \mathbf{N} - I \otimes \mathbf{e}^T)(\mathbf{N}^T \otimes \mathbf{N}) \frac{d\text{vec}\mathbf{U}}{d\underline{\theta}^T} - 2\text{diag}(\mathbf{E}(\eta))(\mathbf{I} \otimes \mathbf{e}^T)(\mathbf{N}^T \otimes \mathbf{N}) \frac{d\text{vec}\mathbf{U}}{d\underline{\theta}^T} \end{aligned}$$

Derivative of standard deviation of longevity  $d\mathbf{S}(\eta)^T$  calculated as follows

$$\frac{d\mathbf{S}(\eta)^T}{d\underline{\theta}^T} = \frac{1}{2} \left( \mathbf{V}(\eta)^T \right)^{-\frac{1}{2}} d\mathbf{V}(\eta)^T$$

### Method for calculating the sensitivity of $e^\dagger$

The equation for  $e^\dagger$  is

$$e^\dagger = \int_0^\infty f(x)e(x)dx$$

Define

- $\psi_x = P(\text{survive from age } x \text{ to } x+1 \text{ conditional on starting in age } x)$
- The 1 appears in the  $j$ th row of  $\mathbf{e}_j$ .

$$\mathbf{e}_j = \begin{pmatrix} 0 \\ 0 \\ .. \\ 1 \\ 0 \end{pmatrix}$$

- Let  $\mathbf{M}$  be a diagonal matrix whose entries correspond to the probability of transitioning from life to death at age  $x$ . Then, the diagonal entries in  $\mathbf{M}$  will be the  $1-\psi_x$ .
- Let  $\mathbf{B} = \mathbf{MN}$  where  $\mathbf{N}$  is the fundamental matrix in the Markov chain model defined in Appendix A.

The distribution of age at death starting in age  $j$  is given in the  $j$ th column of  $\mathbf{B}$ .

$$\begin{aligned} f^{(j)} &= \mathbf{Be}_j \\ f^{(j)} &= \mathbf{MNe}_j \end{aligned}$$

The sensitivity of  $e^\dagger$ , denoted below as  $\eta^\dagger$ , can be calculated for a particular starting age  $j$  (usually zero) as follows:

$$\begin{aligned}\eta^\dagger &= \mathbf{E}(\eta)^T (f^{(j)})^T \\ \eta^\dagger &= \mathbf{e}^T \mathbf{N} \mathbf{M} \mathbf{N} \mathbf{e}_j \\ d\eta^\dagger &= \mathbf{e}^T (d\mathbf{N}) \mathbf{M} \mathbf{N} \mathbf{e}_j + \mathbf{e}^T \mathbf{N} (d\mathbf{M}) \mathbf{N} \mathbf{e}_j + \mathbf{e}^T \mathbf{N} \mathbf{M} (d\mathbf{N}) \mathbf{e}_j\end{aligned}$$

apply **vec**

$$\begin{aligned}d\eta^\dagger &= (\mathbf{e}_j^T \mathbf{N}^T \mathbf{M}^T \otimes \mathbf{e}^T) d\text{vec} \mathbf{N} \\ &\quad + (\mathbf{e}_j^T \mathbf{N}^T \otimes \mathbf{e}^T \mathbf{N}) d\text{vec} \mathbf{M} \\ &\quad + (\mathbf{e}_j^T \otimes \mathbf{e}^T \mathbf{N} \mathbf{M}) d\text{vec} \mathbf{N} \\ d\eta^\dagger &= (\mathbf{e}_j^T \mathbf{N}^T \mathbf{M}^T \mathbf{N}^T \otimes \mathbf{e}^T \mathbf{N}) d\text{vec} \mathbf{U} \\ &\quad + (\mathbf{e}_j^T \mathbf{N}^T \otimes \mathbf{e}^T \mathbf{N} \mathbf{M} \mathbf{N}) d\text{vec} \mathbf{U} \\ &\quad + (\mathbf{e}_j \mathbf{N}^T \otimes \mathbf{e}^T \mathbf{N}) d\text{vec} \mathbf{M}\end{aligned}$$

## 4.4 Conclusion

The measures and methods outlined here will be utilized throughout the course of the dissertation in order to obtain a better understanding of shifting mortality and differentials in life span disparity. The broad scope and high quality of the data available in the Human Mortality Database makes what once would have been a difficult undertaking in terms of data collection efforts into a research project that can be pursued with relative ease.

Table 4.1: Geographical areas included in the Human Mortality Database and corresponding periods of data coverage.

Country or Area	Years
Australia	1921-2004
Austria	1947-2005
Belarus	1959-2007
Belgium	1841-1913, 1919-2006
Bulgaria	1947-2005
Canada	1921-2005
Chile	1992-2005
Czech Republic	1950-2006
Denmark	1835-2007
England and Wales	1841-2006
Estonia	1959-2007
Finland	1878-2007
France	1816-2006
Germany	1956-2006
Hungary	1950-2005
Iceland	1838-2007
Ireland	1950-2006
Italy	1872-2005
Japan	1947-2006
Latvia	1959-2007
Lithuania	1959-2007
Luxembourg	1960-2006
Netherlands	1850-2006
New Zealand	1876-2003
Norway	1846-2006
Poland	1958-2006
Portugal	1940-2007
Russia	1959-2006
Scotland	1855-2006
Slovakia	1950-2006
Slovenia	1983-2006
Spain	1908-2006
Sweden	1751-2007
Switzerland	1876-2007
Taiwan	1970-2007
Ukraine	1959-2006
United States	1933-2005

Table 4.2: Geographical areas included in the World Health Organization's collection of 1,802 life tables, which do not overlap with HMD, and corresponding periods of data coverage.

Country or Area	Years
Argentina	1966-1970, 1977-1979, 1982-1997
Australia	1911
Chile	1909, 1920, 1930, 1940, 1950, 1955-1982, 1984-1991
Colombia	1960, 1964
Costa Rica	1956-1983, 1985-1998
Croatia	1982-1998
Cuba	1970-1998
Czechoslovakia	1934
El Salvador	1950, 1971
Georgia	1981-1992, 1994-1996
Greece	1928, 1956-1998
Guatemala	1961, 1964
Honduras	1961, 1974
India	1971
Iran (Islamic Republic of)	1974
Israel	1975-1998
Matlab (Bangladesh)	1975
Mauritius	1990-1998
Mexico	1958-1959, 1969-1973, 1981-1983, 1985-1998
Moldova	1981-1998
Panama	1960
Peru	1970
Philippines	1964, 1970
Portugal	1920, 1930
Republic of Korea	1973
Romania	1963, 1969-1978, 1980-1998
Singapore	1955-1998
Slovenia	1982
South Africa (colored pop.)	1941, 1951, 1960
Sri Lanka	1946, 1953
Taiwan, Province of China	1920, 1930, 1936
Thailand	1970
The former Yugoslav Republic of Macedonia	1982-1997
Trinidad and Tobago	1990-1995, 1997
Tunisia	1968
United States of America	1900-1916, 1920-1932
Yugoslavia	1982-1997

# Chapter 5

## Age pattern of mortality change and trends in variability in age at death

Shifting mortality was conceptualized as a shift in mortality rates across age over time; however, trends in variability of age at death, which are indicative of changes in the shape of the death distribution over time, are used to evaluate whether or not a country is exhibiting signs of shifting mortality. In this chapter, I reexamine the relationship between mortality change and trends in variability age of death in order to evaluate whether shifting mortality is an inevitable outcome of the mortality transition. Through simulation exercises and perturbation analysis, I arrive at a better understanding of how initial mortality conditions and the age-pattern of mortality change interact to produce mortality compression, expansion, or shifting. The results presented in this chapter, based on hypothetical changes in age-specific mortality rates, are complimented by the results of a decomposition analysis of actual trends in variability of age at death, which are presented in the next chapter. In this chapter, I demonstrate that proportional mortality change that is fixed across ages does not necessarily lead to a parallel shift in the death distribution and that certain initial mortality conditions are particularly primed for compression.

A full overview of the literature related to the topic of mortality compression, expansion, and shifting is presented in Chapter 2. Here, I offer a short review of papers by authors that have offered possible explanations for shifting mortality in order to set the stage for the analysis that I pursue in this chapter. To date, most studies examining the reasons for the transition from mortality compression to shifting mortality focus on the age pattern of mortality change. For instance, an explanation for shifting mortality related to divergence in the age pattern of all-cause mortality change appears in Wilmoth and Horiuchi's 1999 piece on trends in variability of age at death (Wilmoth and Horiuchi, 1999). Using historical mortality evidence from Sweden, the authors document that life expectancy rose steadily throughout the period of observation (1751-1995). In contrast, the *IQR*, a measure of variability of age at death corresponding to age span between the 25th and 75th percentiles of the cumulative death distribution, remained relatively stable from 1751-1875, declined

rapidly during the period 1876-1955, and then remained relatively stable at its new lower level from 1955 onwards. The authors observe that during the period of rapid decline in *IQR*, the average annual rate of proportional mortality decline varied greatly across age with much more rapid progress at younger ages. In contrast, during periods in which the *IQR* remained relatively stable, the average annual rate of mortality decline was similar across ages.<sup>1</sup>

Similar results have been found using mortality models. Focusing on mortality between ages 70 and 90, Thatcher et al. show that in cases where age-specific mortality can be described using a logistic model ( $m_x = \frac{\alpha e^{\beta x}}{1+\alpha e^{\beta x}}$ ) variability of age at death is only responsive to changes in the slope parameter ( $\beta$ ) (Thatcher et al., 2008). As long as mortality change is non-divergent across age (i.e. proportional change in mortality over time at rates unvarying across age), the slope parameter remains constant, and variability of age at death does not change. Bongaarts, observing a relatively fixed slope parameter for the age-pattern of senescent mortality (as described by a logistic model) in more developed countries over the latter half of the 20th century, builds a mortality projection model based upon the assumption that these country-specific slope parameters will continue to remain fixed in the future and senescent mortality will just shift to higher ages (Bongaarts, 2005).

Using the Siler model of mortality change, which parameterizes the entire age pattern of mortality, and simulating morality change over a long period, starting with conditions of high infant and child mortality, Canudas-Romo finds that mortality compression eventually gives way to shifting mortality assuming a fixed pattern of mortality change that is unvarying across adult ages—the range of ages at death which eventually come to dominate the measure of variability of age at death (Canudas-Romo, 2008). The results of Canudas-Romo's simulation using the Siler mortality change model also indicate that the pace of mortality compression slows after infant mortality has reached a relatively low level. This suggests that the potential for compression depends on current mortality conditions.

Wilmoth and Horiuchi's suggestion that divergent age patterns of mortality change (with younger age groups experiencing faster progress) lead to a decline in the variability of age at death is especially appealing if it also true that if the age pattern of change in mortality is similar across ages (especially the ages where deaths are concentrated), then variability of age at death will remain constant. The results of Thatcher et al. seem to suggest that similarity in the age-pattern of mortality change is a necessary condition for observing shifting mortality. Is it a sufficient condition? This line of thought leads to a simple and easily tested hypothesis: if mortality change is proportional across age (the pattern of mortality change is similar across age on a relative scale), then there will be no change in the variability of age at death. Instead of becoming more or less variable, the death distribution will just parallel shift in either direction. If this relationship holds true, similarities in the relative pace of change in mortality across age might be the underlying cause of the current shifting

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<sup>1</sup>In the most recent period, the pattern is not similar across all ages but it is in the ages where the majority of deaths are concentrated.

mortality scenario.

In this chapter, I seek to test this simple hypothesis: does proportional change in mortality that is fixed across age always lead to a shift in the death distribution. First, I examine whether current mortality conditions conform to the assumptions of simple models of mortality change that produce shifting mortality. I then go on to conduct simple simulation exercises that test how measures of variability of age at death respond to changes in age-specific mortality rates under a variety of initial mortality conditions. Through perturbation analysis, I can directly quantify the sensitivity of measures of variability of age at death to changes in age-specific mortality without relying on experimental conditions. This analysis leads to a rejection of the simple hypothesis and a deeper understanding of the complex relationship between measures of variability of age at death, initial mortality conditions, and the age pattern of mortality decline.

## 5.1 Conditions for a simple model of shifting mortality

To begin, I want to consider the conditions under which prior literature has shown that a decline in mortality corresponds exactly to a shift in the mortality curve across age. For example, a proportional decline in age-specific mortality rates fixed across age corresponds to a shift of the mortality curve over age when the shape of the age-specific mortality curve is linear on a log scale. The Gompertz curve, which fits this log-linear criteria, is often used to describe the shape of the mortality curve at adult ages. This model can be expressed as follows:

$$\mu(x) = \alpha e^{\beta x}$$

A Gompertz mortality curve with  $\alpha = 5 \times 10^{-5}$  and  $\beta = .10$  is shown in Figure 5.1(a). It is clear from this figure that a proportional decline in age-specific log mortality rates that is fixed across age is equivalent to a shift of the mortality curve over some years of age. When mortality decline can be expressed as a shift of the mortality curve over age, the death distribution retains its shape yet shifts either forward or backward in age. This is shown in Figure 5.1(b).

Currently, in a number of more developed countries, life table death distributions based on period mortality data are exhibiting signs of shifting akin to what is observed in this simple model of mortality change; however, current mortality conditions and changes in mortality over time do not necessarily conform to this simple model. If the mortality rates could just shift across age, that is, if the age scale was not fixed, a shift in mortality rates across age would necessarily lead to a shift in the death distribution. The age scale is fixed, however, and mortality rates at the youngest ages do not conform to a simple log-linear mortality models like the Gompertz or logistic models. As a result, a downward shift in mortality does not necessarily translate into a shift of the mortality curve over age as seen in Figure 5.2(a). Additionally, proportional mortality change over time is not necessarily fixed

across age. As shown in Figure 5.2(b), the age pattern of mortality change for Sweden has been divergent across age even in the period of time when relative stability in variability in age at death has been observed (1960 onwards). In this figure, the average annual rate of mortality decline is calculated using the formula  $-\log\left(\frac{m_x(t+n)}{m_x(t)}\right)/n$ .

Discrepancies between the simple model of shifting mortality and empirical reality include divergence in the age pattern of mortality change and initial mortality conditions that are not well described by the Gompertz or logistic mortality models. These discrepancies suggest that the simple hypothesis that proportional mortality change that is fixed across age always leads to a shift in the death distribution might not hold. Proportional changes in mortality that are fixed across age will not necessarily translate into a shift in the death distribution if the initial mortality conditions are not linear or semi-linear on a log scale. In the next section, I explore further the dynamic relationship between initial mortality conditions and measures of variability of age at death given proportional change in mortality that is fixed across age and time.

## 5.2 Do proportional changes in mortality that are fixed across age result in shifting mortality?

I have set up a relatively simple simulation experiment to test the hypothesis that a proportional change in mortality that is fixed across age will have no impact on variability of age at death. Starting with an initial set of age-specific mortality rates ( $m_x$ ), I apply a proportional change in mortality that is fixed over age and time over some projection interval, and then I document trends in variability of age at death over the projection horizon. The simple hypothesis would suggest that variability of age at death should remain the same over the projection interval regardless of the initial set of age-specific mortality rates. The simulation is carried out using five different initial vectors of age-specific mortality rates,  $m_x$ , from the Human Mortality Database (HMD, 2009): the single-year age-specific mortality rates of Swedish males in 1800-04, 1850-54, 1900-04, 1950-54, and 2000-04<sup>2</sup>. I project the change in  $m_x$  over 300 years assuming an average annual proportional change  $\phi = .005$  across all ages according to the following equation<sup>3</sup>

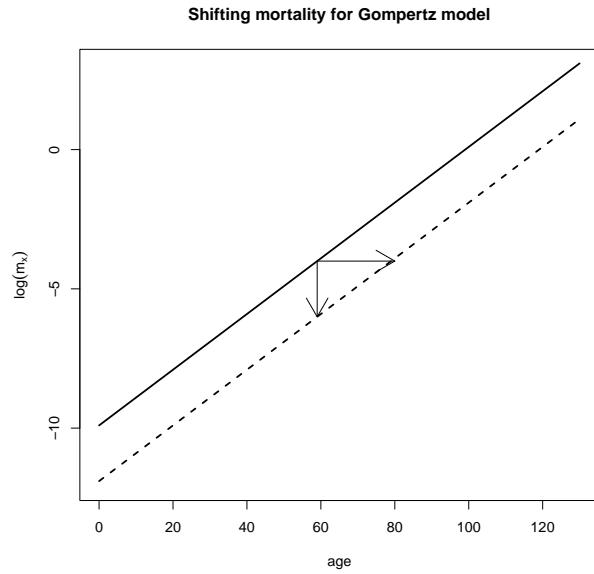
$$m_x(t) = m_x(0) * (1 + \phi)^t \quad (5.1)$$

Variability of age at death is measured using both  $S_{10}$ , the standard deviation of ages at death past age 10, and  $e^\dagger$ , the years of life lost in the life table due to premature death. The

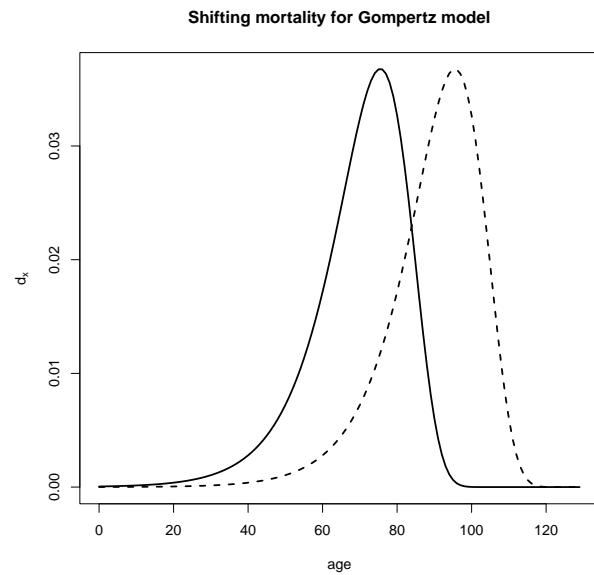
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<sup>2</sup>I was also planning on using an initial  $m_x$  corresponding to the mortality rates of Swedish males in 1751-54, but the mortality rates at older ages for this life table are unusually low ( $m_{85+}(1950) > m_{85+}(1751)$ ) which resulted in deaths heaping up too rapidly in the open ended age interval

<sup>3</sup>The following equation would have been more consistent with how Wilmoth and Horiuchi measured the average annual rate of proportional mortality decline:  $m_x(t) = m_x(0)e^{\phi t}$ .

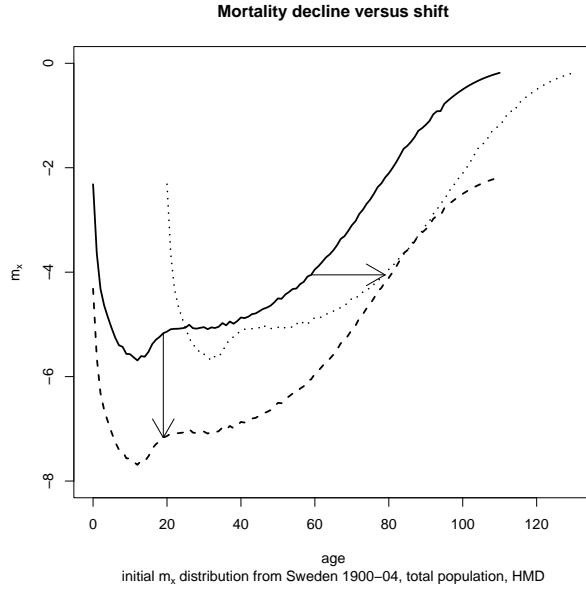


- (a) Changes in age-specific mortality rates under the Gompertz model with proportional change in mortality across age



- (b) Changes in the death distribution under the Gompertz model with proportional change in mortality across age

Figure 5.1: Shifting mortality under the Gompertz mortality change model.



(a) A decline in mortality does not necessarily translate into a shift in mortality



(b) The age pattern of mortality decline is divergent across age

Figure 5.2: Violations of the Gompertz mortality change model found in the mortality experience of actual human populations. The average annual rate of mortality decline is calculated using the formula  $-\log\left(\frac{m_x(t+n)}{m_x(t)}\right)/n$ . Data source: Human Mortality Database (HMD, 2009).

$e^\dagger$  measure takes into account changes in infant mortality while the  $S_{10}$  measure is reflective of the mortality experience during adult ages. Actual trends in  $S_{10}$ , entropy ( $\bar{H} = \frac{e^\dagger}{e_0}$ ), and  $e^\dagger$  for both deaths above age 10 and deaths at all ages for Swedish males and females are shown in Figure 5.3.

One difficulty with carrying out this experiment is that the oldest observable age is fixed at 110. As deaths start to accumulate in the open ended age interval, this can bias the results. With an annual proportional change of  $\phi = .005$ , there did not seem to be too many deaths accumulating in the last age group after projecting forward 300 years (for  $\phi = .01$  there was very obvious bias in the measures of variability of age at death). Still, the trends in  $e^\dagger$  and  $S_{10}$  near the end of the projection interval should be interpreted with caution.

Graphical results of this simple experiment are depicted in Figure 5.4. These results indicate that the relationship between the age pattern on mortality change and variability of age at death is not as straightforward as the simple hypothesis suggests. When variability of age at death is measured as  $S_{10}$ , a proportional change in mortality results in relative stability in  $S_{10}$  over the projection interval for four of our five initial  $m_x$  distributions. For the initial set of age-specific mortality rates,  $m_x$ , corresponding to the mortality experience of Swedish males in 1900-04, a fixed proportional change in mortality across age and time results in a decline in  $S_{10}$  over the projection horizon. The divergence of the trend for the initial  $m_x$  corresponding to 1900-04 from the other initial states is quite striking, and it suggests that trends in the variability of age at death do not have a simple relationship with the age pattern of mortality decline. Rather, there is an interaction between the initial death distribution and the age pattern of mortality change. For some initial mortality conditions, like the conditions Swedish males experienced in 1900-04, measures of variability of age at death are more sensitive to changes in mortality-whether these changes are fixed across ages or not.

If instead of using  $S_{10}$  as the measure of variability of age at death I use  $e^\dagger$ , a different picture of mortality change emerges. In this case, the trends in variability of age at death still look relatively stable for the initial  $m_x$ 's corresponding to 1950-54 and 2000-04; however, the initial  $m_x$ 's corresponding to 1800-04 and 1850-54 show a declining trend in  $e^\dagger$  similar to the trend observed with initial  $m_x$ 's corresponding to 1900-04 except less steep. The difference between trends observed for  $e^\dagger$  and  $S_{10}$  corresponding to initial  $m_x$  vectors from 1800-04 and 1850-54 is the result of the responsiveness of  $e^\dagger$  to changes in infant mortality. As will be demonstrated later,  $e^\dagger$  is particularly sensitive to changes in infant mortality especially when the initial states indicate high levels of infant mortality.

The results of this simple experiment of applying a fixed proportional change in mortality over age and time to several initial sets of age-specific mortality rates overwhelmingly demonstrate that the initial hypothesis was not true. An age pattern of proportional mortality change that is similar across ages does not in and of itself lead to stability in the measure of variability of age at death. Proportional change in mortality that is fixed across age while perhaps a necessary condition is certainly not a sufficient condition for shifting mortality.

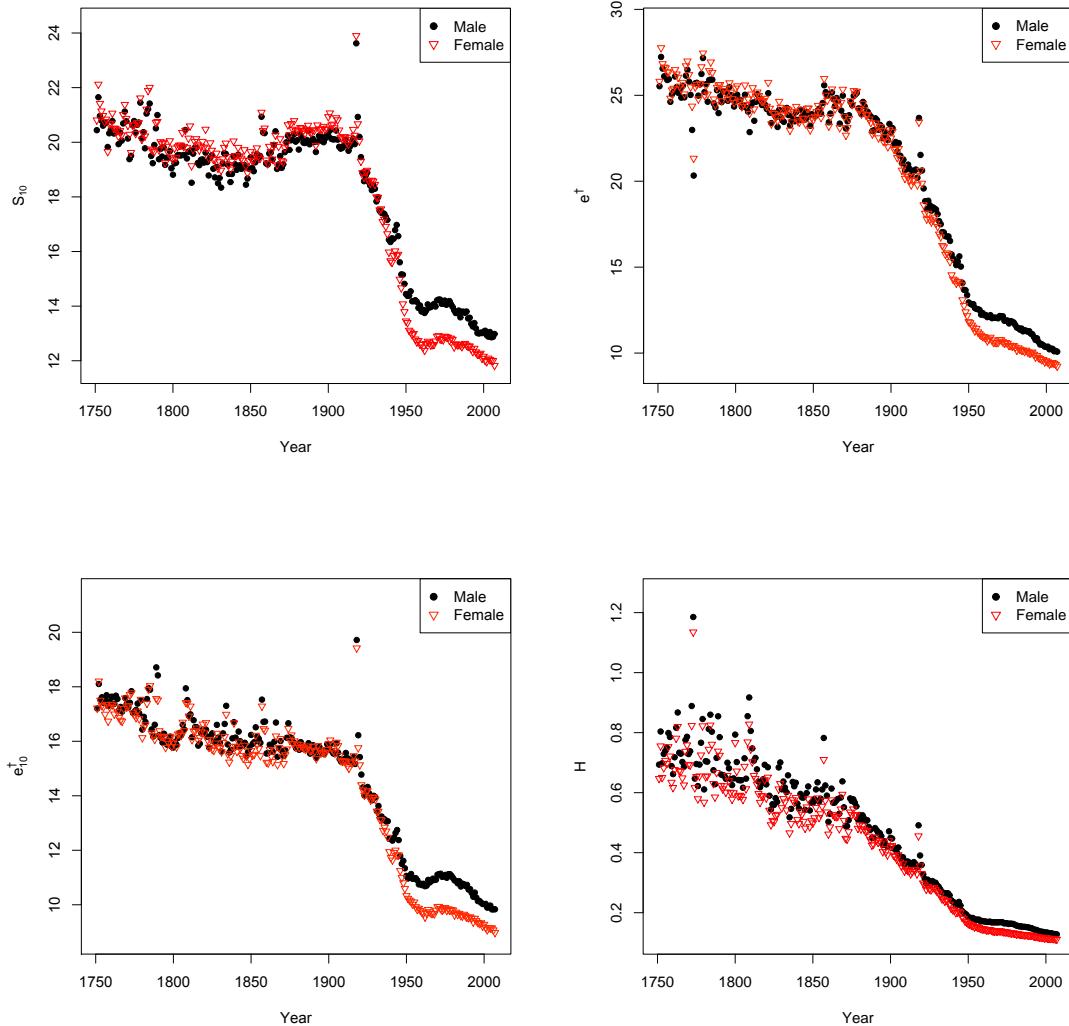


Figure 5.3: Trends observed in  $S_{10}$ ,  $\bar{H}$ ,  $e^\dagger$ , and  $e_{10}^\dagger$ , Sweden, 1751-2007. Data source: Human Mortality Database (HMD, 2009).

