Exploring shifting mortality and gender differences in variability of age at death in France: a cause-of-death analysis.

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Abstract

The recent wane 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 this paper, I examine two issues regarding the transition to shifting mortality that have yet to be fully resolved. First, how can trends in variability of age at death remain stable even in the face of possible changes in cause-of-death composition? Second, why have trends in variability of age at death for different groups stabilized at different levels of variability rather than at a similar (possibly biological) level? Using a historical cause-of-death series from France and decomposition analysis, I am able to pursue answers to these questions through examination of sex-specific trends in variability of age at death and the evolution of the gap in variability of age at death between the sexes.

Please note that this paper contains colored figures.
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In the early stages of the epidemiological transition, reductions in mortality, especially early in life, led to rapid gains in life expectancy as well as greater concentration of ages at death. While life expectancy continues to rise today in most industrialized countries, declines in variability of age at death, a measure of how spread out deaths are across age, are tapering off and trends are becoming flat. This change has led some researchers to declare that these countries are either transitioning or are about to transition from an era of mortality compression to a new era of mortality change: the shifting mortality era [Wilmoth and Horiuchi, 1999, Bongaarts, 2005, Cheung and Robine, 2007, Canudas-Romo, 2008].

While the flattening of trends in measures of variability of age at death has been documented using a variety of measures and the implications of this trend have been explored, there are still many unresolved questions regarding the possible transition from an era of mortality compression to an era of shifting mortality. First and foremost, how is it possible that improvements in mortality are not necessarily accompanied by mortality compression? Efforts towards answering this fundamental question have focused on non-divergence in the age-pattern of mortality change [Wilmoth and Horiuchi, 1999, Thatcher et al., 2008]. In this paper, 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 paper is explaining 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. Figure 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. Figure 1 reveals that across countries females gained an advantage over males in terms of lower variability of age at death during an era of rapid mortality compression. In this paper, 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.

This paper contains six sections. In the next section, I present background information from the literature on the epidemiological transition, changes in the relationship between male and female mortality observed during the transition, current knowledge about what factors produce shifting mortality, and studies of differences in variability of age at death between groups. After offering an overview of the data, measure, and 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.

1 Background

1.1 Epidemiological transition theory and mortality compression

Epidemiological transition theory, originally put forth by Omran in 1971, describes the substantial declines in mortality observed over the recent course of human history from a cause-of-death perspective. Omran describes three distinct stages: (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]. Similarly, Fries believed that biological limits to life span would prevent progress against mortality at the oldest ages although in contrast to Omran he anticipated that progress against chronic diseases would concentrate morbidity in the oldest ages [Fries, 1980].

In contradiction to the assumptions and predictions of Omran and Fries, 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]. In this stage, mortality compression can give way to shifting mortality.

In the discussion of my results in this paper, I adopt Robine's classification of the stages that characterize the epidemiological transition. Thus, when I refer to the third stage of the transition in later sections, I am referring to Robine's stage *The Age of the Conquest of the Extent of Life*.

1.2 Sex differences in mortality

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 2 (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.

Of critical importance for understanding how females come to experience lower variability of age at death in comparison to males is understanding why females gained such a substantial mortality advantage over males 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, a rise in accidents, and the decrease in maternal mortality 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 gap in variability of age at death between male and female mortality over time is largely influenced by differences in infectious, external cause, and cancer related mortality between males and females.

1.3 Response of measures of variability of age at death to changes in mortality and understanding differences in variability of age at death across groups

In this paper, I look at how changes in age and cause-specific mortality produce mortality compression, mortality expansion, or shifting mortality. 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 Wimoth 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.[1]

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 (β) [Thatcher et al., 2008]. As long as mortality change is fixed across age on a proportional scale (non-divergent 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

[1] 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.

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 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 non-divergent 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].