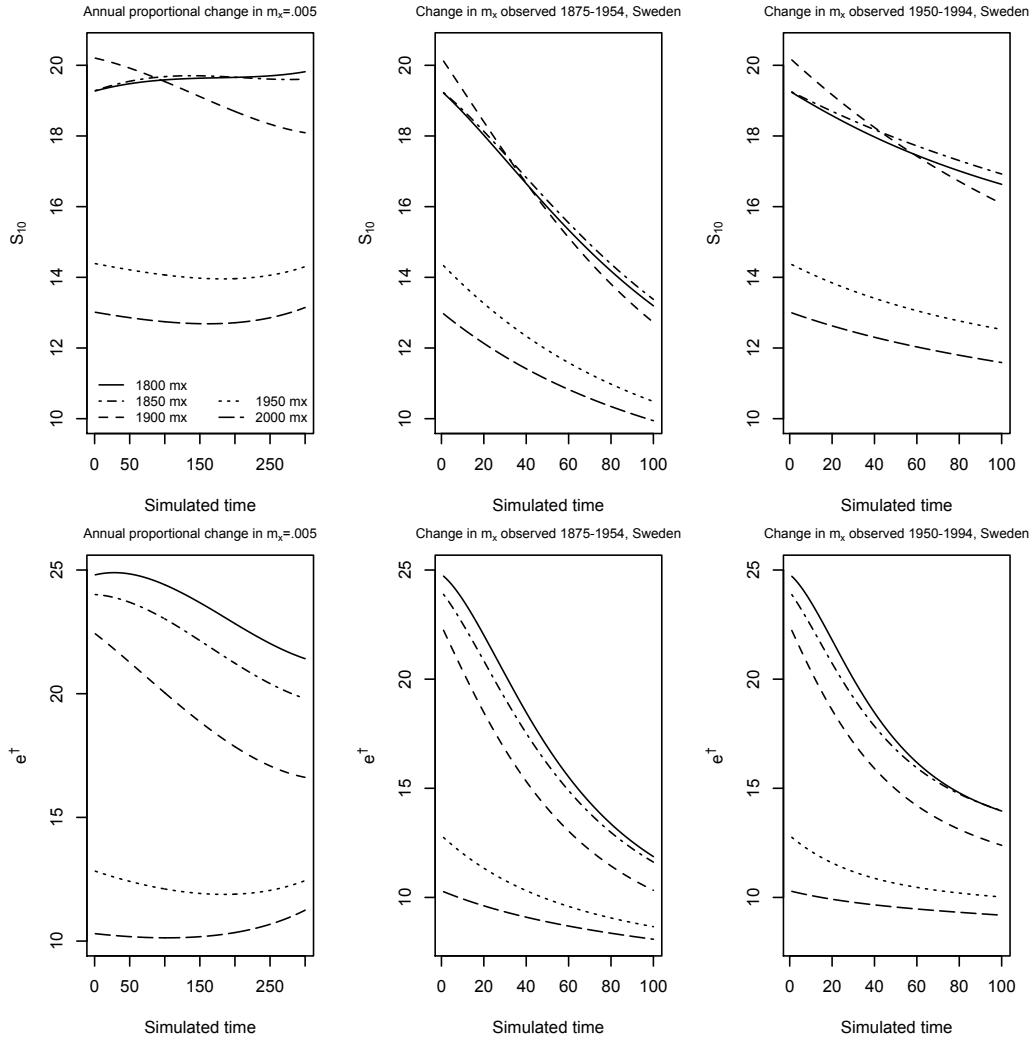


Figure 5.4: Trends observed in  $S_{10}$  and  $e^{\dagger}$  under various projection scenarios. Titles of individual plots indicate the pattern of age-specific mortality change observed over the projection horizon.

### 5.3 Further examination of the relationship between age pattern of mortality change and variability of age at death

In order to gain more insight into the relationship between the age pattern of mortality change and initial mortality conditions, I conducted a similar experiment but used patterns of mortality change that varied over age while remaining fixed over time. Two different patterns of age-specific mortality change were utilized: the age patterns of average annual proportional mortality change observed during the periods 1875-79 to 1950-54 and 1950-54 to 1990-94 among Swedish males. The data on mortality change, like the set of initial mortality vectors, also come from the Human Mortality Database (HMD, 2009). The underlying age patterns of average annual mortality change along with the smoothed age pattern utilized in the analysis are shown in Figure 5.5. The projections in this experiment only extend out 100 years because further extending the time horizon resulted in too many deaths being heaped into the open ended age group.

Wilmoth and Horiuchi observed in their paper that the age pattern of mortality change was much more divergent across ages during the period 1875-79 to 1950-54 when the IQR was declining rapidly in comparison to the period 1950-54 to 1990-94 when the age pattern is much flatter across the ages where the majority of deaths are concentrated (Wilmoth and Horiuchi, 1999). Therefore, I expect that applying the age pattern of mortality change observed in 1875-79 to 1950-54 to the initial  $m_x$  vectors will result in faster mortality compression (reduction in variability of age at death) than when the average annual proportional change observed from 1950-54 to 1990-94 is used to make the projections. I am especially interested in observing how rates of decline in  $S_{10}$  and  $e^\dagger$  differ depending on the initial  $m_x$  distribution.

The results of this analysis are shown in Figure 5.4. As anticipated, applying the average annual mortality change observed in the period 1875-79 to 1950-54 to the five initial  $m_x$  vectors resulted in faster mortality compression than using the mortality change observed 1950-54 to 1990-94. When variability of age at death is measured using  $S_{10}$ , the fastest rates of mortality compression using either pattern of mortality change are observed when the initial  $m_x$  corresponds to the  $m_x$  observed for Swedish males 1900-04. Again, it appears that this initial distribution is more “primed for compression” in comparison to the other initial sets of  $m_x$ . If  $e^\dagger$  is employed to measure variability instead of using  $S_{10}$ , the initial set of  $m_x$ ’s corresponding to 1800-04, 1850-54, and 1900-04 exhibit similar rates of mortality compression although the set of  $m_x$ ’s observed in 1800-04 exhibits slightly faster compression near the end of the projection interval, but this results should be interpreted with caution since trends near the end of the projection interval could be influenced by death heaping in the open ended age interval.

The contrast between the rates of decline in both  $S_{10}$  and  $e^\dagger$  for the initial  $m_x$ ’s corresponding to 1950-54 and 2000-04 in comparison to the initial  $m_x$ ’s corresponding to 1800-04,

1850-54, and 1900-04 is noteworthy. Applying the same proportional change in mortality over time, results in much less mortality compression if the initial mortality state corresponds to 1950-54 or 2000-04 in comparison to earlier eras. Nonetheless, especially for the projection with the age pattern of mortality change corresponding to what was observed 1875-79 to 1950-54, variability of age at death does decrease over the projection interval when  $m_x$ 's corresponding to 1950-54 and 2000-04 are used as starting states. Therefore, the shifting mortality phenomenon currently being observed in industrialized countries is not due solely to having reached some sort of threshold level of mortality beyond which variability of age at death is free of influence from the age pattern of mortality change. As will be demonstrated later, though, the ages at which mortality change matters most for these measures of variability of age at death have become increasingly compressed.

This simple analysis of applying fixed rates of proportional mortality change over time has yielded a number of insights. Greater divergence in the age pattern of mortality decline results in faster mortality compression than age patterns that are similar using a number of initial mortality conditions. Also, the rate of decline in variability of age at death given a fixed change in mortality over time depends on the initial mortality conditions. Certain initial mortality states are “primed for compression.” We investigate this further in the next section using perturbation analysis.

## 5.4 Sensitivity of $S_{10}$ and $e^\dagger$ to change in mortality

The simulations discussed in the last two sections suggest that the pace of mortality compression depends on the initial mortality state. In this section, I quantify the potential for changes in age-specific mortality rates to affect measures of variability of age at death using perturbation analysis. This method is currently being developed by Hal Caswell, and it is an extension of the methods found in (Caswell, 2006; Caswell, 2009, in press). A more detailed description of how I implement this method is provided in Chapter 4. In this section, I calculate the sensitivities of  $S_{10}$  and  $e^\dagger$  to changes in age-specific mortality rates using the initial set of  $m_x$  vectors that were used in the analysis in the previous section. I anticipate that the measures of variability of age at death will appear particularly sensitive to changes in mortality for the set of  $m_x$ 's corresponding to the mortality conditions observed for Swedish males in 1900-04.

The sensitivity of  $S_{10}$  to changes in age-specific mortality are presented in Figure 5.6. The graphs indicate the response of  $S_{10}$  to either absolute or proportional *increases* in mortality at each age. The age pattern of sensitivity is remarkably consistent across the different initial conditions; however,  $S_{10}$  grows more sensitive to absolute changes in mortality at younger ages over time as mortality rates at younger ages becomes increasingly low.

The bottom graph shows the absolute response of  $S_{10}$  to proportional changes in mortality at a particular age and reveals systemic differences in age patterns of responsiveness across the set of initial conditions. As anticipated, when  $S_{10}$  is estimated from the set of

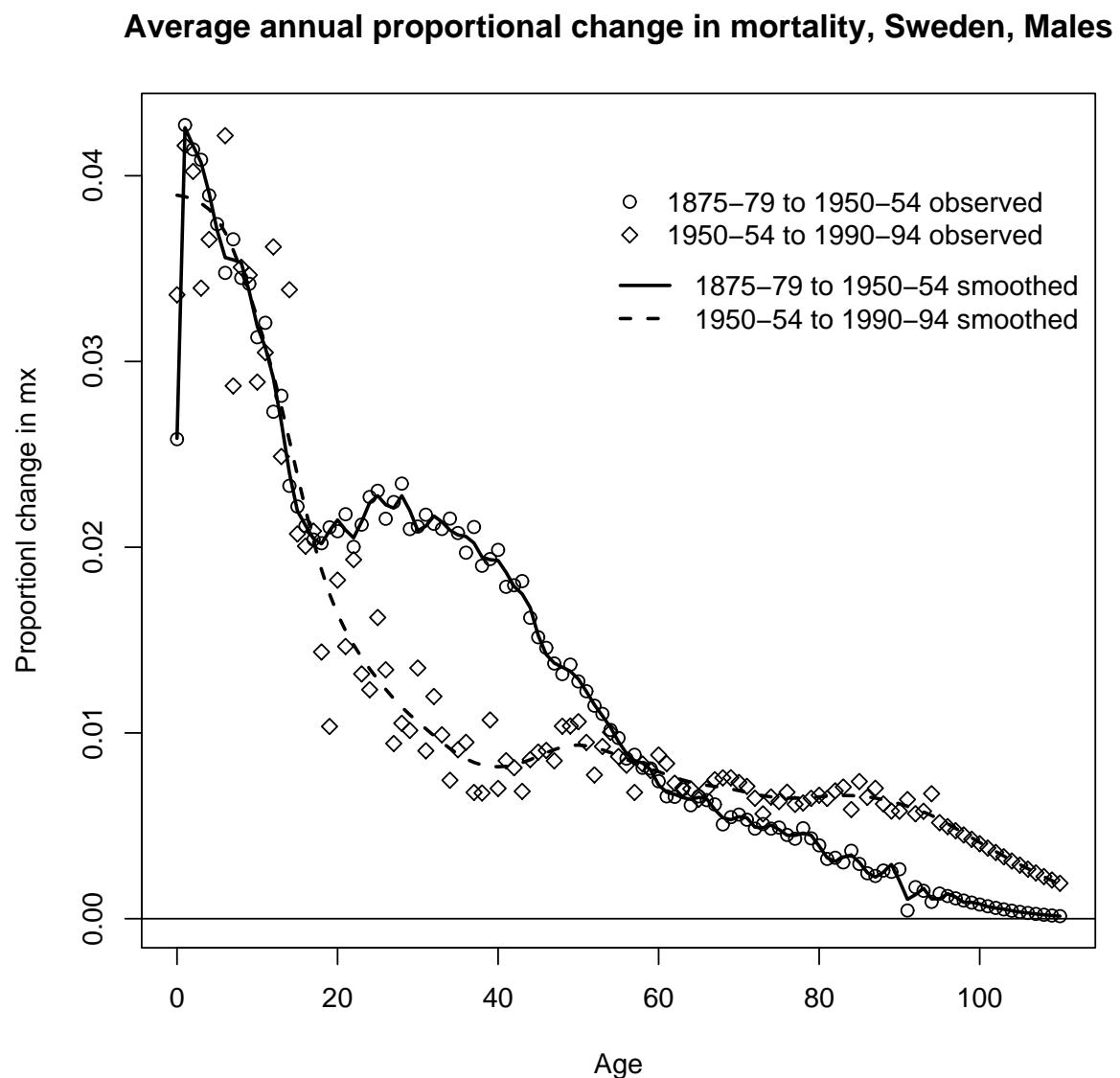


Figure 5.5: Observed and smoothed average annual proportional change in mortality for Swedish males 1875-79 to 1950-54 and 1950-54 to 1990-94. Data source: Human Mortality Database (HMD, 2009). Note: Data fit to with a cubic spline using `smooth.spline()` function in R.

$m_x$ 's corresponding to mortality in 1900-04 for Swedish males, the measure exhibits greater sensitivity to changes in mortality in the age groups 15-40 in comparison to  $S_{10}$  estimated from the set of  $m_x$ 's corresponding to 1800-04 and 1850-54. This suggests that rapid decreases observed in variability of age at death from 1876-1955 were attributable not only to divergence in the age pattern of mortality but also partly to the greater sensitivity of measures of variability of age at death to changes in age-specific mortality due to the initial mortality conditions.

This view is reinforced by looking at the sensitivity of  $e^\dagger$  to changes in age-specific mortality rates. As shown in Figure 5.7, the measure  $e^\dagger$  exhibits much more sensitivity to changes in mortality in infancy, childhood, and young adult ages for the initial set of  $m_x$ 's corresponding to 1900-04 in comparison to the other initial sets of  $m_x$ 's. For the initial  $m_x$ 's corresponding to 1800-04 and 1850-54,  $e^\dagger$  is not very sensitive to mortality declines from roughly age 10 to age 30 and after some point close to age 40 declines in mortality would actually result in an increase in  $e^\dagger$  (Zhang and Vaupel refer to this crossover age as the “age separating early from late deaths” (Zhang and Vaupel, 2009)). In contrast, declines in mortality up to almost age 60 will result in a decrease in  $e^\dagger$  and  $e^\dagger$  will respond more readily to the same proportional change in mortality at younger ages if the initial mortality conditions correspond to the set of  $m_x$ 's from 1900-04. For the initial sets of mortality rates corresponding to 1950-54 and 2000-04, declines in mortality into even older ages would result in a decline in  $e^\dagger$ , but  $e^\dagger$  is much less sensitive changes in mortality in infancy, childhood, and early adulthood in comparison to the set of  $m_x$ 's corresponding to 1900-04.

The sensitivity of  $e^\dagger$  to changes in infant mortality for Sweden during the period 1751-2004 is shown in Figure 5.8. As can be seen in this graph, if we consider absolute changes in  $m_0$ ,  $e^\dagger$  became more sensitive to changes in infant mortality over time as a result of increasing life expectancy over this period. If we consider proportional changes in mortality at age 0, the sensitivity of  $e^\dagger$  to these changes peaks around 1900. The decrease in the sensitivity of  $e^\dagger$  to changes in infant mortality after this point can be attributed to the decline in infant mortality rates.

As mortality conditions improve over time (from 1800-04 to 2000-04)  $e^\dagger$  becomes less responsive to changes in infant and childhood mortality while both  $e^\dagger$  and  $S_{10}$  become less responsive to changes in mortality during early adulthood. As a result of the decreasing influence of early life mortality, the shape of the sensitivity curve changes in a very fundamental way. Ignoring the “accident hump” around age 20, there is a general trend towards a concentration of the ages where  $S_{10}$  and  $e^\dagger$  are most sensitive to changes in mortality. Considering just the ages where increases in mortality lead to an increase in variability of age at death, for both  $e^\dagger$  and  $S_{10}$ , the measures actually become more sensitive to changes in mortality at older age ages in comparison to younger when the initial  $m_x$ 's correspond to 1950-54 or 2000-04. In contrast, for the initial  $m_x$ 's correspond to 1800-04, 1850-54, and 1900-04, changes in mortality at younger ages are always more important for  $S_{10}$  and  $e^\dagger$  than changes near the crossover age (the age where sensitivity of  $e^\dagger$  or  $S_{10}$  to changes in mortality is zero). The ages where an increase in mortality leads to decrease in  $S_{10}$  become

increasingly compressed over time as mortality conditions improve. Mortality conditions in 1900 were “primed for compression” not only because  $e^t$  and  $S_{10}$  were more responsive to changes in mortality at younger ages in comparison to other initial  $m_x$ ’s, but also because the ages where declines in mortality would lead to increase in  $S_{10}$  were more compressed than for the set of  $m_x$ ’s corresponding to 1800-04 and 1850-54.

In the next chapter, I examine actual trends in  $S_{10}$  for Sweden in the 19th and 20th centuries. Through decomposition analysis, I am able to quantify the contribution of changes in age-specific mortality rates to changes in  $S_{10}$  over time. These age-specific contributions reflect both the potential of mortality change at that age to impact  $S_{10}$  (as reflected in the sensitivity curves) as well as the actual pace of mortality decline at that age relative to other age groups. While these decomposition results are reflective of the actual age patterns of mortality change over time, which might be divergent or more similar across age, the results are nonetheless very closely related to the results of the sensitivity analysis that only reflect what would occur if proportional mortality change was fixed across age.

## 5.5 Conclusion

This analysis has revealed that both the age pattern of mortality change and current mortality conditions are important in determining the future course of variability of age at death. The rapid declines in variability of age at death observed in Sweden from 1876-1955 were due to both divergence in the age pattern of mortality decline and the greater sensitivity of measures of variability of age at death to changes in age-specific mortality rates during this time period. Similarly, the current stability in  $S_{10}$  is likely due partly to the similarity in rates of mortality change across age as well as the compression of the age interval where measures of variability of age at death are most sensitive to changes in mortality.

Given the changes over the course of the 20th century in the age pattern of sensitivity of measures of variability of age at death to changes in mortality, it is unlikely that we will observe further rapid declines in life span variability in countries like Sweden. As the ages where mortality change matters most for trends in variability become increasingly compressed, a pattern of mortality change that is divergent across age becomes less important.

Still, it is not clear that the age pattern of mortality change should be completely disregarded as we contemplate future mortality change. While it is tempting to adopt new forecasting models that take advantage of the shifting conditions currently observed, these mortality shifts may not necessarily be sustained in the long term if the age pattern of mortality change becomes more divergent across age in the future. In Chapter 8, I examine this issue from a cause-of-death perspective and find that changes in cancer related mortality could be particularly important for future trends in mortality compression.

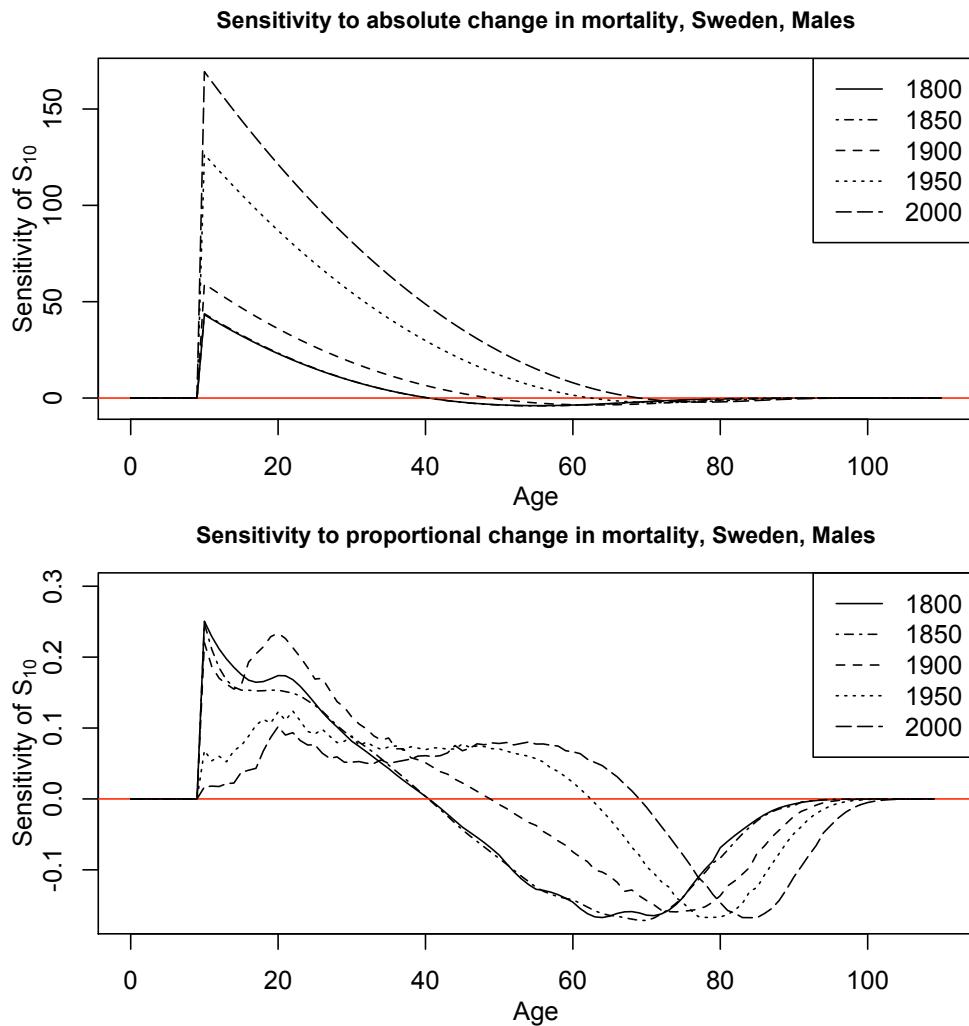


Figure 5.6: Sensitivity of  $S_{10}$  to either absolute or proportional changes in mortality at different ages. Data source: Human Mortality Database (HMD, 2009).

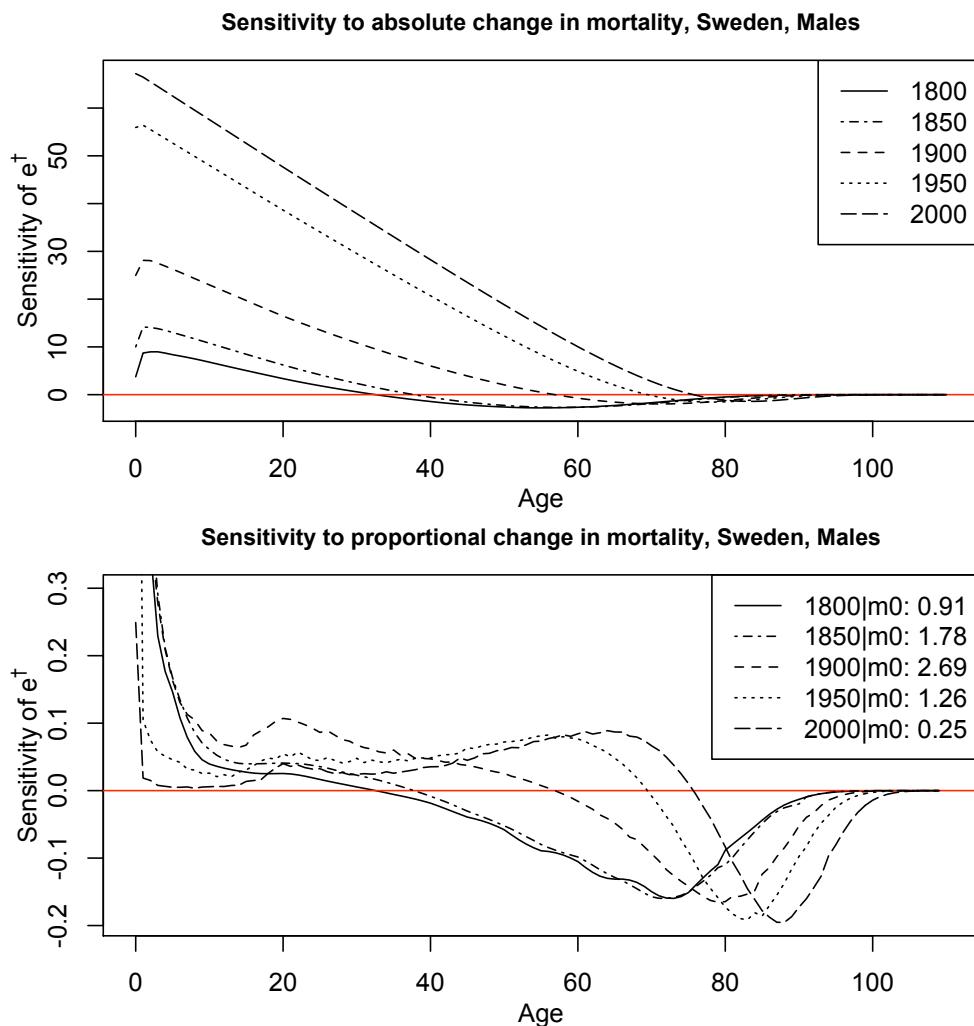


Figure 5.7: Sensitivity of  $e^t$  to either absolute or proportional changes in mortality at different ages. Data source: Human Mortality Database (HMD, 2009).

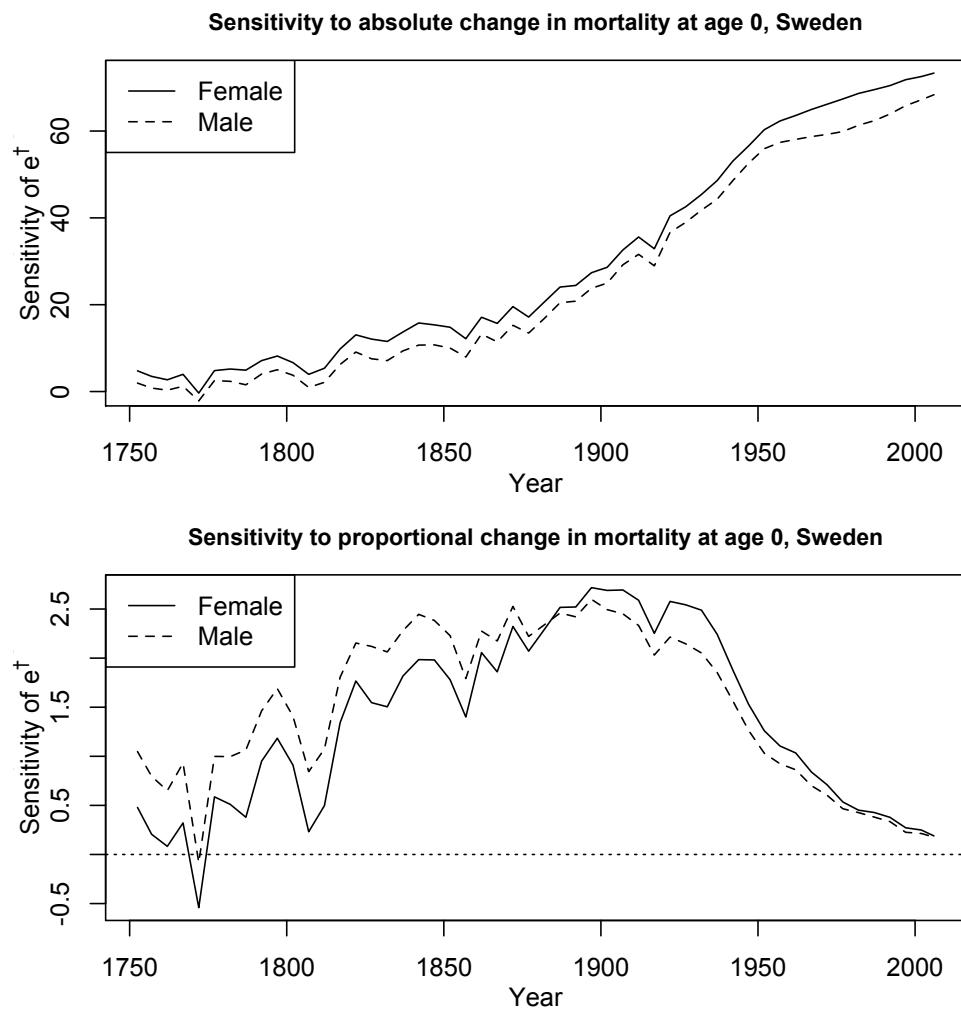


Figure 5.8: Sensitivity of  $e^{\dagger}$  to either absolute or proportional changes in mortality at age 0 for Swedish males. Data source: Human Mortality Database (HMD, 2009).

# Chapter 6

## Decomposing trends in variability of age at death

In the last chapter, I demonstrated that the potential for change in variability of age at death is dependent upon initial mortality conditions and that the age pattern of the sensitivity of  $S_{10}$  to changes in age-specific mortality has changed fundamentally in Sweden over the course of the epidemiological transition. In this section, I use decomposition analysis to show how actual changes in age-specific mortality influence actual trends in variability of age at death in Sweden as well as a number of other countries included in the Human Mortality Database with long historical records. Using decomposition techniques, I am able to quantify the contribution of age-specific changes in mortality to changes in  $S_{10}$  in the eras of mortality compression and shifting mortality. By comparing the decomposition results to the results of the sensitivity analysis presented in the last chapter, I find that divergence in the age pattern of mortality change is important in explaining trends in variability of age at death in both the era of mortality compression and the era of shifting mortality.

In addition to examining the total effect of changes in age-specific mortality rates,  $m_x$ , I also look at both the direct effect of age-specific mortality rates,  $m_x$ , on the death distribution,  $d_x$ , as well as the effect of  $m_x$  through the survivorship component of the death distribution,  $l_x$ . This analysis reveals that the direct effect is the dominant influence on trends in variability of age at death; however, the survival effect has become increasingly important at older ages in recent decades.

### 6.1 Decomposition of temporal trends in measures of variability of age at death

Temporal trends in  $S_{10}$  for a number of countries are presented in the Introduction (Chapter 1). As can be seen in the figures presented there and as was discussed in the literature review in Chapter 2, a period of rapid compression in mortality (as indicated by

declines in  $S_{10}$ ) occurred in the first half of the 20th century across a number of countries whose historical mortality experiences are documented in the HMD. In recent decades, there has been a transition from mortality compression to shifting mortality as  $S_{10}$  has stabilized while life expectancy continues to increase. In this section, I present results of decomposition analyses which quantify how changes in age-specific mortality rates,  $m_x$ , have produced these trends in  $S_{10}$ .

### 6.1.1 Decomposition methods

I decompose sex-specific trends in  $S_{10}$  in order to quantify the contributions of changes in age-specific mortality rates,  $m_x$ , to overall changes in sex-specific  $S_{10}$  over time using the HWP method, which is described in Chapter 4 (Horiuchi et al., 2008). These decompositions are carried out using life table data from ten countries in the HMD with long historical records: Belgium, Denmark, England and Wales, Finland, France, Italy, Netherlands, Norway, Sweden, and Switzerland. Using life tables with single year age groups and five year periods produces estimates of  $S_{10}$  that are both accurate and stable. I examine change over time across three forty year periods: 1880-84 to 1920-24, 1920-24 to 1960-64, and 1960-64 to 2000-04. During both the first and third time periods, the change in  $S_{10}$  is relatively modest in comparison to the large decline in  $S_{10}$  observed between 1920-24 to 1960-64 across all countries.

The results of the sensitivity analysis presented in Chapter 5 suggest that the age pattern of contributions of changes in age-specific mortality to changes in  $S_{10}$  will be modified over the course of the period of observation (1880-84 to 2000-04). Mortality change at younger ages will be most important in determining trends in variability of age at death in the earlier part of the period of observation while contributions from changes in mortality at older ages will become increasingly important in the latter part. This pattern will arise both because the age pattern of mortality change is more rapid at younger ages in comparison to older ages in the first part of the twentieth century (see Figure 5.2(b)) and because  $S_{10}$  is more sensitive to changes in mortality at younger ages during this earlier period (see Figure 5.6).

### 6.1.2 Decomposition results

Graphical results of the decomposition analysis for Sweden are presented in Figure 6.1 and tabular results for all countries included in the analysis are shown Table 6.1. Since many graphical figures showcasing decomposition results will be presented in the following chapters of the dissertation, it is worthwhile to explain in detail how to interpret Figure 6.1. Points below the zero line in these graphs indicate that changes in mortality at that age over the period of interest act to decrease  $S_{10}$ . Points above the line indicate that changes in mortality at this age over the period of interest act to increase  $S_{10}$ . The actual change in  $S_{10}$  can be found by integrating over the area delineated by the points representing age-specific contributions. Figure 6.1 reveals that the rapid decline in  $S_{10}$  observed during the period

1920-24 to 1960-64 was largely due to mortality improvements at younger ages with the peak contribution around age 20.

The tabular results in Table 6.1 confirm that the peak contributions to declining  $S_{10}$  during the period 1920-24 to 1960-64 are attributable to mortality improvements in the youngest age group, 10-29, for all countries included this analysis. During the period 1920-24 to 1960-64, declines in mortality in the 10-29 and 30-49 age group across countries contribute to declining trends in  $S_{10}$  (except for Finnish males 30-49). In contrast, declines in mortality in the older age groups, 50-69 and 70+, act to increase  $S_{10}$ , but these contributions are overwhelmed by negative contributions due to mortality improvements in the younger ages.

For the most recent period, 1960-64 to 2000-04, the results depicted in Figure 6.1 and tabulated in Table 6.1 indicate that the contributions of changes in mortality at younger ages, which would decrease  $S_{10}$  further, are increasingly being matched by mortality improvements at older ages, which act to increase  $S_{10}$ . Notice that the age pattern of contributions of changes in age-specific mortality rates to changes in  $S_{10}$  over the period 1960-64 to 2000-04 shown in Figure 6.1 only has one hump at the oldest ages unlike the age pattern of the sensitivity curve for 1950 and 2000 shown in Figure 5.6, which contains two humps on either side of the crossover age.

The decomposition results reflect the cumulative effect of both the sensitivity of the initial mortality conditions and divergence in the age pattern of mortality. Thus, the lack of a hump before the crossover age in the decomposition results is due divergence in the age pattern of mortality change at younger ages with younger age groups experiencing faster declines in mortality relative to older age groups before the crossover age (see 5.2(b)). Importantly, this indicates that changes at younger ages are still affecting trends in variability of age at death, and shifting mortality is not due solely to effects of changes in mortality on either side of the crossover age balancing out. This will become more evident in the cause-of-death analysis presented in Chapter 8.

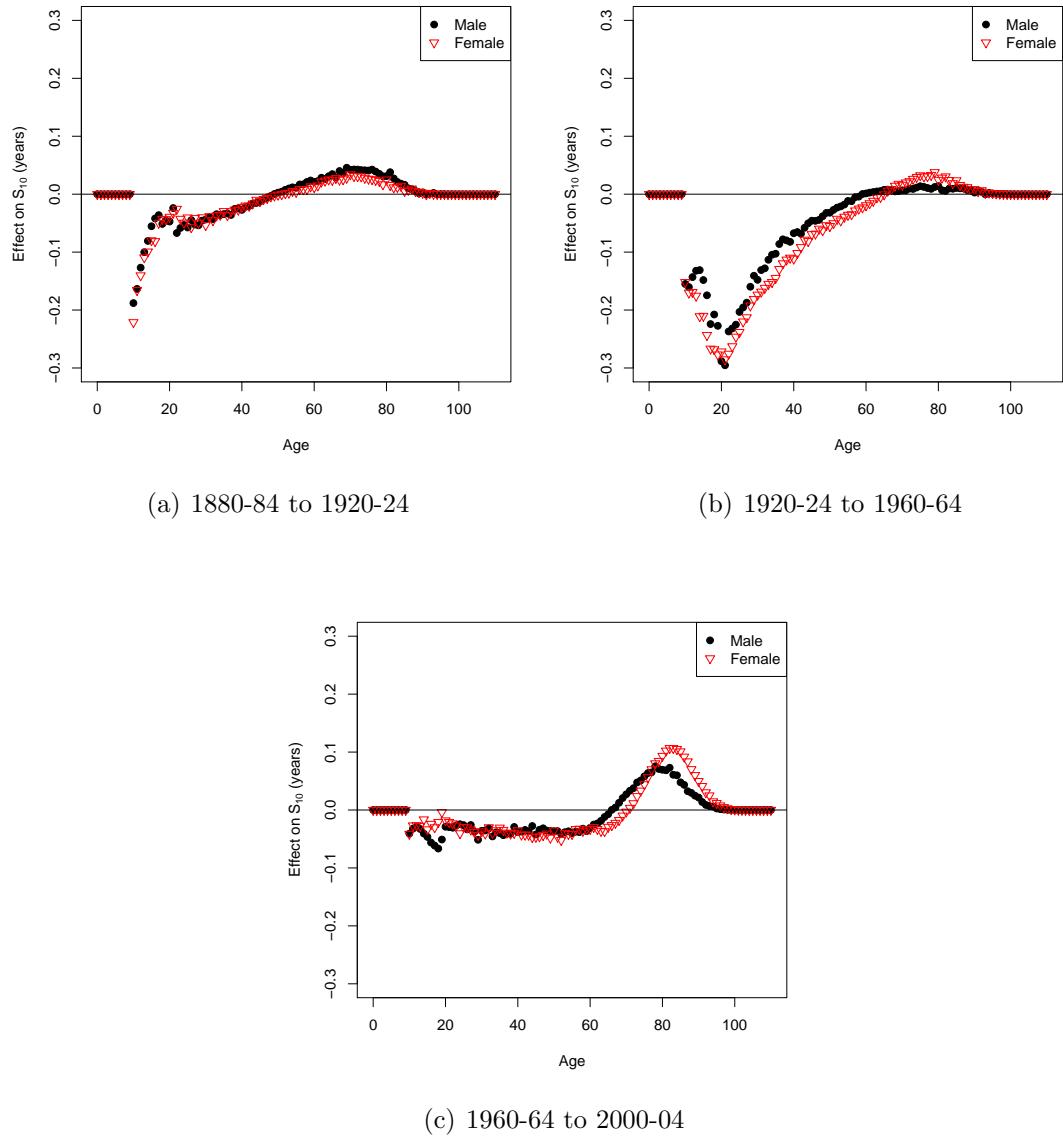


Figure 6.1: Results of decomposition of trends in  $S_{10}$  into the contributions of changes in age-specific mortality rates, Sweden, 1880-2004. Data source: Human Mortality Database (HMD, 2009).

Table 6.1: Contributions of changes in age-specific mortality rates,  $m_x$ , to changes in sex-specific  $S_{10}$  over three periods (in years). A negative sign indicates that the change in age-specific mortality observed between the two periods in that particular age group contributes to a decline in variability of age at death.

	Male			Female		
	1880-84 to 1920-24	1920-24 to 1960-64	1960-64 to 2000-04	1880-84 to 1920-24	1920-24 to 1960-64	1960-64 to 2000-04
	1920-24	1960-64	2000-04	1920-24	1960-64	2000-04
<b>Belgium</b>						
10-29	-1.24	-3.02	-0.67	-1.87	-4.07	-0.52
30-49	-0.66	-1.29	-0.62	-0.73	-2	-0.75
50-69	0.35	0.04	-0.16	0.22	-0.1	-0.66
70+	0.2	0.46	1.18	0.26	0.8	1.6
Total	-1.35	-3.81	-0.28	-2.12	-5.38	-0.33
<b>Denmark</b>						
10-29	-2.51	-2.68	-0.48	-3.01	-3.49	-0.37
30-49	-0.82	-1.23	-0.23	-0.78	-1.93	-0.64
50-69	0.44	0	-0.15	0.2	-0.28	-0.32
70+	0.37	0.41	0.74	0.04	0.8	1.43
Total	-2.52	-3.49	-0.12	-3.56	-4.91	0.1
<b>England and Wales</b>						
10-29	-1.63	-2.7	-0.62	-1.92	-3.57	-0.43
30-49	-0.73	-1.49	-0.52	-1.06	-1.77	-0.74
50-69	0.51	0.01	-0.38	0.44	-0.12	-0.72
70+	0.18	0.27	1.55	0.38	0.83	1.41
Total	-1.67	-3.91	0.04	-2.16	-4.64	-0.48
<b>Finland</b>						
10-29	-0.08	-4.54	-0.81	-0.53	-6	-0.59
30-49	0.01	-1.08	-1.07	-0.18	-2.22	-0.89
50-69	0.04	0.23	0.04	0.47	0.01	-0.88
70+	0.32	0.23	1.57	0.4	0.44	1.96
Total	0.3	-5.17	-0.26	0.17	-7.77	-0.4
<b>France</b>						
10-29	-1.45	-3.54	-0.74	-1.7	-4.89	-0.72
30-49	-0.24	-1.42	-0.77	-0.45	-2.18	-0.91
50-69	0.11	0.29	-0.09	0.36	-0.04	-0.86
70+	0.02	0.68	1.72	0.18	1.22	1.8

*Continued on next page*

	Male			Female		
	1880-84 to 1920-24	1920-24 to 1960-64	1960-64 to 2000-04	1880-84 to 1920-24	1920-24 to 1960-64	1960-64 to 2000-04
	Total	-1.58	-3.99	0.12	-1.6	-5.89
<b>Italy</b>						
10-29	-1.59	-3.91	-1.08	-1.87	-5.3	-0.94
30-49	-0.26	-1.31	-1.03	-0.41	-2.2	-1.15
50-69	0.64	0.19	-0.48	0.77	0.02	-1.02
70+	0.08	0.76	1.41	0.38	1.26	1.82
Total	-1.14	-4.27	-1.19	-1.13	-6.22	-1.29
<b>Netherlands</b>						
10-29	-2.54	-2.7	-0.71	-2.52	-3.49	-0.39
30-49	-1.02	-1.13	-0.51	-1.07	-2.12	-0.39
50-69	0.47	0.11	-0.41	0.29	-0.36	-0.4
70+	0.4	0.71	0.49	0.4	1	1.18
Total	-2.69	-3.01	-1.14	-2.9	-4.97	0
<b>Norway</b>						
10-29	-1.5	-4.56	-0.46	-1	-5.74	-0.17
30-49	-0.31	-1.71	-0.51	-0.48	-2.51	-0.49
50-69	0.28	0.1	-0.39	0.27	-0.33	-0.52
70+	0.22	0.2	0.72	0.27	0.32	1.29
Total	-1.32	-5.96	-0.64	-0.94	-8.27	0.1
<b>Sweden</b>						
10-29	-1.39	-3.87	-0.77	-1.45	-4.53	-0.56
30-49	-0.48	-1.57	-0.74	-0.48	-2.21	-0.8
50-69	0.44	-0.1	-0.45	0.28	-0.39	-0.67
70+	0.58	0.21	1.1	0.39	0.51	1.52
Total	-0.85	-5.33	-0.86	-1.27	-6.62	-0.51
<b>Switzerland</b>						
10-29	-1.72	-2.45	-1.08	-2	-3.96	-0.59
30-49	-0.67	-1.38	-0.9	-0.71	-2.18	-0.86
50-69	0.45	0.29	-0.42	0.58	-0.11	-0.93
70+	0.28	0.82	1.63	0.47	1.31	1.79
Total	-1.66	-2.72	-0.76	-1.66	-4.95	-0.59

## 6.2 A closer examination of the relationship between $m_x$ and $d_x$ taking into account direct and survival effects of changes in $m_x$

When I measure the effect of changes in age-specific mortality rates on trends in  $S_{10}$ , I am trying to quantify how changes in age-specific mortality,  $m_x$ , affect the death distribution,  $d_x$ . In the continuous case, the death distribution,  $d_x$ , is the product of age-specific mortality rates,  $m_x$ , and survivorship,  $l_x$  ( $d_x = l_x m_x$ ). Also,  $l_x$  can be derived solely from  $m_x$  using the equation  $l_x = e^{-\int_0^x m_a da}$ . Therefore,  $d_x$  can be expressed solely as a function of  $m_x$  as follows:

$$d_x = e^{-\int_0^x m_a da} m_x$$

As the above equation indicates,  $m_x$  affects  $d_x$  through two pathways: directly through the  $m_x$  term and also through the survivorship term,  $e^{-\int_0^x m_a da}$ . In this section, I consider how the changes in  $m_x$  are operating through these two pathways to affect changes in variability of age at death as measured by  $S_{10}$ . The effect on  $S_{10}$  of change in  $m_x$  that occurs in through the  $m_x$  term can be considered the direct effect, and the effect on  $S_{10}$  of a change in  $m_x$  that occurs through the survivorship term can be thought of as the survival effect. For ease of discussion, it will be helpful to relabel the two  $m_x$  terms to distinguish the direct term from the survivorship term  $m_{x,s}$ :

$$d_x = e^{-\int_0^x m_{a,s} da} m_{x,d}$$

In considering how  $m_x$  influences  $d_x$  directly and through the survivorship term, it is useful to take a step back and consider the relative importance of  $l_x$  and  $m_x$  for  $d_x$  by age. In Figures 6.2 and 6.3,  $l_x$ ,  $m_x$ , and  $d_x$  for Sweden in 1751 and 2000 are depicted. As can be seen in both of these graphs,  $d_x$  is strongly influenced by  $m_x$  at younger ages, where  $l_x$  is close to one. At older ages,  $d_x$  is highly influenced by  $l_x$  as  $l_x$  approaches zero and  $m_x$  approaches one. It follows that changes in  $m_x$  at younger ages should have greater impact through the direct component while the survivorship component will become more important for mortality changes at older ages. The decomposition results presented below confirm these intuitions.

### 6.2.1 Decomposition methods

In order to estimate the direct and survival effects of changes in  $m_x$  on trends in  $S_{10}$ , I adopt the same methodology that I used to decompose the changes in  $S_{10}$  into the total effect of changes in age-specific mortality rates,  $m_x$ . This basic method is outlined in Chapter 4. Here, I outline how I adopt the method in order to estimate the separate contributions of  $m_{x,d}$  and  $m_{x,s}$ .

$S_{10}$  can be expressed in terms of  $m_{x,d}$  and  $m_{x,s}$ :

$$(S_{10}(t))^2 = \frac{\sum_{x=10}^T \left( x + a_x - \sum_{x=10}^T x(\exp(-\sum_0^x m_{x,s}(t)))m_{x,d}(t) \right)^2 \times (\exp(-\sum_0^x m_{x,s}(t)))m_{x,d}(t)}{\sum_{x=10}^T (\exp(-\sum_0^x m_{x,s}(t)))m_{x,d}(t)}$$

In this case,  $S_{10}$  is function of both  $m_{x,s}$  and  $m_{x,d}$ :

$$S_{10} = f(m_{1,s}, m_{2,s}, \dots, m_{T,s}, m_{1,d}, m_{2,d}, \dots, m_{T,d})$$

Take partial derivatives of  $S_{10}$  with respect to  $m_{i,s}$  and  $m_{i,d}$  to find the contribution of changes in these components to changes in  $S_{10}$ :

$$c_{i,s} = \int_{m_{i,s,1}}^{m_{i,s,2}} \frac{\partial S_{10}}{\partial m_{i,s}} \frac{dm_{i,s}}{dt} dt \text{ or } c_{i,d} = \int_{m_{i,d,1}}^{m_{i,d,2}} \frac{\partial S_{10}}{\partial m_{i,d}} \frac{dm_{i,d}}{dt} dt$$

To give a general picture of how this decomposition works, consider the contribution,  $c_{i,s}$ , of a change in age-specific mortality through the survivorship term at a particular age  $i$ ,  $m_{i,s}$ , to change in  $S_{10}$  between two time points. Computationally, estimating  $c_{i,s}$  involves changing  $m_{i,s}$  slightly over a narrow interval of time between time 1 and 2 while holding all of the other age-specific mortality components constant (even  $m_{i,d}$ ) at the value observed at the midpoint of the narrow interval and computing the change in  $S_{10}$ . This is repeated for each narrow interval between time 1 and 2, and the total effect is found by summing across intervals.

In reality,  $m_{i,s}$  is always equal to  $m_{i,d}$ , and the two components will always vary together. This, however, does not prohibit the examination of their separate effects. By taking partial derivatives of  $S_{10}$  with respect to  $m_{i,s}$  and  $m_{i,d}$ , which is the basis for this method of decomposition, one is able to distinguish the direct effect of changes in  $m_x$  from the survival effect.

The methodology is consistent with what was used for the initial decompositions of changes in  $S_{10}$  into the contributions of  $m_x$ ; however, the number of covariates has doubled. Note that these two sets of decompositions are related in that the total effect of a particular change in  $m_x$  on  $S_{10}$  should be the sum of the direct and survival effects :

$$c_i = c_{i,s} + c_{i,d}$$

### 6.2.2 Decomposition results

Greater understanding of the underlying mortality forces at work in the most recent period of relative stability in  $S_{10}$  comes from decomposing changes in  $S_{10}$  into the direct ( $m_{x,d}$ ) and survival ( $m_{x,s}$ ) effects of changes in age-specific mortality. The contributions of direct and survival effects to changes in  $S_{10}$  by age are depicted graphically for Sweden in

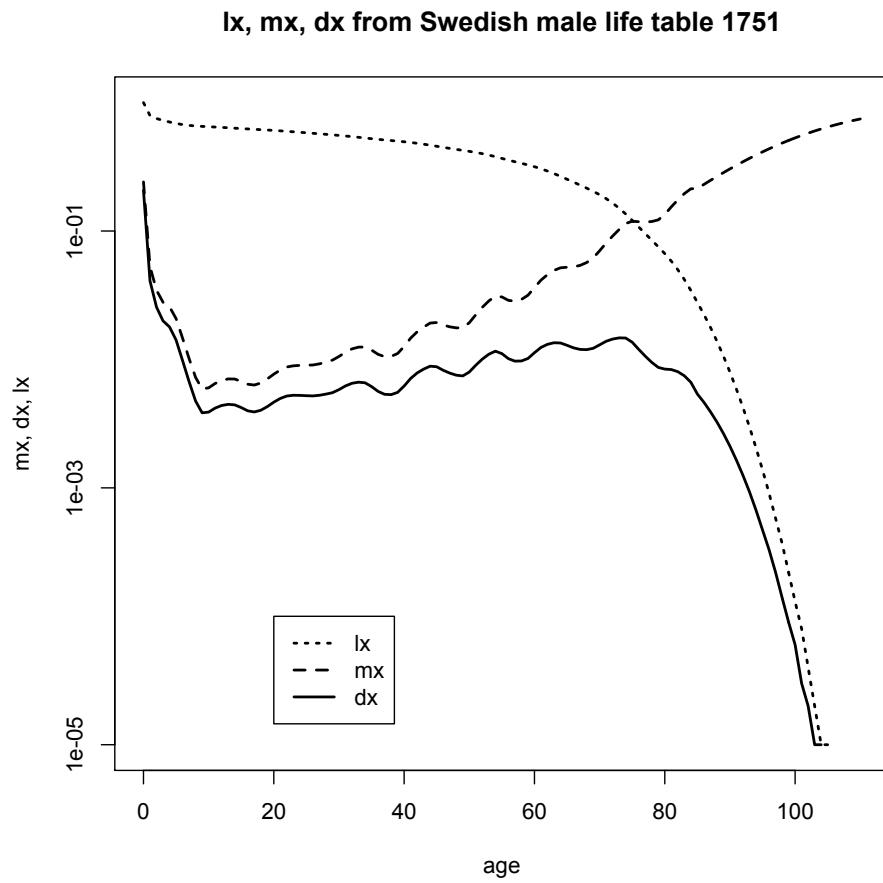


Figure 6.2:  $l_x$ ,  $m_x$ ,  $d_x$  for Sweden 1751. Data source: Human Mortality Database (HMD, 2009).

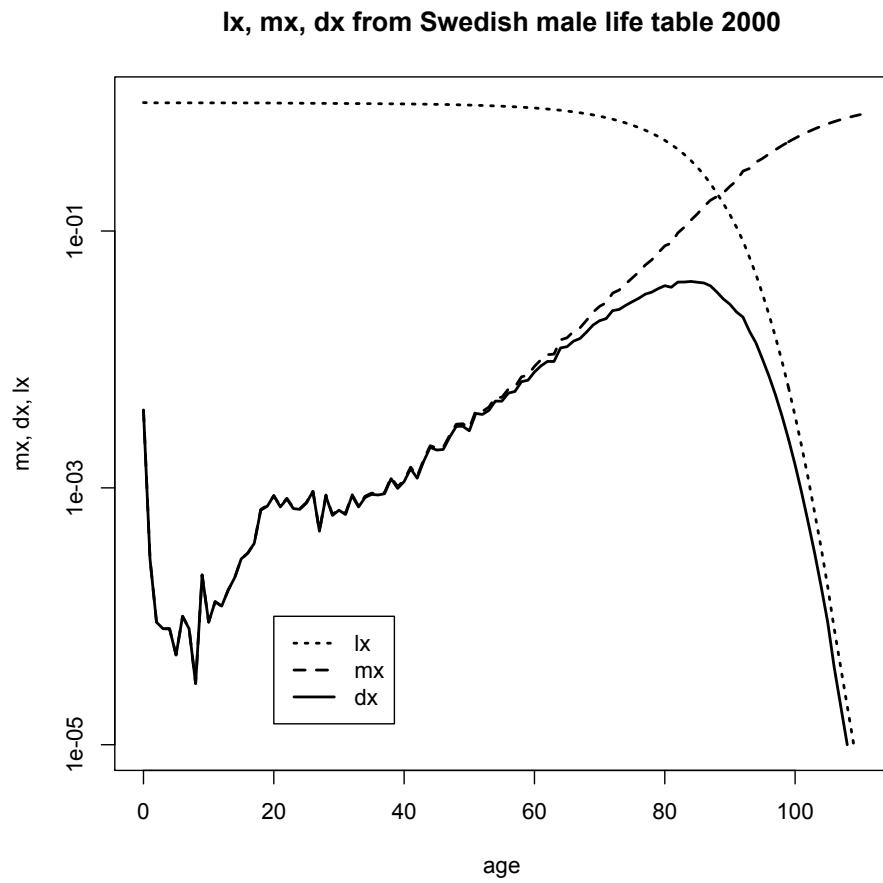


Figure 6.3:  $l_x$ ,  $m_x$ ,  $d_x$  for Sweden 2000. Data source: Human Mortality Database (HMD, 2009).

Figure 6.4. In these graphs, the total effect of changes in  $S_{10}$  across the different age groups is largely driven by the direct effect, changes in  $m_{x,d}$ . The total effect of change in mortality closely parallels the direct effect line across all of the age groups except for the oldest age groups in the third period. Intuitively, this makes sense because  $d_x$  is largely dependent upon the  $m_x$  component (as opposed to the  $l_x$  component) at younger ages as demonstrated in Figures 6.2 and 6.3. Rapid declines in mortality at younger ages largely operated through the direct component,  $m_{x,d}$ , to decrease  $S_{10}$  during the period of rapid change from 1930-1960. Effects of the direct component,  $m_{x,d}$ , are probably also larger than the survival effects because  $m_{x,d}$  is not operationalized through an exponential term like  $m_{x,s}$ .