To my knowledge, there have been no previous studies of transition from mortality compression to shifting mortality that utilize cause-of-death data. The studies outlined above suggest that in order to observe shifting mortality the cause-of-death composition has to shift in such a way as to produce a non-divergent age pattern of mortality change (or at least non-divergent across the ages that have the most potential for influencing trends in variability of age at death). In Section 4, I discuss why we might expect to see this type of pattern during the third stage of the epidemiological transition.

Previous studies have examined differences in variability of age at death or life span disparity observed among countries using a cause-of-death approach. Edwards and Tuljapurkar show that exceptionally high variability of age at death in the United States in comparison to other more developed countries can not be explained solely by the higher rates of external cause mortality observed in the United States [Edwards and Tuljapurkar, 2005]. Shkolnikov et al. decompose differences in the Gini index between the United States and the United Kingdom and find that US disadvantage in external cause and cardiovascular disease mortality at younger ages and US advantage in respiratory and cardiovascular disease mortality at the oldest ages are important factors leading to higher levels of life span disparity observed in the US in comparison to the UK [Shkolnikov et al., 2003]. Examining differences in S_{10} between Sweden and the US, Nau and Firebaugh find that differences in mortality due to heart disease, traffic accidents, homicides, and infectious disease are most important in explaining the higher variability of age at death observed in the US in comparison to Sweden [Nau and Firebaugh, 2009].

2 Data, measure, methods

In order to document sex-specific trends in variability of age at death, I utilize the measure S_{10}, the standard deviation of ages at death above age 10, popularized by Edwards and Tuljapurkar [Edwards and Tuljapurkar, 2005]. This measure can be calculated with data from a life table according to the following formula:

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

In the above equation, x indicates age, T is the oldest age group, 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 ($e_{10} + 10$), and d_x is the number of deaths in the period life table that occur at age x.

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}, which is utilized in this analysis, 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 [Wilmoth and Horiuchi, 1999], $e^\dagger$ [Zhang and Vaupel, 2009], and SDM [Canudas-Romo, 2008]) and measures that do not fully account for changes in premature mortality because they only measure dispersion above the modal age at death (e.g. $SD(M+)$ [Kannisto, 2001]). Trends in S_{10}, as shown in Figure 1, clearly reflect the stages of epidemiological transition with an initial period of high variability (first stage of transition) followed by rapid declines (indicative of mortality compression-second stage of transition) and then a clear disruption of these declining trends into flatter trends in variability of age at death (indicative of shifting mortality and corresponding to the third stage of the transition proposed by Robine [Robine, 2001]).

The life table data needed to compute trends in sex-specific S_{10} for France is drawn from the Human Mortality Database (HMD) [HMD, 2009]. The French series of period life tables in the HMD cover the period 1816-2006. I utilize the series of data representative of the mortality experience of the civilian population of France rather than the series representing the experience of the total population thus excluding war related mortality suffered by members of the military. This choice should not influence the results of my cause-of-death decomposition analyses, though, because I focus on changes observed between discrete non-war periods: 1925-29 to 1960-64 and 1960-64 to 1995-99. I use the periods 1925-29 and 1995-99 because they represent the first and last five-year time periods available in the cause-of-death data. In the period 1960-64, the declining trends in S_{10} are disrupted so by choosing this as my middle time point I have divided my data series into a component representing the mortality compression scenario (1925-29 to 1960-64) and a component representing the shifting scenario (1960-64 to 1995-99, at least for French males).

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]. 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 HMD in order to obtain age and cause-specific mortality rates for each sex.[2]

[2]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, $m_x, i = m_x * p_x, i$

I decompose sex-specific trends in S_{10} over time and differences in S_{10} between the sexes at fixed points in time using a general decomposition method proposed by Horiuchi et al. [Horiuchi et al., 2008]. More details on the decomposition method are provided in the Appendix. 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.

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, 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 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 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 transfered into the infectious category in order to see how this impacts the results of the analysis.

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 3(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 3(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.

In this article, I am interested in understanding how variability of age at death changes in response to changes in the composition-of-deaths 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 Figures 3(c) and 3(d), 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 4 and 5 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 analayses presented in Section 5.