Compared to the second period, in the third period, the survival effect becomes more prominent although it is not necessarily as large as the direct effect. This again seems somewhat intuitive as  $d_x$  is more highly influenced by the survivorship component  $l_x$  at older ages and changes in mortality in the oldest ages have become more important in recent times. The survival component,  $m_{x,s}$ , acts to balance the direct component,  $m_{x,d}$ . Specifically, from ages 60-90, a large positive hump for the  $m_{x,d}$  effect is balanced by a smaller negative  $m_{x,s}$  hump. At the oldest ages (90+), the survivorship effect,  $m_{x,s}$  becomes positive. The total effect line follows the survivorship effect at this point as the direct effect becomes slightly negative. This survival effect at the oldest ages was nonexistent before the most recent period. Could it become more important in the future and lead to an increase in  $S_{10}$ ?

### 6.3 Conclusion

In this chapter, I have used decomposition analysis in order to better understand how underlying changes in age-specific mortality rates produce mortality compression and shifting mortality. The major declines in  $S_{10}$  observed before the period of relative stability were driven by declines in mortality in the young adult ages. While declines in mortality at younger ages in more recent time still contribute to a decline in  $S_{10}$ , the measure of variability of age at death remains stable because declines in mortality at older ages contribute to an increase  $S_{10}$  and thus balance out improvements at younger ages; however, the decomposition results do not mirror the sensitivity results indicating that the current shifts in the death distribution being observed are not due to a non-divergent age pattern of mortality change as expected.

The analysis presented in this chapter also indicates that the shifting mortality era is unique in that changes in  $m_x$  influence the death distribution,  $d_x$ , through both the direct,  $m_{x,d}$ , and survivorship term,  $m_{x,s}$ , whereas the direct effect dominated in earlier eras.

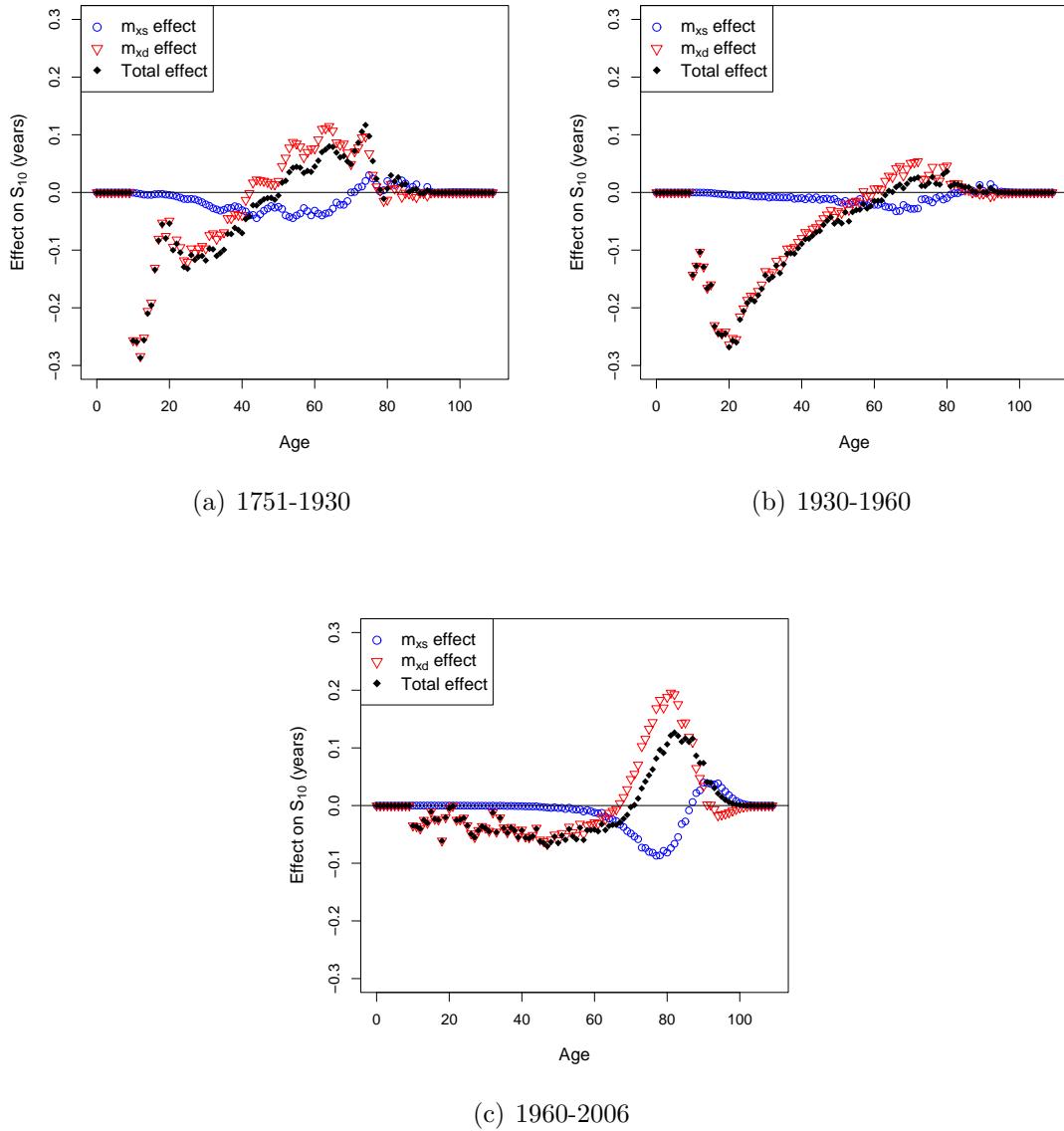


Figure 6.4: Decomposition of changes in  $S_{10}$  into direct ( $m_{xd}$ ) and survivorship ( $m_{xs}$ ) effects for Swedish females over three periods. Data source: Human Mortality Database (HMD, 2009).

# Chapter 7

## Understanding differences in variability of age at death

In this chapter, I examine differences in variability of age at death between groups using decomposition analysis. As demonstrated in Chapter 1, mortality compression gave way to shifting mortality around the mid-point of the 20th century in many of the countries whose mortality experience is documented in the Human Mortality Database. The graphical depiction of trends in  $S_{10}$  for different countries in Figure 1.2 makes apparent that there does not appear to be a biologically determined asymptote in variability of age at death that prompts the flat trends in these measures during the shifting mortality era. Rather countries transition from mortality compression to shifting mortality at different levels of variability. In this chapter, I try to understand how differences in variability of age at death across countries and between the sexes are influenced by differences in age-specific all-cause mortality. In the next chapter, I take up the same question of gender differences in variability of age at death from a cause-of-death perspective using a series of cause-of-death data from France.

This chapter contains two main parts. In the first part of the chapter, I examine the emergence of the gender gap in variability of age at death observed across countries during the course of the epidemiological transition. In the second part of the chapter, I investigate cross-country differences in variability of age at death. Specifically, I try to understand why the United States exhibits higher levels of variability of age at death in comparison to levels of variability observed Sweden. In the case of gender differences, my results indicate that females exhibit lower variability of age at death because they enjoy lower rates of premature mortality in comparison to males. In contrast, cross-country differences between Sweden and the US are the result of both US disadvantage in premature mortality and advantage in old age mortality. These different scenarios prompt a discussion of whether lower variability of age at death is necessarily advantageous.

## 7.1 Emergence of the gender gap in variability of age at death

Presently, females enjoy an advantage over males with regards to life expectancy in most areas of the world. Much research has focused on the emergence of the gender gap in life expectancy and trends in the gap over time (Retherford, 1975; Vallin, 1993; Glei and Horiuchi, 2007). It has also been documented in developed countries that females currently experience lower variability of ages at death in comparison to males (Wilmoth and Horiuchi, 1999; Edwards and Tuljapurkar, 2005; Glei and Horiuchi, 2007). In this section, I investigate the evolution of the gender gap in variability of age at death. In documenting sex-specific trends in variability of age at death for a number of countries, I discover that historically females generally experienced higher or similar levels of variability of age at death in comparison to males. Like the emergence of the gender gap in life expectancy, the gender gap in variability of age at death is a relatively recent phenomenon spurred by changes in the epidemiological environment.

Previous studies utilized age and cause-specific mortality data to explore the development of the female mortality advantage in terms of life expectancy and/or age-specific mortality rates (Enterline, 1961; Retherford, 1975; Vallin, 1993; Waldron, 2003); however, it is unlikely that the results of the decomposition analyses utilized in this chapter and the following chapter to investigate sex-specific differences in variability of age at death will mirror results of these prior studies of the gender gap in life expectancy. As Edwards and Tuljapurkar found in their study of variability of age at death, trends in variability of adult life span do not necessarily follow the same path as trends in life expectancy (Edwards and Tuljapurkar, 2005). Additionally, measures of variability of age at death are more sensitive to changes at the tails of the death distribution in comparison to the measure of the mean of the distribution, life expectancy, especially in situations of low infant mortality. Indeed, the results of the decomposition analyses presented here and in the next chapter reveal that changes in mortality at young adult ages have more influence on gender differences in variability of age at death in comparison to their impact on gender differences in life expectancy.

### 7.1.1 Trends in sex-specific variability of age at death

Interesting trends in sex-specific  $S_{10}$  emerge across countries and geographically defined areas in more developed and less developed regions alike. Most notably, males have not always experienced greater variability of age at death in comparison with females. The gender gap in  $S_{10}$  has emerged only recently.

Trends in sex-specific  $S_{10}$  based on all of the single year period life tables included in the Human Mortality Database are presented in Figures 7.1(a) and 7.1(b). While the trends in  $S_{10}$  across time appear similar for males and females, Figure 7.1(c), which shows female  $S_{10}$  minus male  $S_{10}$  as a function of time, reveals that the relationship between male and

female  $S_{10}$  changed fundamentally around the midpoint of the 20th century. Prior to 1938, females experienced greater  $S_{10}$  than males in the majority of countries and areas included in the HMD with historical records that extend back to this period. In contrast, since 1958, male  $S_{10}$  has been greater than female  $S_{10}$  for all countries and areas represented in the HMD. In the year 2003, the country-specific gender gap in  $S_{10}$  ranged from .235 years in the Netherlands to 2.812 years in Lithuania.

Figure 7.1(d) includes lines that trace the historical trends in sex-specific  $S_{10}$  for four particular countries. Solid lines indicate trends in male  $S_{10}$  while the female experience is documented with broken lines. Countries are coded by color. In general, the gap between male and female  $S_{10}$  has been especially pronounced in the former Soviet countries as illustrated by the sex-specific trend lines for Russia. Unfortunately, the data series for most of the former Soviet countries is not long enough to investigate whether female  $S_{10}$  might have been higher than male  $S_{10}$  in periods prior to the midpoint of the 20th century.

The western European countries in the Human Mortality Database tend to have the longest historical records, and these records provide evidence that female adult life span was once more variable than male. In some western European countries, prior to the first half of the 20th century, there was an obvious gender gap in  $S_{10}$  with males experiencing lower levels variability of age at death. In the remaining countries, male and female  $S_{10}$  levels were similar. The sex-specific trends in  $S_{10}$  for France and Sweden shown in Figure 7.1(d) illustrate the historical experiences of a pronounced gender gap and similar levels of  $S_{10}$  respectively.

Across both the European and non-European countries and areas included in the Human Mortality Database, it is clear that around World War II and in the period immediately following the war females gained a significant advantage over males in terms of lower variability of age at death. As Edwards and Tuljapurkar observed in their work,  $S_{10}$  levels have largely stabilized since the 1960s in more developed countries (Edwards and Tuljapurkar, 2005). Thus for the past fifty years, females have continued to experience lower variability of age at death in comparison to males.

While females experience lower  $S_{10}$  in comparison to males within each country in the most recent period, this relationship does not hold across countries. As can be seen in Figure 7.1(d),  $S_{10}$  for females in the United States and Russia in the most recent years available was higher than the  $S_{10}$  for males in Sweden. It appears that there is greater variability across countries in  $S_{10}$  than there is between sexes in a particular country. For instance, the gap between Sweden and the United States in  $S_{10}$  in the period 2000-04 was 2.6 years for males and 2.0 years for females. In contrast, the difference between Swedish males and females was .9 years, and the difference between males and females in the US was 1.5 years in the same period. I investigate the cross-country differences between Sweden and the United States in more detail in the next section.

Evidence from life tables included in the WHO collection suggests that the female advantage in variability of age at death may have been gained at a later point in time for countries and geographically defined areas in less developed regions in comparison to more developed.

Figure 7.2 shows trends in the difference between female and male  $S_{10}$  for life tables included in the WHO collection of 1,802 life tables that do not overlap with the HMD data set. Similar to countries and areas in more developed regions, historical female disadvantage in  $S_{10}$  prior to the midpoint of the twentieth century is observable in Chile, Sri Lanka, South Africa, and Taiwan. A long historical series from Singapore indicates slightly higher female  $S_{10}$  into the 1960s with male  $S_{10}$  higher than female  $S_{10}$  in more recent years. Single year observations from the 1970s for India, Iran, Bangladesh<sup>1</sup>, Peru, and the Republic of Korea offer evidence of higher female  $S_{10}$  in comparison to male during this period. These single year observations offer evidence that the more rapid gains by females in the period around the midpoint of the 20th century that led to the emergence of the gender gap in  $S_{10}$  within countries and areas in more developed regions did not necessarily take place during the same period for countries and areas in less developed regions. Still, the trend towards an eventual female advantage in  $S_{10}$  observable across geographically defined areas with more complete records indicates that this advantage might be the end product of the epidemiological transition.

### 7.1.2 Sensitivity of results to measure of variability of age at death

In this section, I compare sex-specific trends in  $S_{10}$  to trends obtained using other measures of variability of age at death in order to verify the results described above. I compare trends in  $S_{10}$  to trends observed using the measure  $e^\dagger$ , the years of life lost in the life table due to premature death, calculated either using the entire death distribution ( $e_0^\dagger$ ) or only using deaths occurring over age 10 ( $e_{10}^\dagger$ ). Just using the death distribution above age 10 to calculate  $e_{10}^\dagger$  allows me to make a clearer comparison between  $e^\dagger$  and  $S_{10}$ . I also analyze sex-specific trends in entropy ( $\bar{H}$ ), a measure related to  $e^\dagger$ , which indicates the level of premature mortality relative to the level of life expectancy:  $\bar{H} = \frac{e^\dagger}{e_0}$ .  $S_{10}$ ,  $e_{10}^\dagger$ , and  $e_0^\dagger$  are absolute measures of life span disparity while  $\bar{H}$  is a relative measure of disparity (relative to the level of life expectancy).

Sex-specific trends in  $S_{10}$ ,  $e_{10}^\dagger$ , and  $e_0^\dagger$ , and  $\bar{H}$  for Sweden were shown in Chapter 5 in Figure 5.3. When variability of age at death is measured using  $S_{10}$ , there seems to be a slight disadvantage for females prior to the 20th century. In contrast, trends in  $e_0^\dagger$  and  $e_{10}^\dagger$  indicate more similarity in variability of age at death for males and females prior to the 20th century. When life span disparity is measured relatively using  $\bar{H}$  rather than absolutely, levels appear to always be slightly lower for females, whom generally experience greater life expectancy than males. Also, when life span disparity or variability of age at death is measured absolutely a gap in these measures emerges between males and females in the latter half of the 20th century. In contrast, when measured relatively using  $\bar{H}$ , no such gap emerges. This difference in trends based on whether absolute or relative measures are used is especially interesting. The focus on the current gap in variability of age at death between females and males hinges on absolute measures of life span disparity being more relevant

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<sup>1</sup>  $S_{10}$  for Bangladesh is based on data collected from the MATLAB demographic surveillance system

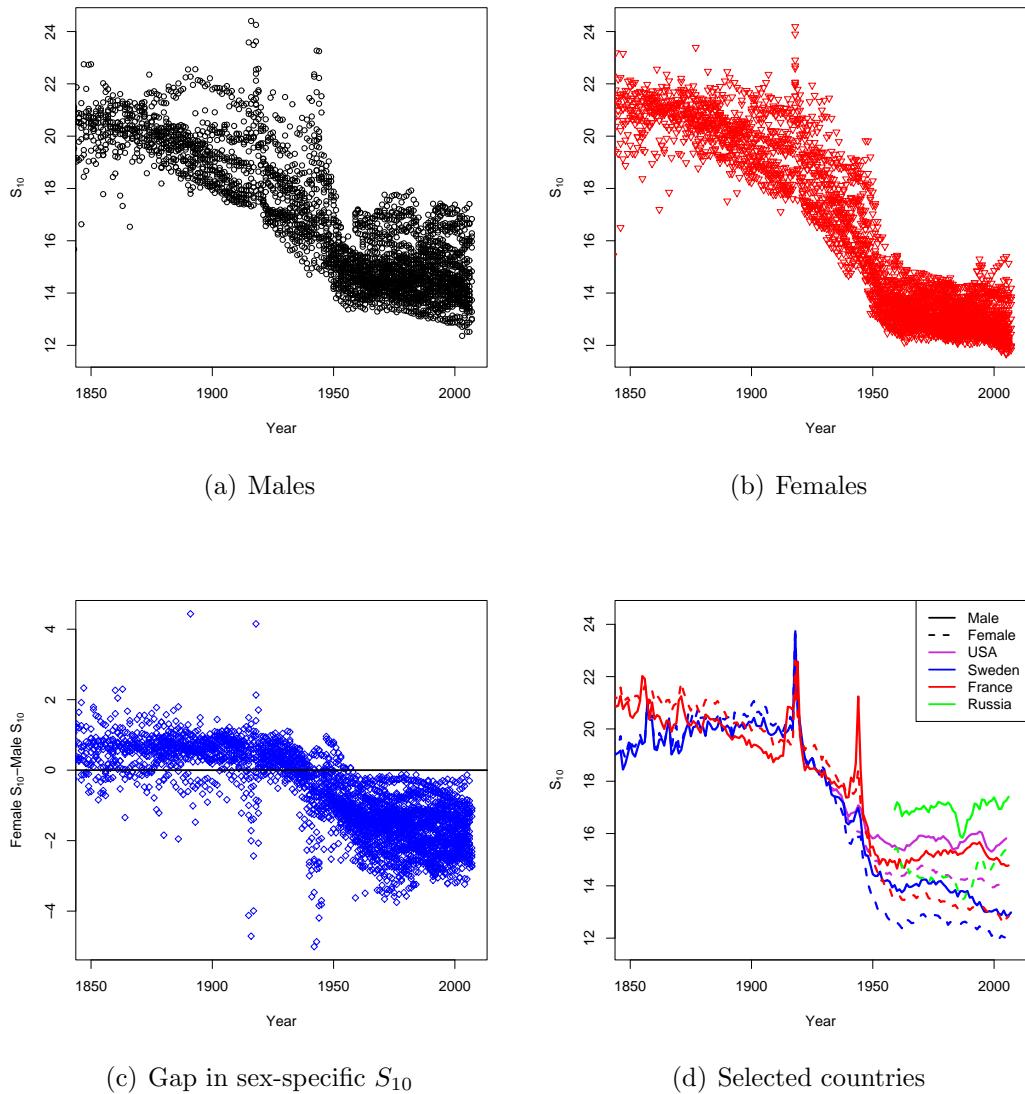


Figure 7.1: Trends in sex-specific  $S_{10}$  and the gender gap in  $S_{10}$  among countries and areas included in the HMD data set. Data source: Human Mortality Database (HMD, 2009).

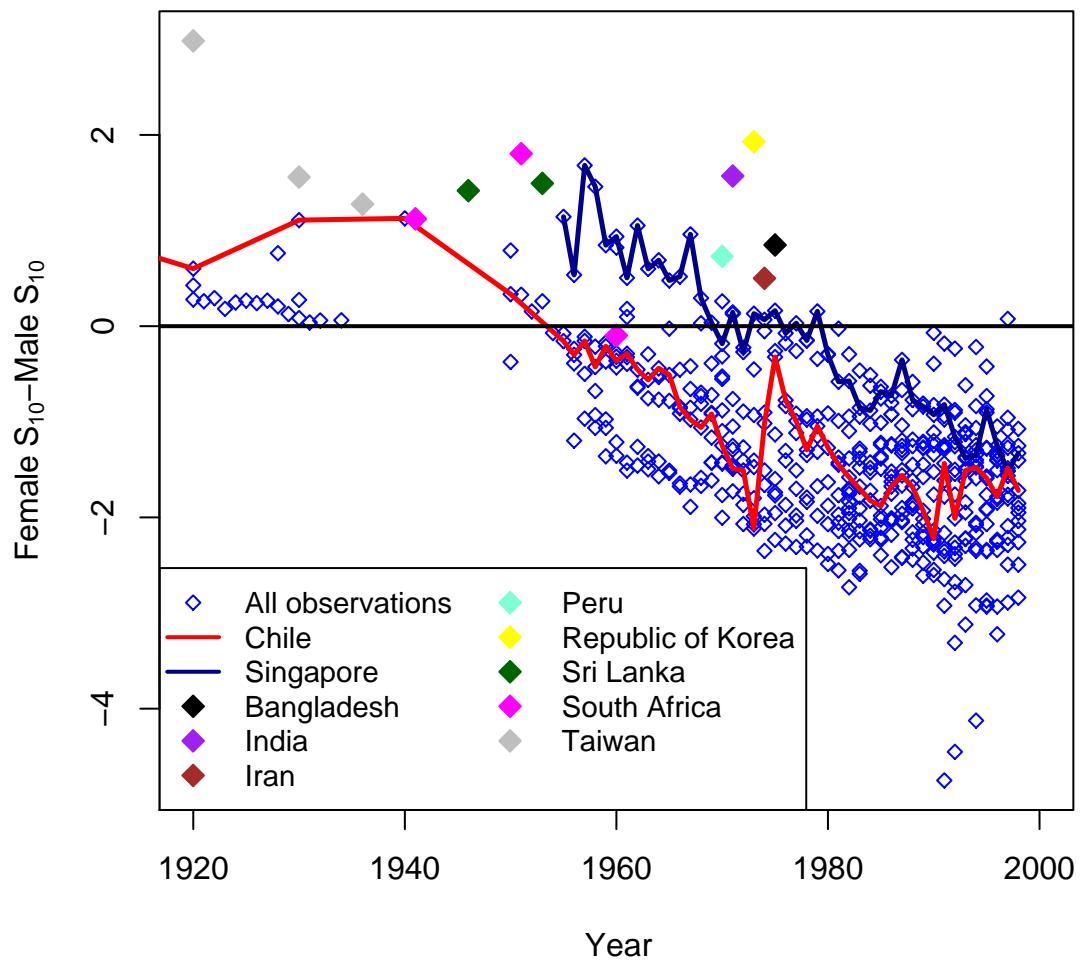


Figure 7.2: Trends in the gender gap in  $S_{10}$  among countries and areas in the WHO collection of 1,802 life tables that do not overlap with the HMD data set. Data source: WHO 1,802 life tables (Murray et al., 2003).

than relative measures. A discussion of the arguments for using absolute versus relative measures of variability is presented in Chapter 4.2.

In the remainder of this section, I carry out decomposition analyses, which I describe in the next section, in order to understand what changes in age-specific mortality led to females experiencing lower variability of age at death in comparison to males when variability is measured using  $S_{10}$ .

### 7.1.3 Decomposition methods

In order to understand the emergence of the gender gap in variability of age at death, I perform two different decomposition analyses. Both sets of analyses are carried out using the HWP decomposition method, which I described in Chapter 4 (Horiuchi et al., 2008). First, I decompose sex-specific trends in  $S_{10}$  in order to quantify the contributions of changes in age-specific mortality rates,  $m_x$ , to overall changes in sex-specific  $S_{10}$  over time. The results for this analysis indicate how changes in age-specific mortality have impacted trends in variability of age at death for each sex separately. I examine change over time across three forty year periods: 1880-84 to 1920-24, 1920-24 to 1960-64, and 1960-64 to 2000-04. In the first period,  $S_{10}$  levels were similar for males and females and the trend in  $S_{10}$  was either stable or exhibited slow decline. Rapid decline in  $S_{10}$  occurred during the second period from 1920-24 to 1960-64 for both sexes although the pace of decline was faster for females. The final period from 1960-64 to 2000-04 was largely characterized by stagnation in  $S_{10}$  with females experiencing lower levels of variability in comparison to male. The results of this analysis were also presented in Chapter 6 where I examine the transition from mortality compression to shifting mortality. In this chapter, I reinterpret these results with an aim of understanding how the gender gap in variability of age at death emerged during this transition.

Secondly, I decompose the differences in variability of age at death between males and females at fixed points in time in order to see how differences in the age pattern of mortality between males and females contribute to the gender gap in  $S_{10}$  at a specific time point. The results of this analysis indicate how differences in age-specific mortality between males and females at a particular point in time contribute to differences in variability of age at death observed between the sexes. I decompose differences in  $S_{10}$  at four different points in time: 1880-84, 1925-29, 1960-64, and 2000-04. The first two time periods fall before females experienced consistently lower variability of age at death in comparison to males. By taking differences between the age-specific contributions to the gender gap in  $S_{10}$  in 1925-29 and 1960-64, I am able to quantify specifically how changes in the relationship between male and female age-specific mortality rates led to the emergence of the gender gap in  $S_{10}$  between these two time periods. I choose to use the period 1925-29 rather than 1920-24 because this is the first period that I have cause-of-death data available for the decompositions of gender differences in variability of age at death that I pursue in Chapter 8.

The decompositions of sex-specific trends in  $S_{10}$  and differences in  $S_{10}$  between males

and females at fixed points in time are carried out using life table data from ten countries in the HMD with sufficiently long historical records: Belgium, Denmark, England and Wales, Finland, France, Italy, Netherlands, Norway, Sweden, and Switzerland. Using life tables with single year age groups and five year periods produces estimates of  $S_{10}$  that are both accurate and stable.

### 7.1.4 Decomposing sex-specific trends in $S_{10}$

In order to understand the emergence of the gender gap in variability of age at death, I have decomposed sex-specific trends in  $S_{10}$  over time into the contribution of changes in age-specific mortality rates. The results of these decompositions indicate what changes in the age profile of mortality are especially important for explaining the more rapid decline in  $S_{10}$  among females in comparison with males around the midpoint of the 20th century. In addition, these results offer insight into how changes in age-specific mortality rates in the most recent period are contributing to relative stability in the  $S_{10}$  measure for both sexes.

Between 1920-24 and 1960-64, male  $S_{10}$  decreased by 3.99 years and female  $S_{10}$  decreased by 5.89 years in France. The individual contributions of changes in age-specific mortality rates to the these changes in sex-specific  $S_{10}$  over this time period are presented graphically in Figure 7.3(a). During this time period, mortality rates were declining at every age as seen in Figure 7.4. Female mortality rates declined at a faster pace than male during this time period (Figure 7.4), and, at younger ages, the decomposition results indicate that this translated into greater age-specific contributions to the decline in  $S_{10}$  for females in comparison to males (Figure 7.3(a)).

Table 6.1 shows the decomposition results for all ten countries over the three time periods aggregated over broader age groups. These results confirm more generally the insights gained from the graphical analysis of these results for France. For all countries included in the decomposition analysis, it is clear that during the period 1920-24 to 1960-64 most of the decline in  $S_{10}$  was due to mortality improvements in the 10-29 age group for both males and females. In Norway, for females, 5.74 years of the total 8.27 year decline in  $S_{10}$  over this period was attributable to mortality declines in the 10-29 year old age group while for males improvements in this same age group contributed 4.56 years to a total decline of 5.96 years. The contributions of improvements in mortality in the 10-29 and 30-49 age groups to the declines in  $S_{10}$  during the 1920-24 to 1960-64 period were always greater for females compared to males across all of the countries examined.

Declines in  $S_{10}$  and the age-pattern of contributions of changes in age-specific mortality to the decline in  $S_{10}$  were more similar for males and females in this first period examined, 1880-84 to 1920-24, in comparison to the second time period, 1920-24 to 1960-64. These decomposition results suggest that differential changes in cause-of-death composition at younger ages around the midpoint of the twentieth century are particularly important in explaining the emergence of gender gap in variability of age at death. The emergence of the gender gap in variability of age at death in France is investigated from a cause-of-death

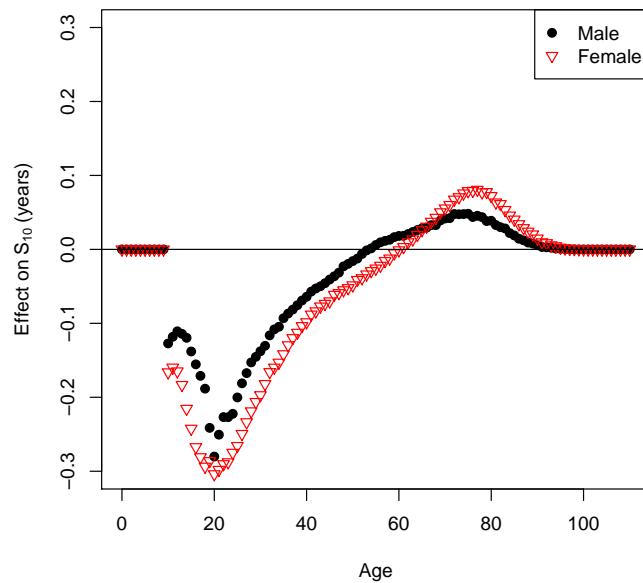
perspective in Chapter 8.

### 7.1.5 Decomposing differences in $S_{10}$ between females and males

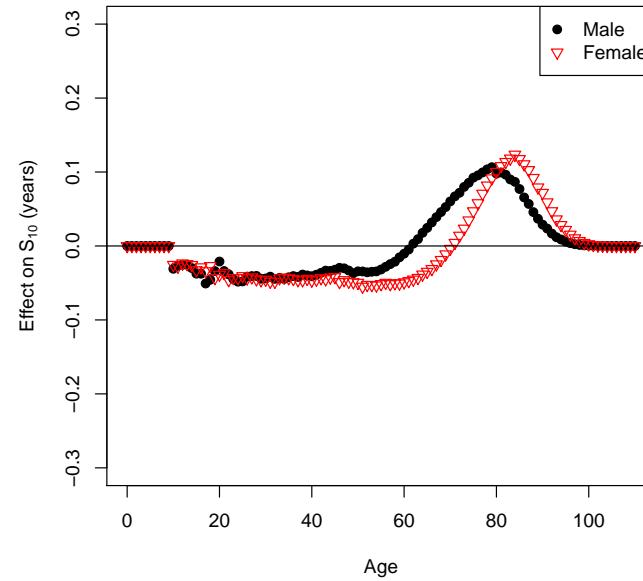
A deeper understanding of the evolution of the gender gap in  $S_{10}$  over time is gained by decomposing the difference in  $S_{10}$  between the sexes at a fixed point in time into the contributions of differences in age-specific mortality rates. Vallin used this technique to analyze the emergence of the gender gap in life expectancy in France between the periods 1925-1929 and 1974-1978 (Vallin, 1993). In these analyses, I decompose differences in  $S_{10}$  between the sexes at four fixed points in time: 1880-84, 1925-29, 1960-64, and 2000-04. I am able to explore how changes in the relationship of male and female age-specific mortality rates influenced the emergence of the gender gap in variability of age at death by taking the differences of age-specific contributions in 1925-29 and 1960-64.

Before delving into the decomposition results, let us consider the changes in the relationship between male and female age-specific mortality rates over time using France as an example. During the 20th century, in France, the ratio of male to female age-specific mortality rates,  $m_x$ , in the younger age groups rose quite steadily as illustrated in Figure 7.5 (a similar figure appears in (Vallin, 1993)). While females have always had a mortality advantage in the older adult ages, recently a bimodal pattern in the ratio of male  $m_x$  to female  $m_x$  has developed as women also gain an advantage in the younger adult ages. Notice in Figure 7.5 that the first mode occurs around age 20, the same age where the peak contributions are made to declining  $S_{10}$  during the period 1920-24 to 1960-64 as shown in Figure 7.3(a). It is likely that the emergence of the female advantage in mortality at young adult ages is particularly important in explaining the emergence of the gender gap in  $S_{10}$ . As the decomposition of sex-specific trends in  $S_{10}$  reveals, improvements in mortality at younger ages act to decrease  $S_{10}$  while improvements at older ages increase  $S_{10}$ .

The decomposition of differences in  $S_{10}$  between females and males at fixed points in time for France is shown in Figure 7.6(a). This figure indicates how differences between female and male mortality rates at each age contribute positively or negatively to the difference in  $S_{10}$  between French females and males. In 1880-84, the difference between female and male  $S_{10}$  was .66 years. Figure 7.6(a) reveals that females experienced slightly higher variability of age death because their mortality advantage at older ages and their slight disadvantage under age 20 resulted in a greater dispersal of ages at death relative to males. In more recent periods, females experienced lower levels of  $S_{10}$  relative to males because female mortality advantage below age 60 decreased their dispersal of ages at death relative to males and this was not compensated by the effect of female mortality advantage at older ages, which increased dispersal. The results of the decomposition of differences in  $S_{10}$  between the sexes for all countries included in the analysis are presented in Table 7.1. The decomposition results for other countries confirm the insights gained from the graphical analysis of the decomposition results for France: from a fixed time perspective, female levels of  $S_{10}$  are currently lower than males as a result of their mortality advantage at younger ages.



(a) 1920-24 to 1960-64



(b) 1960-64 to 2000-04

Figure 7.3: Contributions of changes in age-specific mortality rates to changes in sex-specific  $S_{10}$  over time, France. Data source: Human Mortality Database (HMD, 2009).

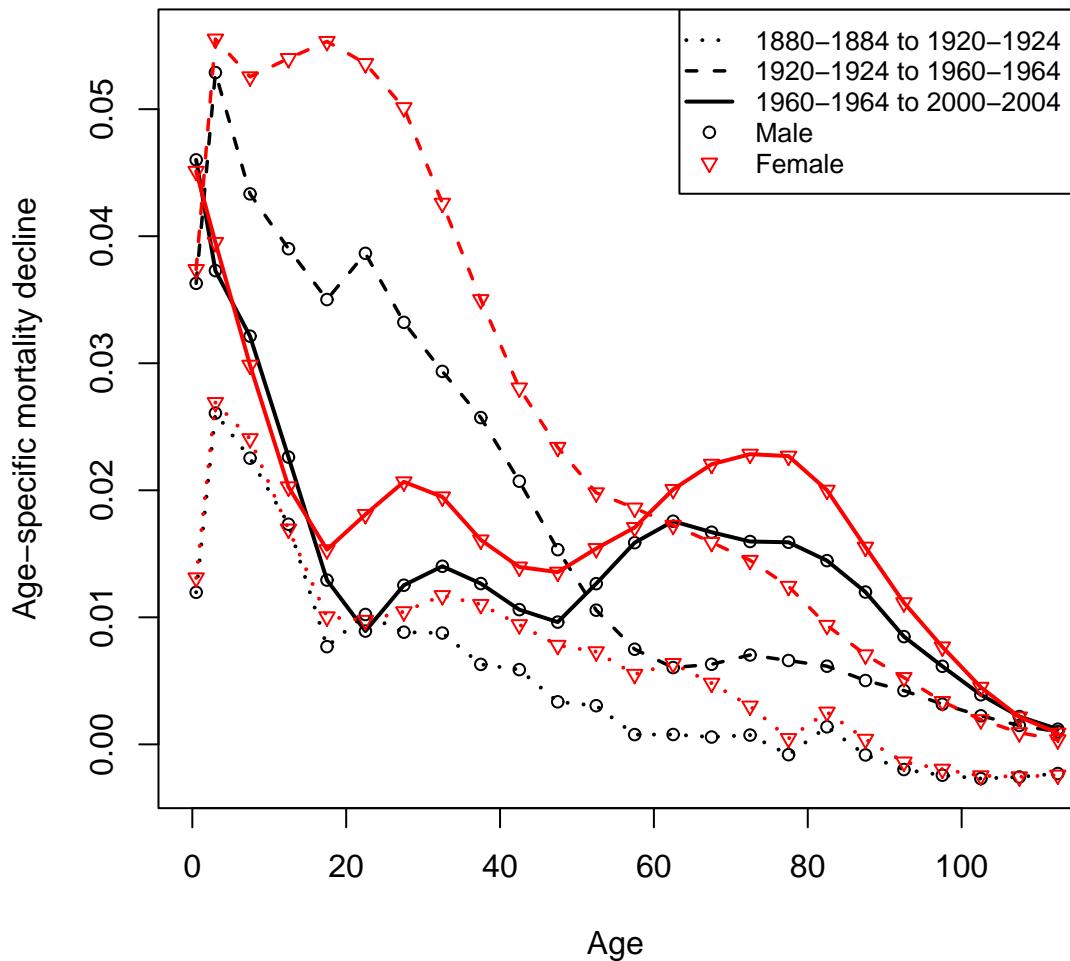


Figure 7.4: Age-specific mortality decline by sex, France. Proportional age-specific mortality decline,  $\phi_x$ , is measured between two time periods using the following equation:  $\phi_x = \frac{\log(m_x(t)/m_x(0))}{t}$ . Data source: Human Mortality Database (HMD, 2009).

Taking the differences between the contributions of age-specific mortality rates to the difference in  $S_{10}$  in 1925-59 and 1960-64 highlights what changes in the relationship between male and female age-specific mortality rates between these two time points were important in the creation of the gender gap in  $S_{10}$ . This is illustrated for France in Figure 7.6(b). These results indicate that females gained an advantage over males in terms of lower variability of age at death because of greater improvements in mortality at younger and older middle adult ages (note the bimodal pattern of age-specific contributions to the change in the gender gap in  $S_{10}$  below roughly age 70). At the oldest ages, increasing female advantage in mortality acted to narrow the gap between female and male  $S_{10}$  between these two time points.

The results of gender gap analysis offer evidence that the lower levels of variability of age at death exhibited by females in comparison to males are solely the result of female advantage in mortality at younger ages. Females now enjoy lower mortality rates at every age in comparison to males, but it is only their mortality advantage at younger ages which contributes to females also experiencing greater certainty in timing of death in comparison to males.

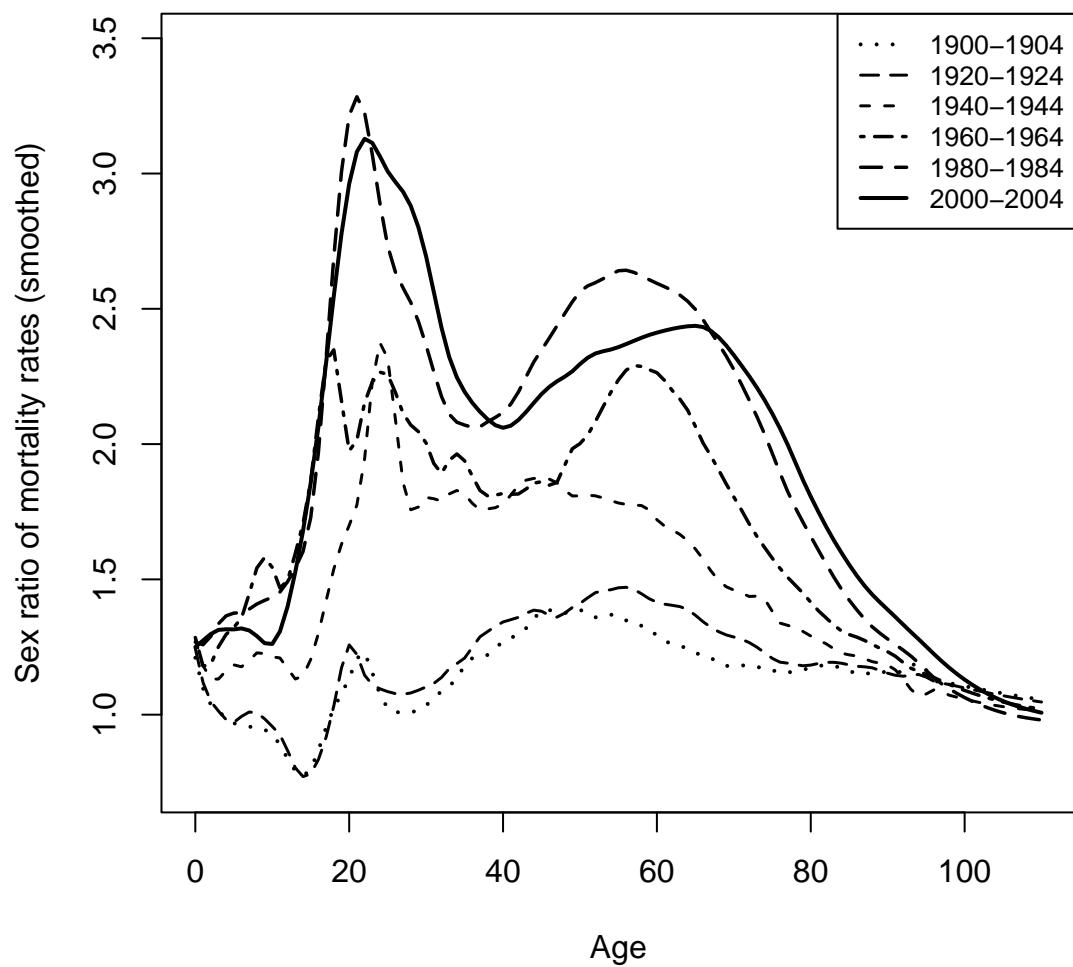
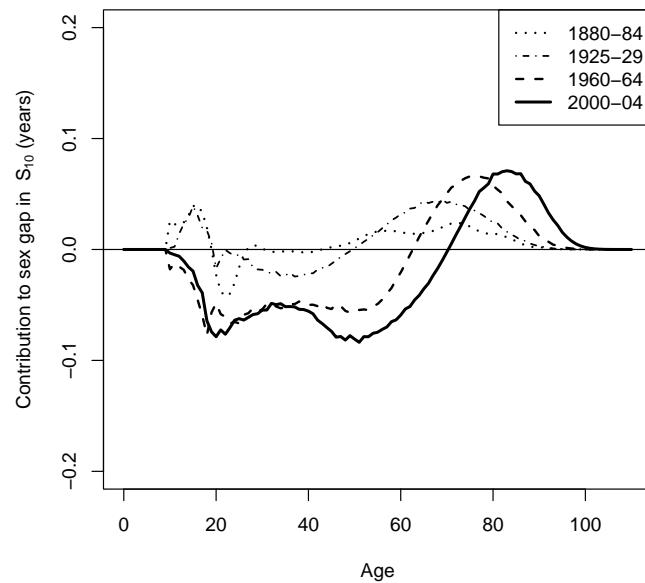
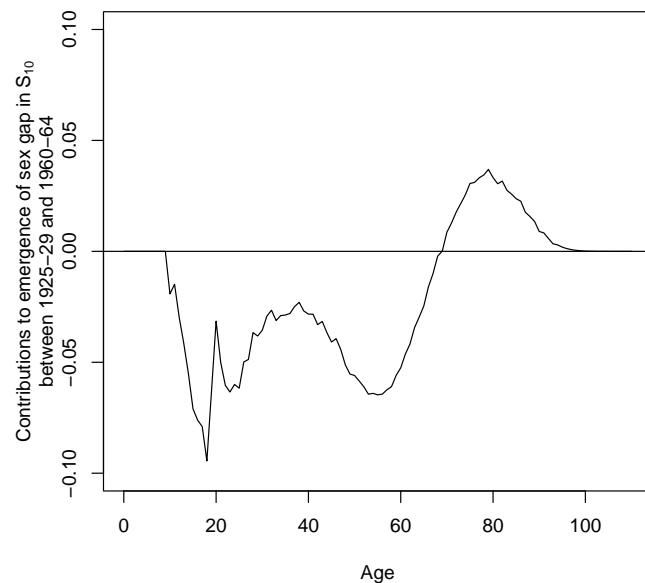


Figure 7.5: Ratio of male  $m_x$  to female  $m_x$ , France. Data source: Human Mortality Database (HMD, 2009).



(a) Contributions of differences in age-specific mortality rates to differences in  $S_{10}$  between females and males at fixed points in time, France.



(b) Contributions of changes in age-specific mortality rates to the emergence of gender gap in  $S_{10}$  between 1925-29 and 1960-64, France.

Figure 7.6: Results of decompositions of differences in  $S_{10}$  between females and males. Data source: Human Mortality Database (HMD, 2009).

Table 7.1: Age-specific contributions to the difference between female and male  $S_{10}$  (in years). A negative sign indicates that gender differences in age-specific mortality in that particular age group contribute to females experiencing lower variability of age at death in comparison with males.

Country	Age group	1880-84	1925-29	1960-64	2000-04
Belgium	10-29	0.1	-0.07	-1.09	-1.01
	30-49	-0.02	-0.19	-0.78	-0.88
	50-69	0.52	0.24	-0.32	-0.78
	70+	0.27	0.45	0.72	1.16
	Total	0.87	0.43	-1.47	-1.51
Denmark	10-29	0.42	0	-0.84	-0.72
	30-49	0.13	0.19	-0.35	-0.77
	50-69	0.45	0.05	-0.35	-0.5
	70+	0.41	0.12	0.5	1.16
	Total	1.4	0.36	-1.04	-0.82
England and Wales	10-29	0.04	-0.2	-0.83	-0.71
	30-49	-0.05	-0.35	-0.49	-0.67
	50-69	0.45	0.29	-0.29	-0.56
	70+	0.32	0.59	1.16	0.98
	Total	0.76	0.32	-0.45	-0.97
Finland	10-29	-0.08	-0.29	-1.18	-1.03
	30-49	0.03	-0.16	-1.34	-1.27
	50-69	0.4	0.9	-0.16	-0.98
	70+	0.29	0.41	0.58	1.04
	Total	0.64	0.86	-2.1	-2.24
France	10-29	0.07	0.09	-0.95	-0.94
	30-49	0	-0.35	-1.02	-1.23
	50-69	0.3	0.54	-0.33	-1.11
	70+	0.29	0.51	1.04	1.19
	Total	0.66	0.79	-1.27	-2.08
Italy	10-29	0.36	0.21	-1.07	-0.96
	30-49	0.13	-0.08	-0.78	-0.81
	50-69	0.09	0.19	-0.32	-0.9
	70+	-0.19	0.2	0.61	1.03

*Continued on next page*

Country	Age group	1880-84	1925-29	1960-64	2000-04
	Total	0.39	0.52	-1.55	-1.65
Netherlands	10-29	0	-0.11	-0.88	-0.54
	30-49	0.16	0.24	-0.54	-0.37
	50-69	0.36	0.04	-0.53	-0.57
	70+	0.18	0.18	0.49	1.16
	Total	0.7	0.35	-1.46	-0.32
Norway	10-29	-0.67	-0.45	-1.22	-1.01
	30-49	0.02	-0.15	-0.82	-0.79
	50-69	0.2	0.15	-0.51	-0.59
	70+	0.31	0.32	0.47	1.06
	Total	-0.14	-0.13	-2.08	-1.33
Sweden	10-29	-0.24	-0.12	-0.91	-0.69
	30-49	-0.08	-0.06	-0.56	-0.6
	50-69	0.32	0.11	-0.35	-0.61
	70+	0.41	0.22	0.52	0.95
	Total	0.41	0.14	-1.3	-0.95
Switzerland	10-29	0.15	-0.06	-1.28	-0.9
	30-49	-0.01	-0.28	-0.83	-0.75
	50-69	0.31	0.3	-0.37	-0.83
	70+	0.03	0.31	0.73	0.9
	Total	0.48	0.27	-1.76	-1.59

## 7.2 Cross-country differences in variability of age at death

In this section, I explore the emergence of the gap in  $S_{10}$  between Sweden and the United States. In the period 1940-44, levels of  $S_{10}$  were similar in the two countries-around 16 years for both males and females. Just as a gender gap in  $S_{10}$  emerged within each country, a gap in  $S_{10}$  also emerged between the two countries with Sweden exhibiting much lower levels of  $S_{10}$  in comparison to the US. In the period 2000-04,  $S_{10}$  for Swedish males and females was 13.0 and 12.1 years respectively; in the US,  $S_{10}$  was 15.6 years for males and 14.1 years for females. In this section, I try to determine whether the patterns of change in age-specific mortality that produced the cross-country differences in  $S_{10}$  between Sweden and the US are similar to the changes that produced the gender gap in  $S_{10}$  within these countries as documented in the last section.

Figure 7.7 shows trends in  $S_{10}$  for both sexes as well as the total population within each country. This graph and the statistics presented above indicate that currently the within country gender gap in  $S_{10}$  is smaller than the between country gap in sex-specific  $S_{10}$ . The larger difference across countries in comparison to the gender gap within countries can

likely be explained by the fact that males and females within a particular country share an epidemiological environment.

In order to better understand the emergence of the gap in  $S_{10}$  between Sweden and the US, I decompose the differences in  $S_{10}$  between Sweden and the US during three periods 1935-39 (before the emergence of the gap), 1960-64 (after the emergence of the gap), and 2000-04 (the most recent period). These decomposition were performed for both sexes separately as well as for the total population. By taking differences of the age-specific contributions to the gap in  $S_{10}$  between the periods 1935-39 to 1960-64, I quantify how changes age-specific mortality led to the emergence of a gap in  $S_{10}$  between Sweden and the US.

The results of the decomposition of differences in  $S_{10}$  between Sweden and the US for each sex and the total population at the three fixed time points are depicted in Figure 7.8. The age-specific contributions to the differences in  $S_{10}$  between Sweden and the US in 1935-39 reveal that the small gap was the result of Sweden experiencing more favorable mortality conditions at some ages while the US experienced more favorable conditions at other ages. In particular, at the youngest ages, it appears that the US has an advantage in mortality which counteracts a gap between Sweden in the US. Between the ages 20 and 40, Sweden has a mortality advantage which increases the gap between Sweden and the US. It's difficult to interpret contributions in the middle of the age distribution because positive or negative contributions depend on the mean age of death for deaths above age 10. At the oldest ages, the US has a mortality advantage, which stretches out the death distribution, thus increasing the gap between the US and Sweden.

In 1935-39, mortality differences between Sweden and the US in the roughly 60-80 age group as well as the 10-20 age group act to close the gap between the US and Sweden. Over time, as can be seen in the decomposition of differences in 1960-64 and 2000-04, Sweden gains an advantage over the US with regards to mortality in the 10-20 age group, and thus mortality differences at these ages in 1960-64 and 2000-04 widen the gap in  $S_{10}$  observed between the US and Sweden at these fixed time points. The contributions of changes in mortality around the ages 60-80 are difficult to interpret because the mean age is changing across time in this age group, and whether improvements in mortality contribute positively or negatively to the gap in  $S_{10}$  depends on the crossover age for each country and how this age changes over time. Throughout all three periods, the mortality advantage for the United States at the oldest ages acts to widen the gap in  $S_{10}$  between Sweden and the United States.

The age-specific contributions to the emergence of the gap in  $S_{10}$  between Sweden and the United States depicted in Figure 7.9 reveal that, similar to the results related to the emergence of the gender gap, contributions around age 20 are especially important for the emergence of the gap in  $S_{10}$  between Sweden and the US during the period 1935-39 to 1960-64. Also similar to the emergence of gender gap, differences in mortality in the age group 30-40 don't seem to be so important. Differences in mortality in the age groups above age 40 all contribute the to emergence of the gap in  $S_{10}$  between Sweden and the US during the period 1935-39 to 1960-64. In contrast, looking at the emergence of the gender gap in France between 1925-29 and 1960-64 in Figure 7.6(b), changes in mortality at the oldest ages act

to close the gender gap in  $S_{10}$  as women continue to experience an advantage at the oldest ages.

Looking at the age-specific contributions to the change in gap in  $S_{10}$  between 1960-64 to 2000-04 also shown in Figure 7.9, we see that the male gap likely grew a little bit during this period with small contributions coming from nearly all age groups. In contrast, changes in mortality in the age groups 70 to 90 for females acts to close the gap while changes at other ages either act to either maintain or slightly widen the gap. The emergence of the gap in  $S_{10}$  between Sweden and the US was largely the result of mortality changes between the periods 1935-39 to 1960-64; however, the gap continues to widen to some extent in the more recent period especially for males.

### 7.3 Is lower variability of age at death advantageous?

A broad overview of the literature concerning the significance of measures of variability of age at death as indicators of certainty in timing of death and life span disparity was presented in Chapter 3. In this section, I suggest how the results of the decomposition analyses presented above can contribute to our understanding of how to interpret differences in variability of age at death. In particular, I address the issue of whether lower variability of age at death is necessarily advantageous. I consider this issue from the vantage point of both an individual and society.

The differing results obtained when decomposing gender differentials in variability of age at death versus cross-country differentials offer a useful starting point for this discussion. In the case of gender differentials in variability of age at death, the decomposition results suggest that lower variability is solely the result of greater reductions in premature mortality for females in comparison to males; however, in the case of cross-country differentials, the lower variability exhibited by Sweden is due to both lower rates of premature mortality and higher rates of mortality at older ages relative to the US. In both cases, the group with lower variability of age at death also experienced higher life expectancy, which suggests a mortality advantage. Additionally, this lower variability of age at death for both groups was partly attributable to lower rates of premature mortality. This seems to be a precondition for considering lower variability of age at death advantageous (i.e. lower variability of age at death would not be advantageous if it was due to deaths being clustered in younger ages-imagine a death distribution transposed around the median age at death).

The lower variability of age at death observed among females in comparison to males seems to be entirely advantageous based on the decomposition results. The female death distribution is both more equitable and indicates higher average life span-both advantages from the societal point of view. At the individual level, females also experience an advantage because they enjoy greater certainty in timing of death while also experiencing mortality advantage at every age in comparison to males.

The question of whether lower variability of age at death is advantageous for the Swedish

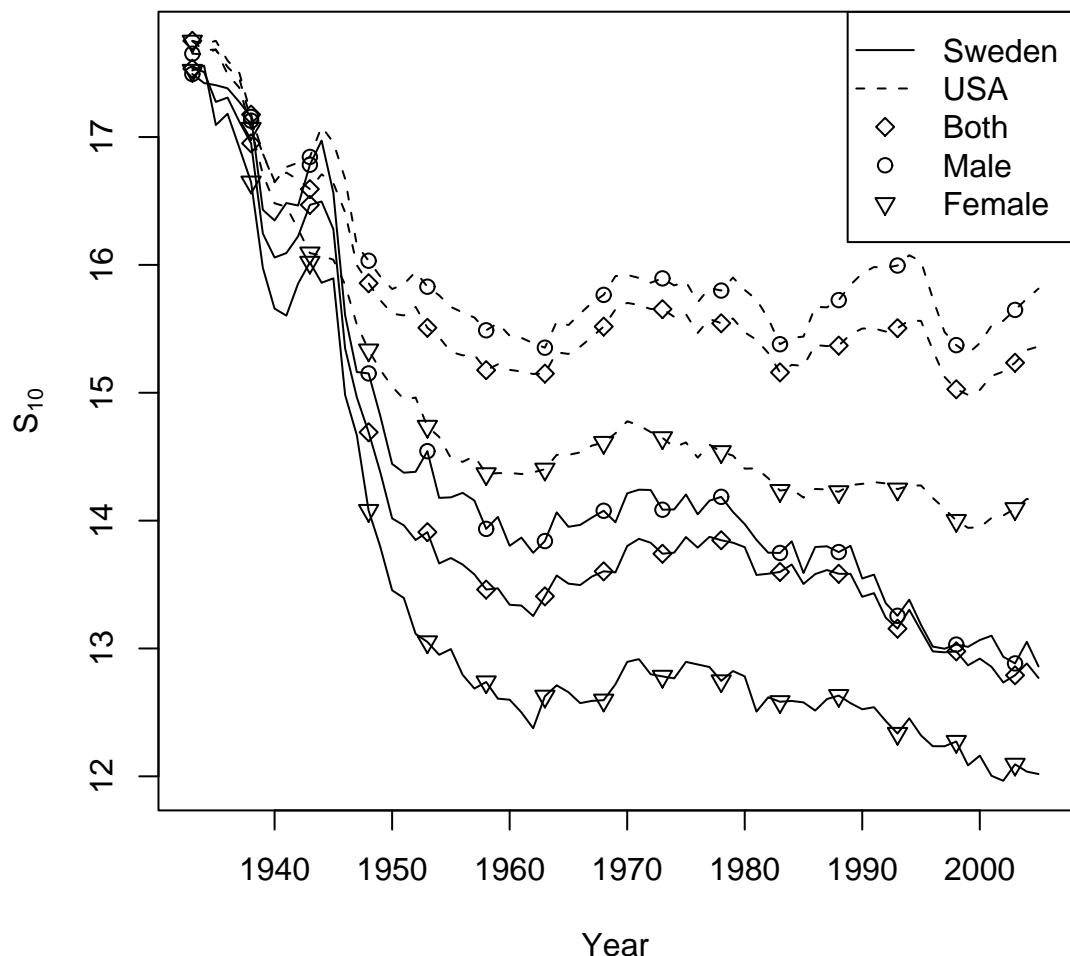
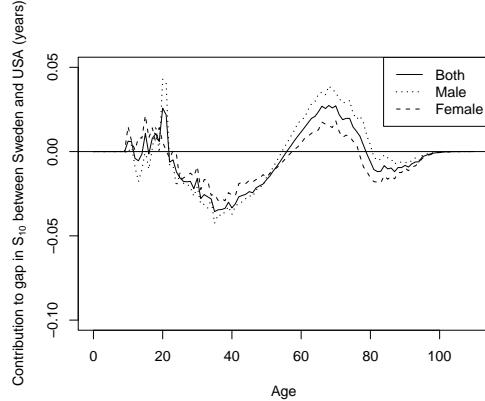
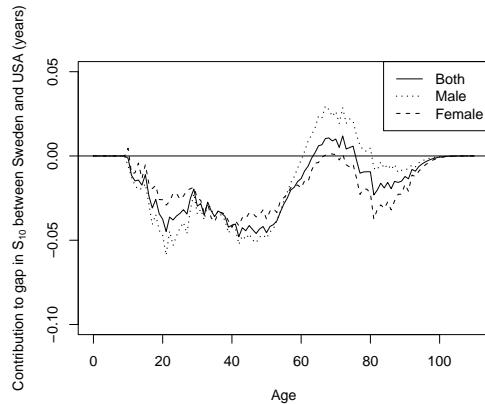


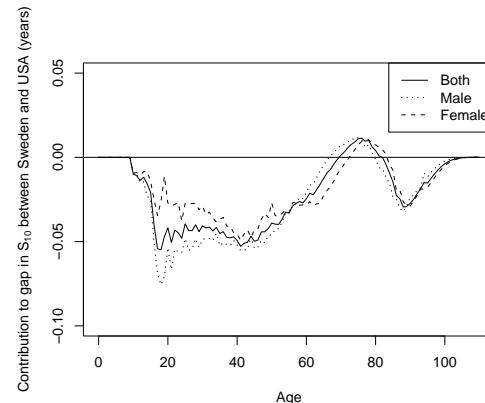
Figure 7.7: Trends in  $S_{10}$  for Sweden and the United States. Data source: Human Mortality Database (HMD, 2009).



(a) 1935-39



(b) 1960-64



(c) 2000-04

Figure 7.8: Age-specific contributions to gap in  $S_{10}$  between Sweden and the United States at fixed points in time. Data source: Human Mortality Database (HMD, 2009).

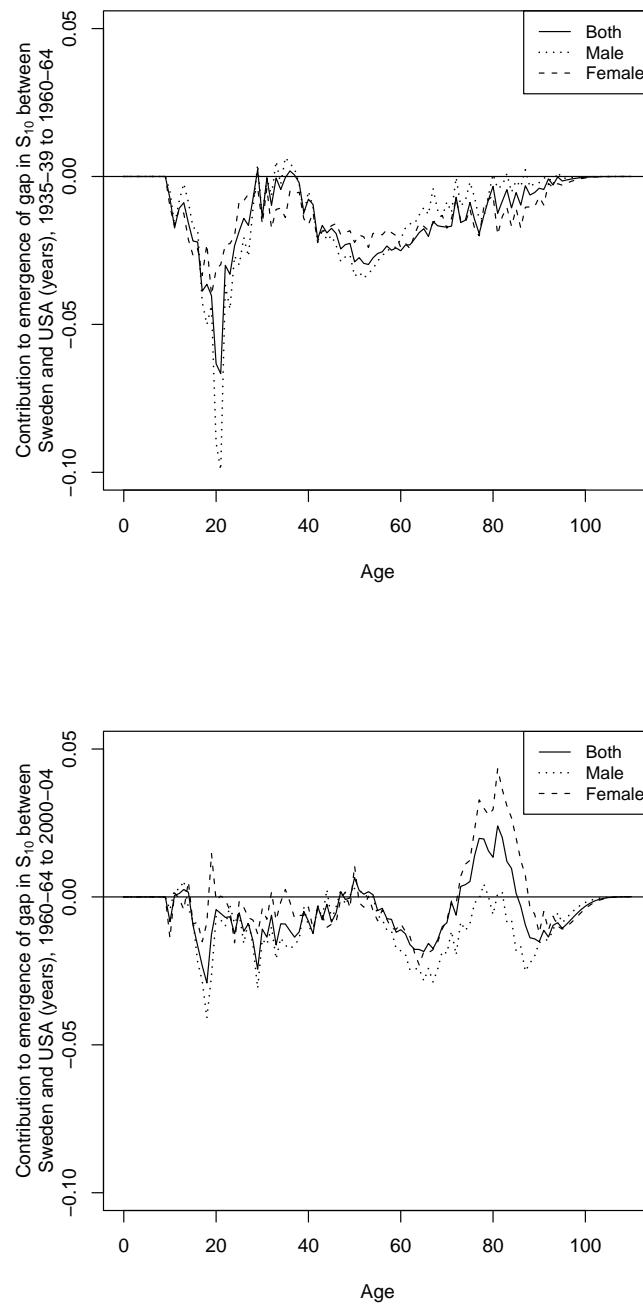


Figure 7.9: Age-specific contributions to changes in the gap in  $S_{10}$  between Sweden and the United States over two periods. Data source: Human Mortality Database (HMD, 2009).

population in comparison to the United States is less clear cut. From the societal vantage point, lower variability of age at death seems advantageous for Sweden because the distribution of ages at death is more equitable and this population also enjoys higher life expectancy. Although theoretical results from the literature suggest that individuals value certainty in timing of death, there has not been much empirical research done assessing individual preferences about mortality risk with regards to certainty (Edwards, 2009a). Individuals may be inclined to risk living in a population with greater variability of age at death like the United States if there is a possibility that they will be able to live longer, especially if they are likely to end up in an advantaged group within the population.

This discussion highlights the importance of taking into account average life time duration when discussing whether a particular distribution of deaths is advantageous. Although the case can be made that there is value in certainty in timing of death and an equitable distribution of deaths regardless of mean life time, it would be hard to describe lower variability coupled with low life expectancy as advantageous in comparison to higher life expectancy with only slightly higher variability. This suggests that relative measures of variability may be most useful when trying to assess whether lower variability is advantageous across a large cross section of countries as suggested by (Smits and Monden, 2009).

## 7.4 Conclusion

Across the countries and geographical areas represented in the Human Mortality Database and the WHO collection of 1,802 life tables, there is a consistent pattern revealing the emergence of a gender gap in variability of age at death during the course of the epidemiological transition with females experiencing lower variability in comparison to males. Decomposition analyses of sex-specific trends in  $S_{10}$  and differences in  $S_{10}$  between males and females at fixed points in time demonstrate that females experienced greater declines in variability of age at death in comparison to males during the era of rapid mortality compression because of greater reductions in premature mortality. In the next chapter, I document the particular changes in the epidemiological environment that produced the emergence of the gender gap in France using age and cause-specific mortality data.

Examining cross-country differences between Sweden and the US and the emergence of a gap in variability between these two countries produced results not entirely similar to the analysis of the emergence of the gender gap. The gap between Sweden and the US is driven not only by Swedish advantage in mortality at younger ages, which decreases Swedish variability relative to the US, but also by US advantage in mortality at older ages, which increases US variability relative to Sweden. In order to determine whether lower variability of age at death in Sweden is advantageous over the higher variability experienced in the US, more research needs to be done assessing individual preference for mortality risk.

## Chapter 8

# Cause-of-death composition and trends in variability of age at death

The recent waning in mortality compression indicated by stable trends in variability of age at death has been depicted as the dawning of new era of mortality change: the shifting mortality era. In earlier chapters of the dissertation, I have examined the transition from mortality compression to shifting mortality in a variety of ways but only considering changes in all-cause mortality. In this chapter, I examine two issues regarding the transition to shifting mortality using a cause-of-death approach. First, how can trends in variability of age at death remain stable even in the face of possible changes in cause-of-death composition? In terms of all-cause mortality, efforts at understanding the development of shifting mortality have focused on non-divergence in the age-pattern of mortality change (Wilmoth and Horiuchi, 1999; Thatcher et al., 2008), which I reviewed in Chapter 5. In this chapter, I take this question a step further by asking not only how changes in age-specific mortality affect variability of age at death but how changes in age and cause-specific mortality can produce shifting mortality. Using age and cause-specific mortality data from France that covers the period 1925-1999, I explore how changes in cause-of-death composition over this period have impacted sex-specific trends in variability of age at death. To my knowledge, this is the first study examining the transition to the shifting mortality era from a cause-of-death perspective.

The second issue regarding shifting mortality that I take up this chapter is explaining from a cause-of-death perspective why trends in variability of age at death for different groups have stabilized at different levels of variability rather than at a similar (possibly biological) level. In the previous chapter, Figure 7.1 shows sex-specific trends in variability of age at death, as measured by the standard deviation of ages at death above age 10,  $S_{10}$ , for France, Russia, Sweden, and the United States. As can be seen in this figure, despite exhibiting relatively stable trends in  $S_{10}$  since 1960, there is a great deal of variation in  $S_{10}$  among the different countries and between the sexes within each country. In the last chapter, I explored the reasons for the emergence of the gender gap in variability of age at death and

cross-country differentials using information on all-cause mortality. In this chapter, I focus exclusively on differences in variability of age at death between males and females in France because there is a high quality series of cause-of-death data available for France that extends back to 1925. Additionally, useful insights about the nature of shifting mortality can be gained by examining the divergence in sex-specific trends in  $S_{10}$  since 1960, with French females displaying a small, yet consistent downward trend in contrast to more flat trends and slight mortality expansion for French males.

Background information from the literature on the epidemiological transition, gender differentials in mortality, current knowledge about what factors produce shifting mortality, and studies of differences in variability of age at death between groups is presented in Chapter 2. Below, I offer a more targeted discussion of changes in the relationship between male and female cause-specific mortality observed over the course of the epidemiological transition. After offering an overview of the methods that I utilize in my analysis, I provide an overview of trends in life expectancy, variability of age at death, and cause-of-death composition in France over the course of the epidemiological transition. Next, I speculate on the likely effect of changes in cause-of-death composition on variability in age at death before presenting the results of two sets of decomposition analyses. Finally, I offer a summary of the main results of my analysis. Importantly, in the case of France, I find that differences in neoplasm (cancer) related mortality help to explain why females have continued to experience consistent declines in variability of age at death over the past half century while trends for males have stabilized and even indicate slight mortality expansion.

## 8.1 Sex differences in mortality over the course of the epidemiological transition

Much of my analysis focuses on how differences in age and cause-specific mortality between French males and females lead to differences in variability of age at death over time. Here, I provide information on what is known more generally about changes in the relationship between male and female mortality over the course of the epidemiological transition. During the 20th century, in France, the ratio of male to female all-cause age-specific mortality rates,  $m_x$ , in the younger age groups rose quite steadily as illustrated in Figure 7.5 (a similar figure appears in (Vallin, 1993)). While females have always had a mortality advantage in the older adult ages, recently a bimodal pattern in the ratio of male  $m_x$  to female  $m_x$  has developed as women also gain an advantage in the younger adult ages.

As the all-cause decomposition results from Chapter 7 suggest, in order to understand how females come to experience lower variability of age at death in comparison to males, it is essential to focus on the significant advantage in mortality females gain in the young adult ages. Given that the age group in which females make greater gains against mortality in comparison with males overlaps with the peak reproductive ages, one immediately considers

the possible contribution of declines in maternal mortality. Indeed, during the period of rapid mortality compression, female mortality rates were declining in part due to improvements in maternal care. Maternal mortality risk decreased due to improvements in treatment for hemorrhages and the discovery of sulfonamides used to treat puerperal infections (Retherford, 1975).

Decreased maternal mortality risk, however, was not the sole factor driving female mortality advantage in the younger adult ages. In a study published in 1961, Enterline examines what particular causes of death were responsible for the major increase in the sex ratio of mortality rates in the 15-24 age group between 1929 and 1958 within the United States. In addition to the decrease in maternal related mortality, Enterline finds that the decline of tuberculosis (which affected young women more than men) and the greater increase for men in motor vehicle accidents were other major factors leading to a female advantage in this age group. Greater decline in rheumatic fever for women in the 15-24 age group compared to men also played a role (Enterline, 1961).

In comparing trends in male and female mortality in France between the periods 1925-1929 and 1974-1978, Vallin also recognizes the importance of the erosion of the female disadvantage with regards to infectious diseases in younger age groups. Vallin attributes this historical female disadvantage to the low status of women. Thus, progress against infectious disease along with the general improvement in younger women's status have allowed females to gain an advantage over males in the younger adult ages (Vallin, 1993). Vallin shows that changes in infectious disease are not particularly important for the emergence of the gender gap in life expectancy between the periods 1925-1929 and 1974-1978. The female advantage in life expectancy is heavily influenced by differences in degenerative diseases and neoplasm mortality (Vallin, 1993).

Prior research has shown that declines in infectious disease, declines in maternal mortality, and a rise in accidents played an important role in females gaining a mortality advantage at younger ages (Retherford, 1975; Enterline, 1961). At older ages, differences in cancer and degenerative disease related mortality explain mortality differences between males and females (Vallin, 1993). In this analysis, I find that the evolution of the gender gap in variability of age at death over time is largely influenced by differences in infectious, external cause, and cancer related mortality between males and females.

## 8.2 Decomposition methods

In order to investigate the impact of changes in cause-of-death composition on sex-specific trends in variability of age at death, I utilize the French Cause-of-Death Series, which is a collection of age and cause-specific death data from France covering the period 1925-1999. These data were compiled by France Meslé and Jacques Vallin and standardized to the 9th revision of the International Classification of Diseases (ICD) (Vallin and Meslé, 1988; Meslé and Vallin, 1996; Vallin and Meslé, ). These authors provided me with the data directly

for this project, but the data is available at <http://www-causfra.ined.fr>. For each time period covered in the analysis, I use the French cause-of-death series to find the proportion of deaths attributable to a particular class of causes for each sex by age, and then apply these to the total age-specific mortality rates from the Human Mortality Database in order to obtain age and cause-specific mortality rates for each sex.<sup>1</sup>

I decompose sex-specific trends in  $S_{10}$  over time and differences in  $S_{10}$  between the sexes at fixed points in time using the HWP decomposition method (Horiuchi et al., 2008). More details on the decomposition method are provided in Chapter 4. The decompositions of sex-specific trends in  $S_{10}$  over time quantify the individual contributions of changes in age and cause-specific mortality rates,  $m_{x,i}$ , to overall changes in sex-specific  $S_{10}$  between two time points. The results for this analysis indicate how changes in age and cause-specific mortality have impacted trends in variability of age at death for each sex separately. I decompose the changes in  $S_{10}$  observed between the periods 1925-29 to 1960-64 and 1960-64 to 1995-99.  $S_{10}$  declines rapidly during the first period from 1925-29 to 1960-64, and the decomposition results for this period suggest what particular changes in age and cause-specific mortality were responsible for the rapid compression observed during this period. Trends in  $S_{10}$  have largely been stable but do indicate slight expansion for French males during the latter period, 1960-64 to 1995-99, even though life expectancy has continued to rise. The decomposition results corresponding to this period will reveal how male  $S_{10}$  has remained stable despite changes in age and cause-specific mortality. The decomposition for females over this period will indicate what changes in age and cause-specific mortality produced continued mortality compression.

In a second set of decomposition analyses, I investigate more closely what specific changes in the relationship between male and female age and cause-specific mortality have led to females gaining an advantage over males with regards to lower variability of age at death. I focus on the differences in  $S_{10}$  between males and females at three time points: 1925-29 (before the emergence of the gap), 1960-64 (after the emergence of the gap), and 1995-99 (the most recent period for which data is available). By taking differences between the age-specific contributions to the gender gap in  $S_{10}$  in 1925-29 and 1960-64 as well as 1960-64 and 1995-99, I am able to quantify specifically how changes in the relationship between male and female age and cause-specific mortality rates led to the emergence of the gender gap in  $S_{10}$  in the first period and a continued widening of the gap in the second period. Below, I describe the cause-of-death classification scheme that I adopt to carry out my analysis.

### 8.2.1 Cause-of-death classification scheme

Choosing an appropriate classification scheme is a difficult task. In his analysis of the emergence of the gender gap in life expectancy in France between 1925-1929 and 1974-1978,

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<sup>1</sup>The mortality rate for age  $x$  due to cause  $i$  can be found by multiplying the total mortality rate for age  $x$  by the proportion of deaths at age  $x$  due to cause  $i$  observed in the population,  $m_x, i = m_x * p_x, i$

Vallin used a classification scheme with seven categories: parasitic and infectious disease, malnutrition and diseases of the digestive system, accidents and homicides, neoplasm, hereditary and congenital diseases, degenerative diseases (including functional disease), and suicide (Vallin, 1993). His classification system has the advantage of being aetiologically based unlike the major chapters of the ICD which mix aetiological factors with anatomical factors and certain conditions such as maternal related mortality. In comparison with the ICD classification system, an aetiological classification system better captures the epidemiological transition because infectious diseases are only included in one category rather than being mixed in categories with non-infectious causes of death (e.g. the respiratory category of the ICD system includes infectious diseases such as influenza and pneumonia as well as chronic diseases such as asthma and chronic obstructive pulmonary disease (COPD)) (Meslé, 1999).

In addition to providing greater consistency when observing trends in causes of death over the period of analysis (1925-1999), adopting an aetiological classification system similar to Vallin's would have allowed me to directly compare my results for the emergence of the gender gap in variability of age at death with Vallin's results for the emergence of the gender gap in life expectancy (Vallin, 1993; Meslé, 1999). Unfortunately, translating causes of death that have been categorized according to the ICD system into aetiological groups is a tremendous undertaking necessitating the involvement of medical experts (Meslé and Vallin, 1981; Meslé, 1999). Additionally, by choosing a classification system mainly based on the ICD chapters, I am able to quantify the effect that changes in maternal mortality (a particularly interesting class of causes) have on the emergence of the gender gap in  $S_{10}$ .

The categories of causes of death with corresponding ICD codes (9th revision) are presented in Table 8.1. The cause-of-death decompositions are carried out using the following classes of causes: infectious diseases, respiratory diseases, digestive diseases, neoplasm, cerebrovascular diseases and unspecified disorders of the circulatory system, heart diseases, external causes, maternal causes, and mental disorders. The remaining causes are grouped into the "other" category. Additionally, there is a category corresponding to deaths that have been attributed to ill-defined or unknown causes. There is an especially large number of these deaths in the first period examined, 1925-29. In their article describing the construction of the French cause-of-death series, Meslé and Vallin cautioned that the data relating to periods prior to 1950 can not be linked as easily with the post-1950 data as the transitions between the 5th to 6th and 5th to 7th revisions of the ICD were extremely complex (Meslé and Vallin, 1996).

It is particularly difficult to classify diseases included in the ICD chapter respiratory diseases. Trends in this category are particularly hard to interpret because infectious respiratory diseases dominate in the early part of the century while chronic lower respiratory diseases dominate in the latter (Meslé, 1999). For my classification scheme, I include infectious respiratory diseases in the respiratory category but run a sensitivity analysis with these deaths transferred into the infectious category in order to see how this impacts the results of the analysis.

Table 8.1: Cause-of-death classification scheme with ICD-9 codes

Class of causes	ICD-9
Infectious diseases	001-139
Respiratory diseases	460-519
Digestive diseases	520-579
Neoplasm	140-239
Cerebrovascular diseases and unspecified disorders of the circulatory system	430-438, 451-459
Heart diseases	390-429, 440-449
External causes	800-999
Maternal causes	630-676
Mental disorders	290-319
Other causes	240-289, 320-389, 580-629, 680-779
Ill-defined and unknown causes	780-799

### 8.3 Trends in life expectancy, variability of age at death, and cause-of-death composition over the course of the epidemiological transition in France

In this section, I document trends in life expectancy, variability of age at death, and cause-of-death composition in France over the course of the epidemiological transition. Along with Denmark, Sweden, and England and Wales, France was a leading country in terms of timing of the health transition with the transition beginning in the 1790s (Riley, 2005). As the data from the HMD reveals, France has experienced sustained increases in life expectancy since 1816 (see Figure 8.1(a)). In contrast, trends in variability of age at death, as indicated by  $S_{10}$ , are not as consistent with a period of slow decline during the 19th century followed by a period of rapid decline in the first half of the 20th century (see Figure 8.1(b)). In recent decades, trends for males and females have diverged with females continuing to experience a sustained moderate decline in variability of age at death while males have experienced more stable trends but overall a slight increase between 1960-64 to 1995-99.

I am interested in understanding how variability of age at death changes in response to changes in cause-of-death composition over the course of the epidemiological transition. While the French Cause-of-Death series that I utilize does not cover the entire transition, beginning only in 1925, this data series still captures the essential elements of the transition. Using this data series in an analysis of trends in cause-specific mortality in France from 1925-1993, with causes classified according to an aetiological classification system, Meslé observes first a decline in the proportion of deaths due to infectious causes and a rise in the proportion of death due to degenerative diseases and tumor related illnesses during the

period 1925-1960. In the following decades, the proportion of deaths due to degenerative diseases declines (Meslé, 1999).

Just examining changes in the overall shape of the male and female death distributions in Figure 8.2, one notices that for both sexes the life table distribution of ages at death is compressed substantially between 1925-29 and 1960-64 and seems to shift to older ages between 1960-64 and 1995-99 (note that the female distribution also narrows). Figures 8.3 and 8.4 display the life table death distributions for the years 1925-29, 1960-64, and 1995-99 broken down by the proportion of deaths by age and cause observed in the population during these periods. In terms of cause-of-death composition, the most dramatic change visible is the virtual elimination of mortality due to infectious disease in infancy, childhood, and young adulthood between the first and second period. In contrast to the changes observed between the first two periods, cause-of-death composition seems relatively similar in the second two periods (1960-64 and 1995-99). Also of interest to note here is that while the composition of most of the chronic diseases is balanced around the mode in the most recent period the proportion of deaths attributable to neoplasm is heavily skewed towards younger adult ages. This depiction of the age distribution of deaths by cause suggests that cancer related mortality has now replaced infectious disease as the major source of premature mortality and thus trends in cancer related mortality will likely be the most significant factor influencing future trends in variability of death. This will become even more apparent in the results of the decomposition analyses presented in Section 8.5.

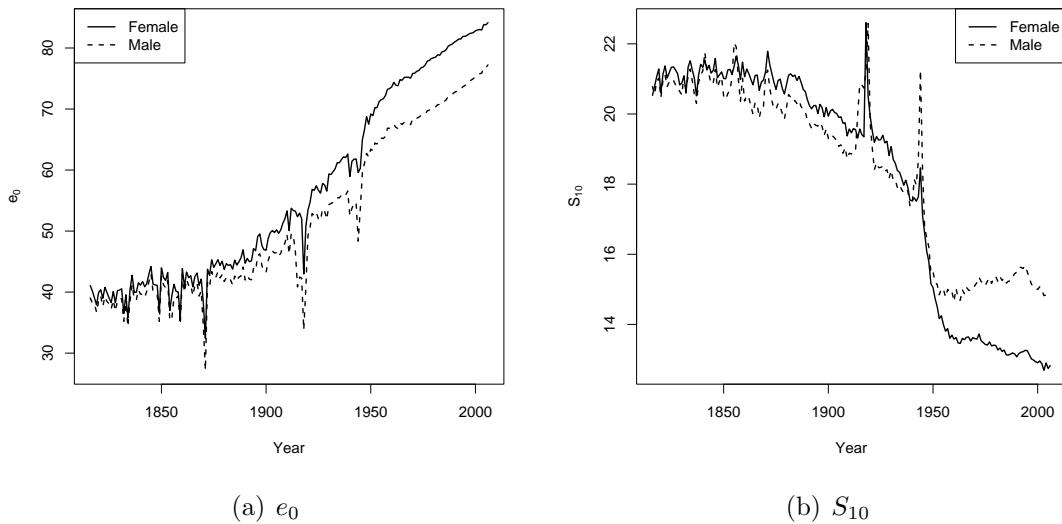


Figure 8.1: Sex-specific trends in life expectancy and variability of age at death, France. Data source: Human Mortality Database (HMD, 2009).

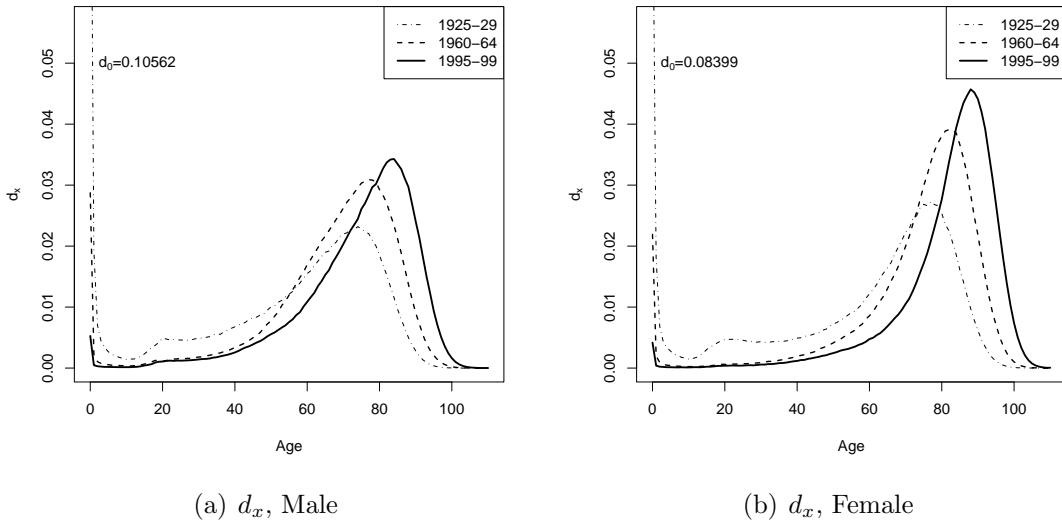


Figure 8.2: Sex-specific trends the distribution of life tables deaths, France. Data source: Human Mortality Database, (HMD, 2009).

## 8.4 Expected effects of changes in cause-of-death composition on variability in age at death

When one considers the broad picture of the relationship between the epidemiological transition and trends in mortality compression, it is clear that progress against infectious disease led to considerable compression in ages at death while more recent progress against chronic diseases has not had the same effect on trends in variability. In this section, I want to offer a description of why one would expect these relationships to hold from a demographic perspective.

First, it is important to realize that unlike life expectancy, for which improvements in mortality at any age lead to an increase in the measure, mortality improvements have different effects on variability of age at death depending on the age at which improvements occur. As Zhang and Vaupel document formally for  $e^t$ , in most cases, there is a crossover age before which improvements in mortality decrease variability and after which improvements in mortality increase variability (Zhang and Vaupel, 2009). At the beginning of the epidemiological transition, when a large proportion of deaths were concentrated in infancy, childhood, and early adulthood, improvements in mortality at these ages had substantial potential to decrease variability of age at death. Thus, during the second stage of the epidemiological transition, *The Age of Receding Pandemics*, rapid mortality compression is observed as significant progress is made against death due to infectious disease, especially at younger

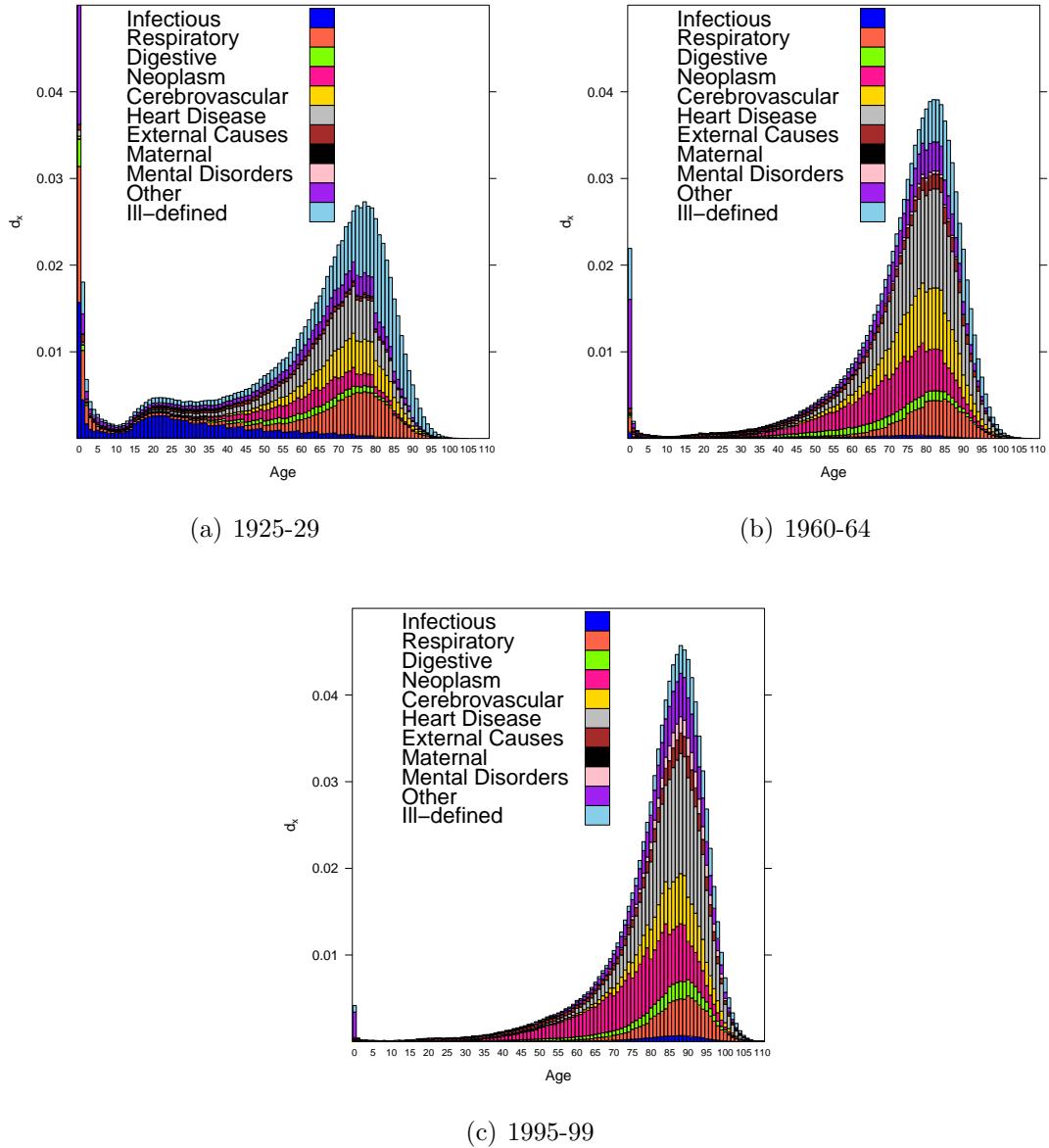


Figure 8.3: Period life table death distributions for French females broken down by cause-of-death. Data sources: Human Mortality Database, French Cause-of-Death Series (HMD, 2009; Vallin and Meslé, ).

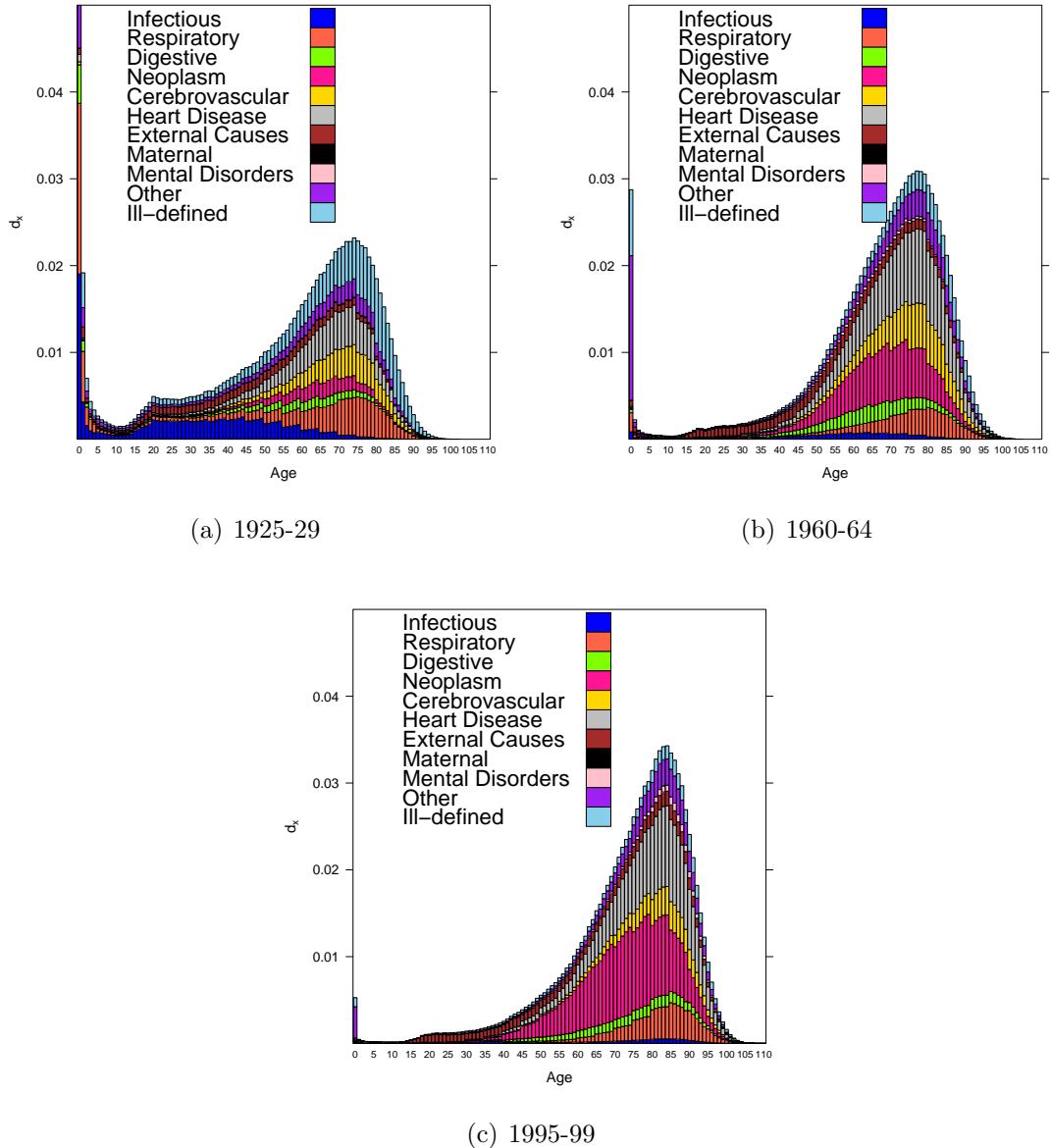


Figure 8.4: Period life table death distributions for French males broken down by cause-of-death. Data sources: Human Mortality Database, French Cause-of-Death Series (HMD, 2009; Vallin and Meslé, ).

ages.

As indicated by the results of the sensitivity analysis presented in Chapter 5, under the mortality conditions observed in more developed countries today, improvements in mortality in young adulthood do not have as much potential to affect variability of age at death as measured by  $S_{10}$  because the left-hand tail of the death distribution is already relatively flat (see Figure 5.6). In more developed countries, the majority of deaths are due to chronic and degenerative disease, which are concentrated in the bulge in the death distribution at older ages. As long as mortality due to these causes is largely balanced on either side of the crossover age, improvements in mortality due to these causes benefit individuals on both sides of the crossover age (non-divergence in the age pattern of mortality change), and these improvements are not constrained by a biological maximum limit to life span, improvements in mortality due to these causes should lead to a shift in the death distribution rather than continued compression. Hence, Robine classifies the third stage of the epidemiological transition as the *The Age of the Conquest of the Extent of Life*, where changes in causes of death that increase life expectancy without necessarily compressing the death distribution dominate. The decomposition results presented in the next section offer a slightly different picture of how changes in cause-of-death composition have led to shifting conditions for French males.

## 8.5 Decomposition results

### 8.5.1 Decomposing changes over time in sex-specific trends in $S_{10}$

Figure 8.5 displays the results of the decompositions of changes in sex-specific  $S_{10}$  between the periods 1925-29 to 1960-64 and 1960-64 to 1995-99. These graphical representations depict the contributions of changes in age and cause-specific mortality between the two time points to changes in  $S_{10}$  over the same period. Corresponding tabular results are presented in Table 8.2

The left hand panels of Figure 8.5 depict the results of decompositions corresponding to the period 1925-29 to 1960-64 for males and females separately. Between 1925-29 and 1960-64, male  $S_{10}$  declined by 3.6 years and female  $S_{10}$  declined by 5.6 years. Decomposition results for this time period for males are shown in Figure 8.5(a). In this figure, bars below the zero line indicate particular age-cause profiles which contribute negatively to the change in  $S_{10}$  over the period 1925-29 to 1960-64. Consistent with epidemiological transition theory, the decomposition results indicate that most of the 3.6 year decline observed for males was due to declines in infectious disease mortality below the crossover age (as represented by the blue bars). Similarly, for females, a substantial portion of the 5.6 year decline in  $S_{10}$  over the period 1925-29 to 1960-64 is due to declines in infectious disease mortality below the crossover age as shown in Figure 8.5(c). As will be discussed in more depth in Section 8.5.2, Figure 8.5 illustrates that  $S_{10}$  declined more rapidly for females largely because the decline in infectious disease contributed more to declines in  $S_{10}$  for females in comparison to

males. The background literature described in Section 8.1 suggests that this is likely due to historical female disadvantage in deaths due to infectious disease and not necessarily more rapid progress against infectious disease for females in comparison to males.

For both males and females, increases in mortality due to a particular cause above the crossover age help to produce the decline in  $S_{10}$  observed during this period (although these contributions are more than offset by declines in mortality above the crossover age due to other causes). The decomposition results presented in Figures 8.5(a) and 8.5(c) suggest that neoplasm related mortality increased above the crossover age thus contributing to the decline in  $S_{10}$  observed for both sexes; however, this result is likely observed because deaths due to neoplasm were disproportionately misclassified into the ill-defined category in the period 1925-29 (notice that declines in mortality due to ill-defined causes above the crossover age make a large positive contribution to changes in  $S_{10}$  over this period).

In the right hand panel of Figure 8.5, decomposition results are presented corresponding to the change in  $S_{10}$  observed between 1960-64 to 1995-99. During this period, trends in  $S_{10}$  for French males and females diverged with male  $S_{10}$  increasing by .4 years and female  $S_{10}$  decreasing by .5 years thus increasing the overall sex gap by almost a year. In contrast to the important role of infectious disease to changes in variability of age at death between 1925-29 to 1960-64, declines in infectious disease do not contribute much to changes in  $S_{10}$  between 1960-64 to 1995-99 as by this point France has entered the third stage of the epidemiological transition. Has this movement into the next stage of the epidemiological transition resulted in a transition from mortality compression to shifting morality? For males, this appears to be the trend, but the decomposition results presented in Figure 8.5(b) suggest that this shifting trend is not the result of the contributions of improvements in chronic and degenerative diseases balancing each other out on either side of the crossover age as suggested in Section 8.4. Rather, declines in male mortality at younger ages mainly attributable to external causes, heart disease, and digestive orders, which contribute to declines in  $S_{10}$ , were matched and exceeded by the contributions of declines in cerebrovascular and heart disease mortality at older ages. In contrast to male trends, Figure 8.5(d) reveals that continued, yet subdued, mortality compression observed among females during the period 1960-64 to 1995-99 can be attributed to continued improvements in mortality due to neoplasm, digestive diseases, and other causes at younger ages. The importance of changes in cancer related mortality in explaining the widening of the sex gap in variability in age at death will become even more apparent in the next section.

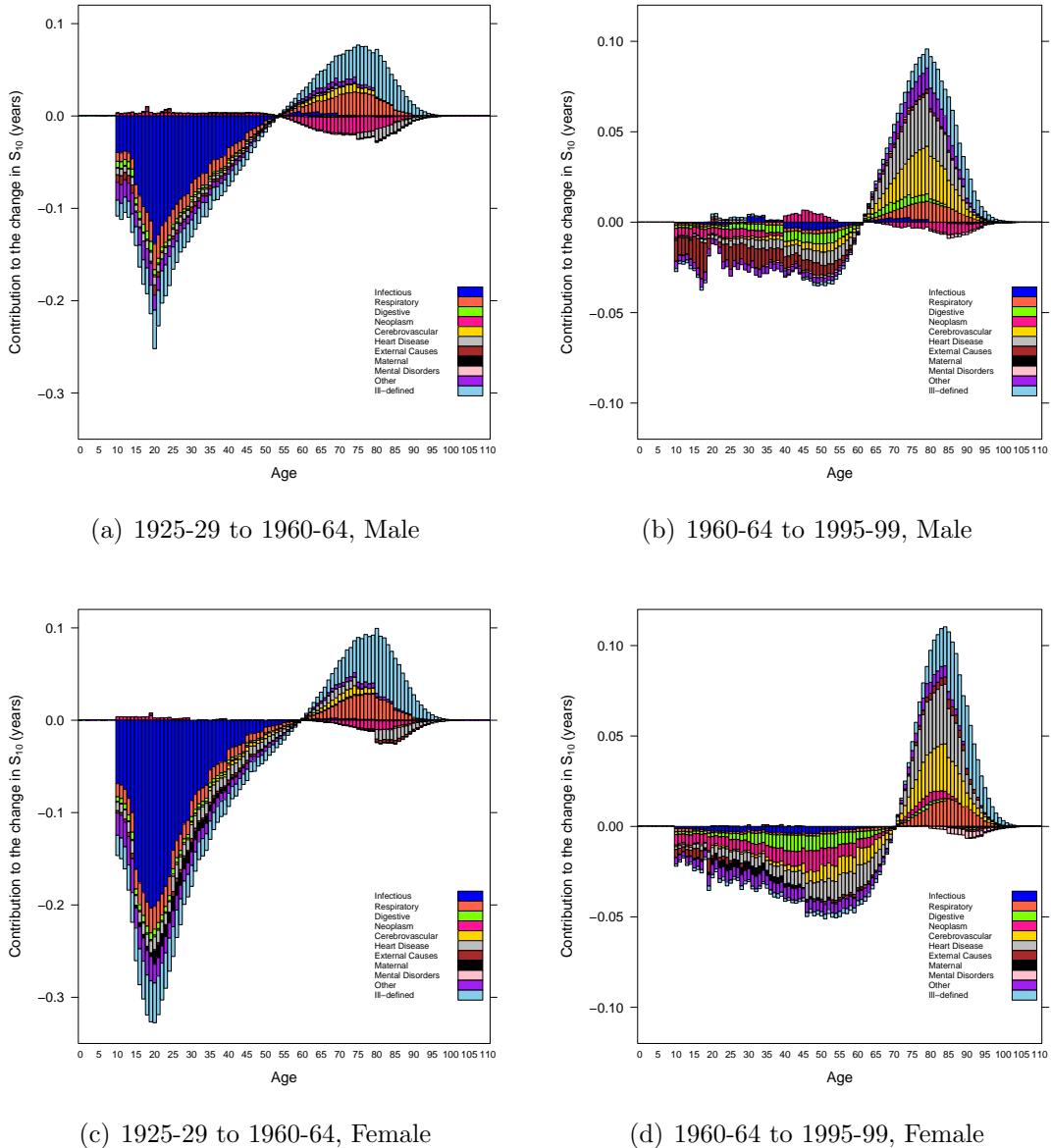


Figure 8.5: Contributions of changes in age-and cause-specific mortality rates to changes in  $S_{10}$  over time, France. Data sources: Human Mortality Database, French Cause-of-Death Series (HMD, 2009; Vallin and Meslé, ).

Table 8.2: Decomposition of change in sex-specific  $S_{10}$  over time by age and cause, France. A negative sign indicates that changes in that particular age and cause-specific mortality rate between periods reduce variability of age at death.

	10-29	30-49	50-69	70+	Total
<b>1925-29 to 1960-64, Female</b>					
Infectious diseases	-2.87	-0.84	-0.02	0.02	-3.72
Respiratory diseases	-0.41	-0.23	0.03	0.40	-0.21
Digestive diseases	-0.12	-0.06	0.00	0.00	-0.18
Neoplasm	0.06	0.00	-0.01	-0.15	-0.10
Cerebrovascular diseases	-0.03	-0.06	0.01	0.06	-0.02
Heart diseases	-0.25	-0.22	0.01	-0.09	-0.54
External causes	-0.04	-0.05	0.00	-0.05	-0.14
Maternal causes	-0.14	-0.07	0.00	0.00	-0.22
Mental disorders	-0.01	0.00	0.00	-0.01	-0.02
Other	-0.43	-0.18	0.00	0.07	-0.54
Ill-defined and unknown causes	-0.67	-0.30	0.03	1.01	0.07
Total	-4.90	-2.02	0.04	1.26	-5.62
<b>1960-64 to 1995-99, Female</b>					
Infectious diseases	-0.04	-0.07	-0.03	-0.01	-0.14
Respiratory diseases	-0.02	-0.02	-0.03	0.22	0.14
Digestive diseases	-0.04	-0.15	-0.10	0.01	-0.28
Neoplasm	-0.11	-0.16	-0.12	0.05	-0.33
Cerebrovascular diseases	-0.02	-0.06	-0.16	0.35	0.11
Heart diseases	-0.07	-0.14	-0.20	0.44	0.03
External causes	-0.06	0.00	-0.02	0.07	-0.01
Maternal causes	-0.04	-0.04	0.00	0.00	-0.08
Mental disorders	0.00	-0.01	-0.01	-0.04	-0.06
Other	-0.10	-0.12	-0.09	0.08	-0.23
Ill-defined and unknown causes	-0.03	-0.04	-0.04	0.46	0.35

*Continued on next page*

	10-29	30-49	50-69	70+	Total
Total	-0.53	-0.80	-0.79	1.64	-0.49
<b>1925-29 to 1960-64, Male</b>					
Infectious diseases	-1.75	-0.78	0.04	0.00	-2.49
Respiratory diseases	-0.34	-0.21	0.14	0.34	-0.08
Digestive diseases	-0.12	-0.04	-0.01	-0.02	-0.18
Neoplasm	0.07	0.05	-0.15	-0.25	-0.28
Cerebrovascular diseases	-0.03	-0.04	0.06	0.06	0.06
Heart diseases	-0.15	-0.07	0.02	-0.13	-0.32
External causes	-0.06	-0.05	0.01	-0.01	-0.10
Maternal causes	0.00	0.00	0.00	0.00	0.00
Mental disorders	0.00	0.02	-0.01	-0.01	-0.01
Other	-0.31	-0.10	0.05	0.04	-0.32
Ill-defined and unknown causes	-0.51	-0.24	0.18	0.72	0.15
Total	-3.18	-1.46	0.32	0.76	-3.57
<b>1960-64 to 1995-99, Male</b>					
Infectious diseases	-0.02	-0.02	-0.02	0.01	-0.04
Respiratory diseases	-0.02	-0.03	0.00	0.16	0.11
Digestive diseases	-0.04	-0.09	-0.02	0.05	-0.10
Neoplasm	-0.09	0.02	0.01	-0.10	-0.16
Cerebrovascular diseases	-0.01	-0.05	0.00	0.39	0.33
Heart diseases	-0.04	-0.11	0.00	0.42	0.27
External causes	-0.19	-0.15	-0.03	0.03	-0.34
Maternal causes	0.00	0.00	0.00	0.00	0.00
Mental disorders	0.01	-0.02	-0.01	-0.01	-0.02
Other	-0.07	-0.07	0.00	0.18	0.05
Ill-defined and unknown causes	0.00	0.00	0.00	0.31	0.30
Total	-0.46	-0.51	-0.07	1.44	0.40

### 8.5.2 Decomposing differences in $S_{10}$ between the sexes

In this section, I decompose differences in  $S_{10}$  between females and males at fixed points in time into the contributions of differences in age and cause-specific mortality between the sexes. My main goal is understanding what changes in cause-of-death composition have produced the recent gap in  $S_{10}$  that has developed as countries have transitioned from the second to third stage of the epidemiological transition. The results of decompositions of differences in  $S_{10}$  between French males and females in 1925-29, 1960-64, and 1995-99 are presented in Figure 8.6 and Table 8.3.

During the first period, 1925-29, shown in Figure 8.6(a), female  $S_{10}$  was higher than male  $S_{10}$  by .79 years. In Figure 8.6, the bars above the zero line indicate particular age-cause profiles which contribute positively to the difference between female and male  $S_{10}$ . Thus,

female disadvantage in infectious disease and maternal mortality at younger ages and female advantage in mortality for all causes at older ages led to a more disperse death distribution for females relative to males in 1925-29. The results of decompositions of differences in  $S_{10}$  between males and females in the periods 1960-64 and 1995-99 can be seen in Figures 8.6(b) and 8.6(c). The results for these two periods exhibit similar patterns of age and cause-specific contributions in contrast to the 1925-29 period. From a fixed time perspective, lower female  $S_{10}$  seems to have been primarily driven by female advantage in external related mortality at younger ages. In the 1995-99 period, the effect of female mortality advantage in neoplasm and heart disease in the middle adult ages on the sex gap in  $S_{10}$  becomes more pronounced.

As seen in Figure 8.7, which shows the age and cause-specific contributions to changes in the sex gap in  $S_{10}$  between 1925-20 to 1960-64 and 1960-64 to 1995-99, the emergence of a significant female advantage in variability of age at death between 1925-29 to 1960-64 was due to females gaining a greater advantage in mortality in both the young adult and the later middle adult ages. As expected, the elimination of infectious disease played an important role in the emergence of the gender gap in  $S_{10}$  between the first two periods. While the decline in maternal related mortality did not make a large contribution to the emergence of the gender gap, an increase in female advantage in external related causes played a major role in the emergence of the gap. Increasing female advantage in heart disease and neoplasms, the major explanatory factors in the in the emergence of the gender gap in  $e_0$ , are not as important for trends in the gender gap in  $S_{10}$  as female advantage in these causes at younger ages acts to decrease  $S_{10}$  while advantage at older ages increases  $S_{10}$ ; however, Figure 8.7(b) suggests that the gap in  $S_{10}$  between males and females grew larger between the periods 1960-64 to 1995-99 primarily due to changes in the relationship between male and female neoplasm related mortality in the middle adult ages.

As mentioned in Section 8.2.1, I reran the decomposition analysis transferring the following infectious diseases from the respiratory disease category to the infectious category: influenza, pneumonia, and meningitis. Transferring these deaths did not substantially alter the basic conclusions from this decomposition analysis about the contributions of changes in the relationship between male and female infectious and respiratory disease mortality on changes in the sex gap in  $S_{10}$  over time. During the period 1925-29 to 1960-64, the difference between female and male  $S_{10}$  changed from 0.79 years to -1.27 years. With infectious respiratory diseases classified in the respiratory category, the contribution of changes in infectious disease mortality to this change of 2.06 years is .39 years and the effect of changes in respiratory mortality is .08 years. When infectious respiratory diseases are classified in the infectious category, the contribution of infectious disease mortality to the change is .39 and the effect of respiratory diseases is .07 (results not shown). The basic finding that infectious disease mortality is an important contributor to the emergence of the gender gap in variability of age at death is not reliant on classifying the infectious respiratory diseases in the infectious category.

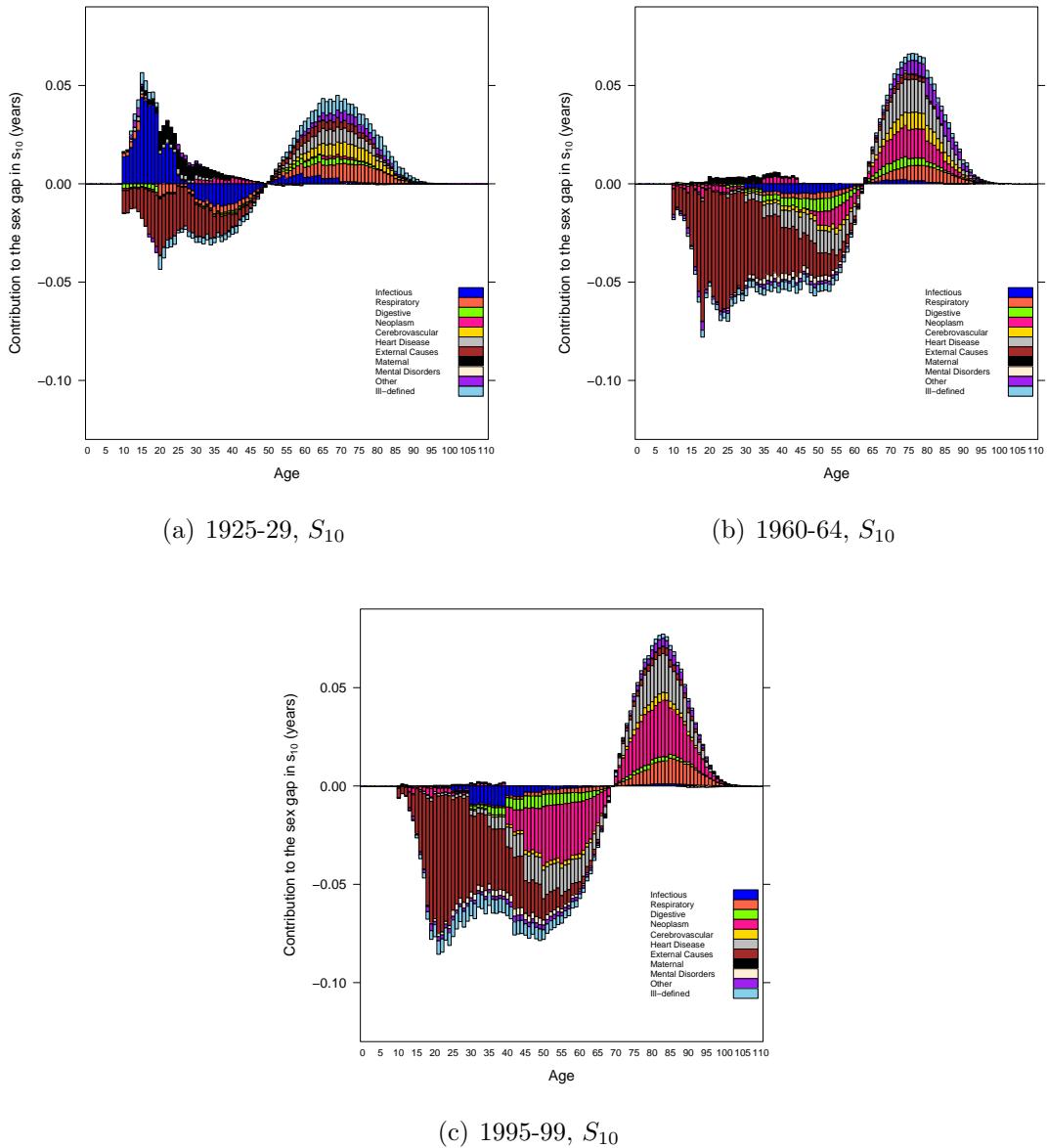


Figure 8.6: Contributions of differences in age-and cause-specific mortality rates to the difference in  $S_{10}$  between females and males at fixed points in time, France. Data sources: Human Mortality Database, French Cause-of-Death Series (HMD, 2009; Vallin and Meslé, ).

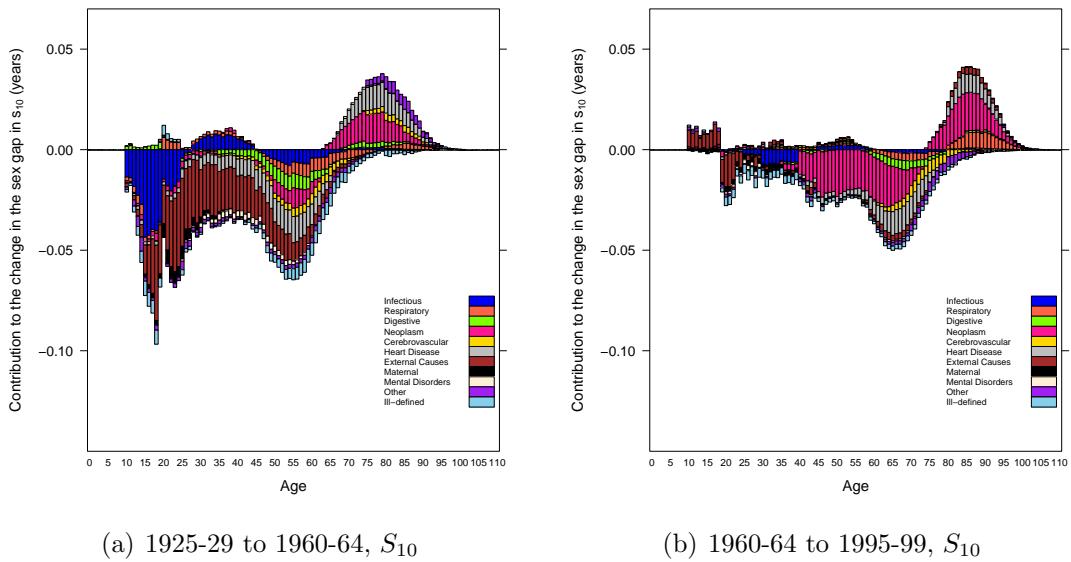


Figure 8.7: Contributions of changes in age-and cause-specific mortality rates to changes between time points in the difference in  $S_{10}$  between females and male, France. Data sources: Human Mortality Database, French Cause-of-Death Series, (HMD, 2009; Vallin and Meslé, ).

## 8.6 Conclusion

The results of this analysis confirm the most basic intuition about trends in variability of age at death during the second stage of the epidemiological transition—declines in infectious diseases lead to rapid mortality compression for both males and females. Female disadvantage in infectious disease mortality during childhood and young adulthood in the first stage of the epidemiological transition and continued disadvantage for males in external cause mortality across stages of the transition led to greater reductions in premature mortality for females in comparison to males during the second stage of the transition, which results in females experiencing much lower variability of age at death in comparison to males in the third stage.

A careful examination of the divergence in trends in  $S_{10}$  for French males and females led to two important results. First, more stable trends for males were not due to a balancing of the effect of improvements in chronic and degenerative diseases as one might expect to see in the shifting mortality era. Secondly, females have continued to experience a decline in  $S_{10}$  in more recent decades albeit substantially smaller than what was observed in the second stage of the mortality transition. This result showcases the potential impact that changes in neoplasm related mortality could have on the future course of trends in variability of age at death—whether shifting trends might give way to mortality compression if scientific advances or behavioral changes (e.g. smoking reduction) lead to a reduction in premature mortality due to cancer.

The potential contribution of cancer to future trends in mortality compression is certainly worthy of further investigation. This study has been based solely on the case of France because the data series allowed examination of both the second and third stages of the epidemiological transition; however, study of the effects of cancer on trends in variability of death in more recent decades does not necessitate such a long data series so there is potential to investigate this in a number of more developed countries with cause-of-death data. Secondly, there are many types of cancer, and time trends differ for different types of cancers. Further analysis is needed to determine specifically what types of cancers may be the most important for determining future trends in variability of age at death.

Table 8.3: Decomposition of differences in  $S_{10}$  between males and females by age and cause, France. A negative sign indicates that gender differences in mortality for a particular cause contribute to females experiencing lower variability of age at death.

	10-29	30-49	50-69	70+	Total
<b>1925-29</b>					
Infectious diseases	0.39	-0.16	0.06	0.01	0.31
Respiratory diseases	-0.02	-0.05	0.09	0.13	0.15
Digestive diseases	-0.02	-0.01	0.05	0.02	0.03
Neoplasm	-0.01	0.04	0.00	0.00	0.04
Cerebrovascular diseases	0.00	-0.01	0.06	0.09	0.13
Heart diseases	0.03	0.01	0.09	0.06	0.19
External causes	-0.39	-0.16	0.07	0.04	-0.43
Maternal causes	0.10	0.04	0.00	0.00	0.14
Mental disorders	0.00	0.00	0.00	0.00	0.00
Other	0.01	0.00	0.03	0.06	0.11
Ill-defined and unknown causes	0.00	-0.05	0.09	0.10	0.14
Total	0.09	-0.35	0.54	0.51	0.79
<b>1960-64</b>					
Infectious diseases	0.00	-0.08	-0.03	0.02	-0.08
Respiratory diseases	-0.01	-0.04	-0.02	0.13	0.07
Digestive diseases	-0.01	-0.07	-0.04	0.06	-0.06
Neoplasm	-0.04	0.03	-0.04	0.21	0.15
Cerebrovascular diseases	-0.01	-0.04	-0.01	0.13	0.07
Heart diseases	-0.01	-0.15	-0.06	0.26	0.03
External causes	-0.83	-0.55	-0.08	0.03	-1.42
Maternal causes	0.03	0.03	0.00	0.00	0.06
Mental disorders	-0.01	-0.06	-0.02	0.01	-0.07
Other	-0.03	-0.03	-0.01	0.13	0.07
Ill-defined and unknown causes	-0.05	-0.07	-0.02	0.06	-0.08
Total	-0.95	-1.02	-0.33	1.04	-1.27
<b>1995-99</b>					
Infectious diseases	-0.01	-0.14	-0.02	0.02	-0.15
Respiratory diseases	-0.01	-0.02	-0.04	0.21	0.14
Digestive diseases	0.00	-0.08	-0.08	0.04	-0.12
Neoplasm	-0.03	-0.15	-0.43	0.48	-0.14
Cerebrovascular diseases	0.00	-0.03	-0.04	0.06	0.00
Heart diseases	-0.02	-0.17	-0.20	0.32	-0.07
External causes	-0.84	-0.55	-0.10	0.06	-1.43
Maternal causes	0.01	0.01	0.00	0.00	0.01
Mental disorders	-0.02	-0.06	-0.03	0.00	-0.11
Other	-0.03	-0.04	-0.03	0.07	-0.02
Ill-defined and unknown causes	-0.09	-0.13	-0.05	0.04	-0.24
Total	-1.06	-1.37	-1.02	1.30	-2.15

# Chapter 9

## Conclusion

The main purpose of the research conducted in this dissertation was twofold. First, I aimed to provide a better understanding of the mortality conditions that underlie the transition from mortality compression to shifting mortality. Secondly, I sought to explain the gaps in variability of age at death between males and females and between certain countries that emerged during the era of mortality compression. In addition to presenting my own results, I have tried to synthesize research conducted in the areas of mortality compression, mortality disparities, and the implications of greater certainty in timing of death. In this chapter, I summarize what I have done in the dissertation, discuss the broader implications of my results, outline the limitations of my study, and describe avenues for further research.

### 9.1 Summary

In my review of the literature in Chapter 2, I identified a number of key questions in this area of demographic research, some of which I have tried to answer in this dissertation. With regards to the transition from mortality compression to shifting mortality, I discovered that interpretation of the timing and geographic coverage of the transition varies substantially depending on the measure of variability of age at death used in the analysis. Most of the research in this area focuses on whether or not a transition has been observed and does not seek to explain the phenomenon. Attempts at explanation tend to focus on non-divergence in the age pattern of mortality change (Wilmoth and Horiuchi, 1999; Bongaarts, 2005; Thatcher et al., 2008). This led me to test whether non-divergence in the age pattern of mortality always leads to a shift in the death distribution and whether current shifting conditions can be attributed to non-divergence in the age pattern of mortality change in Chapters 5 and 6.

In Chapter 2, I also review the traditional mortality disparities literature concerning mortality differentials across predefined groups as well as studies in which measures of inter-individual inequality are used to assess mortality disparities at the population level. My review of the traditional mortality disparities literature suggests that these disparities exist

along a number of dimensions and that they have persisted across time. A review of studies in which measures of life span disparity such as  $S_{10}$ ,  $e^\dagger$ , the Gini index are used suggests that the differentials in life span disparity observed among groups are themselves indicative of disparity and can provide insights into traditional mortality disparities that are obscured when only mean life span is taken into account. Thus, the other main focus of this dissertation project is trying to understand what factors produce the disparity in life span disparity observed among groups.

Those who document the history of the human mortality experience often comment that the changes in mortality observed over the course of the mortality transition must have had some impact on other areas of human life besides death. In Chapter 3, I attempt to place measures of variability of age at death in a larger context by exploring the meaning of changes or differentials in these measures from a biological, economic, psychological, and demographic perspective. My review of the literature suggests that while greater certainty in timing of death may increase the trauma of early deaths for a decedent's social network, these changes have likely lessened overall anxiety about the possibility of dying and empowered humans with a greater sense of control over their lives. This greater sense of agency coupled with a greater certainty about survival prospects have allowed humans to take control of another key aspect of their demographic lives: their fertility behaviors. As a result of increased certainty in timing of death and the related delays and declines in fertility, the life course of humans has been radically altered from our evolutionary history.

In Chapter 5, I reexamine the relationship between the age-pattern of mortality change and trends in variability of age at death in order to evaluate whether shifting mortality is an inevitable outcome of the mortality transition. I do not find support for the simple hypothesis that proportional mortality change that is fixed across age necessarily leads to a shift in the death distribution. I do find that the rapid declines in variability of age at death observed in Sweden from 1876-1955 were due to both the divergence in the age pattern of mortality decline and the greater sensitivity of measures of variability of age at death to changes in age-specific mortality rates during this time period. At the turn of the 20th century, mortality conditions in Sweden were particularly primed for compression. By the middle of the 20th century, there was less potential for further compression as mortality in younger ages had been substantially reduced; however, the simulation results presented in this chapter suggest that further compression is possible.

The decomposition results presented in Chapter 6 confirm the basic intuition that declines in mortality in younger ages led to rapid mortality compression during the second stage of the epidemiological transition. As mortality declines have shifted to older ages in the third stage of the epidemiological transition, there is potential for mortality expansion (i.e. an increase in variability of age at death); however, measures of variability of age at death have largely remained stable over the past half century because declines in mortality at older ages, which contribute to an increase in  $S_{10}$ , are balanced by further declines at younger ages, which act to decrease  $S_{10}$ . The age pattern of the decomposition results presented in Chapter 6 do not mirror the age pattern of the sensitivity results presented in Chapter 5 suggesting

that the current shifts in the death distribution being observed are not necessarily due to a non-divergent age pattern of mortality change. This runs contrary to current theories of shifting mortality and examining changes by age and cause in Chapter 8 provides additional evidence that the conditions that give rise to shifting mortality do not conform to the simple shifting mortality story line that I outline in Chapter 9.2.

In Chapter 6, I also examine the two pathways through which changes in age-specific mortality affect the shape of the death distribution ( $d_x = l_x m_x$ )-directly through the  $m_x$  term and through the survivorship function,  $l_x$ . The results of this analysis indicate that the shifting mortality era is unique in that changes in age-specific mortality rates influence the shape of the death distribution both directly and through the survivorship term at older ages, whereas only the direct effect was evident in earlier eras.

Chapter 7 focuses on gaps in variability in age at death between certain groups that emerge during the era of mortality compression and then continue in the era of shifting mortality. A gap in variability of age at death between males and females emerges within each of the geographical areas represented in the Human Mortality Database over the course of the epidemiological transition with females experiencing greater declines in variability of age at death in comparison to males. The decomposition results presented in the chapter suggest that these gaps emerge because females gained an advantage over males with regards to premature mortality. In Chapter 8, cause-of-death decomposition results for France reveal that this is due to a decline in infectious disease, which young females had suffered disproportionately, and male disadvantage in external cause mortality across the stages of the epidemiological transition.

In Chapter 7, I also examine the gap in variability of age at death that emerges between Sweden and the United States in the era of mortality compression. In this case, I find that similar to the sex gap, greater declines in premature mortality in Sweden in comparison to the United States partly explain the emergence of the gap; however, in contrast to the age patterns that produce the sex differences in variability of age at death within each country, the cross-country gap is also a result of the United States having an advantage in mortality at older ages in comparison to Sweden.

In Chapter 8, I examine the questions of the transition from mortality compression to shifting mortality and sex differentials in variability of age at death from a cause-of-death perspective. I focus on sex-specific trends in variability of age at death in France and investigate the emergence of the sex gap in variability of age at death during the era of mortality compression and a more subtle widening of the gap in variability of age at death between males and females in the second half of the twentieth century during the era of shifting mortality. A careful examination of the divergence in trends in  $S_{10}$  for French males and females leads to two important results. First, more stable trends for males were not due to a balancing of the effect of improvements in chronic and degenerative diseases on either side of the crossover age as one might expect to see in the shifting mortality era. Secondly, females have continued to experience a decline in  $S_{10}$  in more recent decades, albeit substantially smaller than what was observed in the second stage of the mortality

transition, due to continued declines in cancer related mortality at younger ages, which males do not experience. This result showcases the potential impact that changes in cancer related mortality could have on the future course of trends in variability of age at death, which I discuss further in the next section.

## 9.2 Implications

I begin this discussion of implications by briefly reviewing what I expected to find given the background literature in this area. I anticipated that declines in variability of age at death would mostly be attributable to gains made against mortality at younger ages during the era of rapid mortality compression. The compression occurs at an especially rapid pace because divergence in the age-pattern of mortality change leads to more rapid declines in mortality at younger ages. The transition from mortality compression to shifting mortality occurs when mortality at young adult ages has been substantially reduced and the age pattern of mortality change becomes relatively non-divergent across age. In terms of cause-of-death composition, the declines in variability of age at death observed during the era of mortality compression can mainly be attributed to declines in infectious disease and other causes of premature mortality. Once mortality due to these causes has been substantially reduced, shifting mortality occurs because deaths due to chronic and degenerative diseases are balanced on either side of the crossover age and reductions in mortality due to these causes results in a balance of positive and negative contributions to change in variability of age at death.

Many of the results that I present in the dissertation are intuitive and conform to this story. Not surprisingly, my decomposition analyses reveal that the rapid mortality compression, which was observed during the second stage of the epidemiological transition, was mainly the result of the declines in infectious disease at younger ages. Comparing the decomposition results for this period to the results of the sensitivity analysis and the simulation experiments, it is obvious that divergence in the age pattern of mortality change led to faster decline in variability of age at death than what would have been observed otherwise.

While the results of the simulation experiments and perturbation analysis confirm intuitions about what happened during the era of mortality compression, they also offer insights into how the age pattern of mortality change and initial mortality conditions interact to produce mortality compression. The simulation results suggest that a non-divergent age pattern of mortality change does not automatically produce shifting mortality. For instance, starting with the initial mortality conditions experienced by Swedish males in the period 1900-04 and applying proportional mortality change that is fixed across age resulted in mortality compression, albeit substantially smaller than what would be observed with a divergent pattern of mortality change with greater improvements at younger ages. Related to this, the results of the simulations and the sensitivity analysis suggest that certain initial mortality conditions are particularly primed for compression, and that the potential for compression

does not necessarily decrease as mortality declines as one might expect (i.e. initial mortality conditions in Sweden in 1900 lead to more rapid declines in variability age at death in comparison to conditions in 1800 or 1850 in simulation experiments and show greater potential for compression in sensitivity results).

The simple story line breaks even further when evaluated in the era of shifting mortality, although some of the plot points hold. For instance, the sensitivity results suggest that, in contrast to earlier eras, the changes in mortality at ages on either side of the crossover age have the greatest potential to impact measures of variability of age at death. These two humps that appear in opposition on either side of the crossover age in the sensitivity results suggest that it is possible that mortality decline on both sides of the crossover age will result in a balancing of effects and thus no change in measures of variability of age at death. The simulation experiments confirm that non-divergent age patterns of mortality change lead to shifting mortality conditions when initial mortality conditions correspond to those observed among Swedish males in the periods 1950-54 and 2000-04.

Both the all-cause decomposition results and the multiple cause-of-death decomposition results provide evidence that the underlying conditions producing shifting mortality currently are complex and do not conform to the simple story line. While the all-cause decomposition results confirm that shifting conditions are due to a balancing of effects on either side of the crossover age, the age pattern of contributions does not match the sensitivity results suggesting that divergent age patterns of mortality change may be playing a role. The cause-of-death decomposition results for French males reveal that the relative stability in variability of age at death observed between 1960-64 and 1995-99 was not due to the contribution of declines in chronic and degenerative diseases balancing out on either side of the crossover age. Rather, declines in chronic and degenerative diseases mainly acted to increase in variability of age at death during this period while declines in external cause mortality at younger ages counteracted these contributions.

Examining changes in the gap in variability of age at death between males and females by age and cause also provides useful insights into the forces that may shape future trends in variability of age at death. Following the period of rapid compression in mortality where the gap in variability of age at death between the sexes first emerged (1925-29 to 1960-64), there was a continued widening of the gap in France with females experiencing continued albeit slight compression. The cause-of-death decomposition results show that this continued divergence was the result of females making greater progress against cancer related mortality in middle ages. French death distributions broken down by cause reveal that cancer mortality is skewed towards younger ages, and the decomposition results suggest that declines in cancer mortality may be particularly important for future trends in variability of age at death. I will discuss future avenues of exploration for this area of research in Section 9.4.

## 9.3 Limitations

In this section, I discuss the limitations of the analyses carried out in this dissertation. I focus specifically on limitations with regards to my data, measure, and methods.

In this project, I have benefited from having access to extensive collections of historical life tables included in the Human Mortality Database and the WHO collection of 1,802 life tables as well as a long series of cause-of-death data from France; however, these data sources have limitations and additional insights may have been gained by analyzing other data sets. For instance, I would have liked to have access to more recent life tables for developing countries in order to verify that a gap in variability of age at death emerges between females and males in all countries during the course of the epidemiological transition. On a related note, it would be interesting to see how the HIV/AIDS epidemic affects sex-specific trends in variability of age at death. It would also have been useful to have analyzed additional case countries in the cause-of-death decompositions included in Chapter 8. My reliance on the French Cause-of-Death series for making broad inferences about the nature of change in cause-of-death composition by sex during the eras of mortality compression and shifting mortality assumes that the French case is generalizable.

Analyzing trends in cause-of-death composition over the period 1925-1999 with the French Cause-of-Death Series also presents some issues because classification criteria have changed substantially over this time period as the International Classification of Diseases has gone through several revisions. As can be seen in the results presented in Chapter 8, the proportion of deaths classified as ill-defined or unknown decreases substantially over the period analyzed. This misclassification bias has the potential to bias the results indicating the contribution of changes in other causes of death on trends variability of age at death. For example, a presumed increase in cancer related mortality at the oldest ages acts to decrease  $S_{10}$  between 1925-29 and 1960-64 and is balanced by what appears to be a substantial decline in deaths due to ill-defined causes. This result likely does not reflect what is happening with cancer related mortality but instead reflects changes in the classification of cancer related deaths.

With regards to limitations in terms of measures, in most of the analyses that I pursue in this study, I rely on the measure of variability of age at death,  $S_{10}$ . In addition to exploring the effect of different cut off ages besides age ten, it would have been useful to incorporate additional measures of variability of age at death and life span disparity into my analysis. For instance,  $SD(M+)$  is a popular measure for studying the transition to shifting mortality, and it would have been useful to understand how this measure responds to changes in age and cause-specific mortality.

With regards to methods, in my cause-of-death decompositions, I examine discrete changes between periods (e.g. 1960-64 to 2000-04) and do not incorporate mortality data from intervening years. Incorporating data from these additional years will make my cause-of-death decompositions of sex-specific trends in variability of age at death more accurate. Currently, I am assuming a linear path in changes in age and cause-specific mortality rates between 1960-64 to 1995-99, and in incorporating intervening data, I can relax this strong assumption.

tion. In the future, I will want to decompose changes between each five year period within the forty year interval and then sum across intervals. I am already using this method in my all-cause decompositions of trends in sex-specific variability of age at death that I present in Chapter 7.

## 9.4 Further study

The results presented in this dissertation along with the limitations that I describe above spur me to extend my research in this area. With regards to the data limitations described above, it will be useful to extend my cause-of-death decomposition analysis to additional countries. While I will not be able to investigate the transition from mortality compression to shifting morality in most countries since most historical cause-of-death series do not extend as far back as the French series, I will be able to examine trends in variability of age at death across countries in more recent decades to examine the effect of cancer related mortality on current trends in variability of age at death. Specifically, I would like explore whether declines in cancer related mortality may be driving the recent declines in  $S_{10}$  observed in New Zealand, Sweden, and Switzerland as shown in Figure 1.2.

In addition to examining the effect of cancer mortality in general on trends in variability of age at death across countries, I would also like to examine how the effect varies for different types of cancers. Specifically, I am interested in examining the effect of lung cancer mortality on trends in variability of age at death. In a recent study, Wang and Preston find that incorporating cohort smoking histories into mortality projections for the United States results in much faster mortality decline because of the decline in smoking across more recent cohorts (Wang and Preston, 2009). Given the large impact of smoking on mortality in general, it would be useful to adopt a age-period-cohort approach to analyze how declines in smoking across cohorts might be affecting trends in variability of age at death.

The cause-of-death analysis could also be extended by examining the effect of changes in causes-of-death on variability of age at death in a different way. In the decomposition results presented in Chapter 8, the effect of a particular cause depends on the mean age at death in the overall death distribution, which is influenced by all causes of death. It may be useful to consider the shape of a distribution of deaths due to a particular cause, and how changes in mortality due to this cause might affect variability of age at death for that particular cause. This might allow for classification of different causes of death as compressors, expanders, or shifters depending on the effect that reductions in mortality due to that particular cause have on the cause-specific death distribution. For instance, the cause-of-death decomposition results for French males over the period 1960-64 to 1995-99, indicate that declines in cardiovascular mortality mainly act to increase  $S_{10}$  over this period since cardiovascular mortality is largely concentrated after the all-cause crossover age; however, a cause-specific distribution might show that deaths due to cardiovascular mortality are largely balanced on either side of the cardiovascular mortality specific crossover age, and

thus changes in this cause would lead to a shift in this cause-specific distribution.

In this dissertation, I have focused on understanding trends and differentials in variability of age at death, but for the most part I have not addressed an important underlying question: why do we observe this variability in the first place? Clearly, social inequality plays some role in the variability in outcomes that we observe in health and mortality, but prior research suggests that social disparities explain very little of the total variability of age at death observed at the population level. Underlying biological and genetic variation, which is being explored in the biodemographic literature, likely plays an important role in understanding variation. In my future research, I want to continue to focus on the theme of variability with an aim of understanding how biologic and genetic variation along with social disparities and environmental factors interact to create variability in health and mortality outcomes. Armed with a better understanding of the determinants of variability, I hope to explore the implications of these determinants for present and future population health.

## 9.5 *Mors certa, hora incerta*

*Mors certa, hora incerta.* Death is certain, its hour is uncertain. This statement is as true today as it was for the citizens of the Roman Empire. Still, the longevity revolution that has taken place over the past few centuries has substantially altered the ways humans live because deaths are now concentrated in old age. Furthermore, the results of the analyses presented here, like previous studies, suggest that humans do not appear to be approaching a biological limit to life span. While the future is unknowable, the lessons of the past, taken from the historical mortality experience documented here, indicate that there is potential for further improvements in longevity if advances continue to be made against chronic and degenerative diseases.

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