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 $e^{\dagger}$, 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 ages.

Under the mortality conditions observed in more developed countries today, improvements in mortality in childhood and young adulthood do not have as much potential to affect variability because the left-hand tail of the death distribution is already relatively flat.[3] 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 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.

5 Decomposition results

5.1 Decomposing changes over time in sex-specific trends in S_{10}

Figure 6 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 2

Between 1925-29 and 1960-64, male S_{10} declined by 3.6 years and female S_{10} declined by 5.6 years. Consistent with epidemiological transition theory, most of this decline was due to reductions in infectious disease mortality. Decomposition results for this time period for males are shown in Figure 6(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. Thus, these results indicate that most of the 3.6 year decline observed for males was due to declines in infectious disease

[3] There is more potential for infant mortality to impact trends in comparison to changes childhood or young adult mortality but even the potential for infant mortality has diminished over time.

mortality at younger ages, which fall below the crossover age. Above the crossover age (approximately age 55), increases in age and cause-specific mortality between these two periods act to decrease S_{10} with the greatest contribution coming from an apparent increase in neoplasm mortality; 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). As will be discussed in more depth in Section 5.2, Figure 6 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 1.2 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.

Between 1960-64 to 1995-99, 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 6(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 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 6(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, 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.

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 7 and Table 3.

During the first period, 1925-29, shown in Figure 7(a), female S_{10} was higher than male S_{10} by .79 years. In Figure 7, 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 7(b) and 7(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, 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(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 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.

6 Conclusion and Future Directions

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 continual disadvantage for males in external cause mortality across stages of the transition lead 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.

A Decomposition Method

In the paper, I am interested in both decomposing trends in variability of age at death (as indicated by S_{10}) over time and differences in variability of age at death between males and females into the contributions of either changes or differentials in age-specific mortality rates respectively. I carry out these decompositions using the continuous-change method of decomposition developed by Horiuchi et al. [Horiuchi et al., 2008]. 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 age and cause-specific mortality rates, $m_{x,i}$ to the total change in S_{10}.

As a first step to carrying out this decomposition, I express S_{10} in terms of all cause age-specific mortality, $m_{x,i}$.

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

where M_{10} is $e_{10} + 10$ and T is the oldest age group

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

$$S_{10} = \sqrt{\frac{\sum_{x=10}^{T}(x + a_x - \sum_{x=10}^{T} x e^{\sum_0^x -m_x} m_x)^2 \times e^{\sum_0^x -m_x} m_x}{\sum_{x=10}^{T} e^{\sum_0^x -m_x} m_x}}$$

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:

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

Now, I rewrite this equation to incorporate age and cause-specific mortality $m_{x,i}$:

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

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

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

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

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

Then, create N matrices $\mathbf{A}_{k\bullet}$. 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 interval and ranges from 1 to N. $\mathbf{A}_{k\bullet}$ holds the all values of $m_{x,i}$ observed at the midpoint of interval k (e.g. $\mathbf{A}_{xi,5\bullet} = m_{x,i,1} + 4.5 * \Delta m_{x,i}$).

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 $m_{x,i}$ in each interval k is varied to the value observed at the beginning of the kth interval and then to the value observed at the end of the kth interval (these matrices can be denoted $\mathbf{A}_{xi,k+}$ and $\mathbf{A}_{xi,k-}$). 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}:

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

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Data websites

Data from the Human Mortality Database can be downloaded at www.mortality.org. French cause-of-death data is available at http://www-causfra.ined.fr.

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Table 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

Table 2: Decomposition of change in sex-specific S_{10} over time, 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
	1925-29 to 1960-64, Female				
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
	1960-64 to 1995-99, Female				
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
Total	-0.53	-0.80	-0.79	1.64	-0.49
	1925-29 to 1960-64, Male				
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
	1960-64 to 1995-99, Male				
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

Table 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
			1925-29		
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
			1960-64		
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
			1995-99		
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

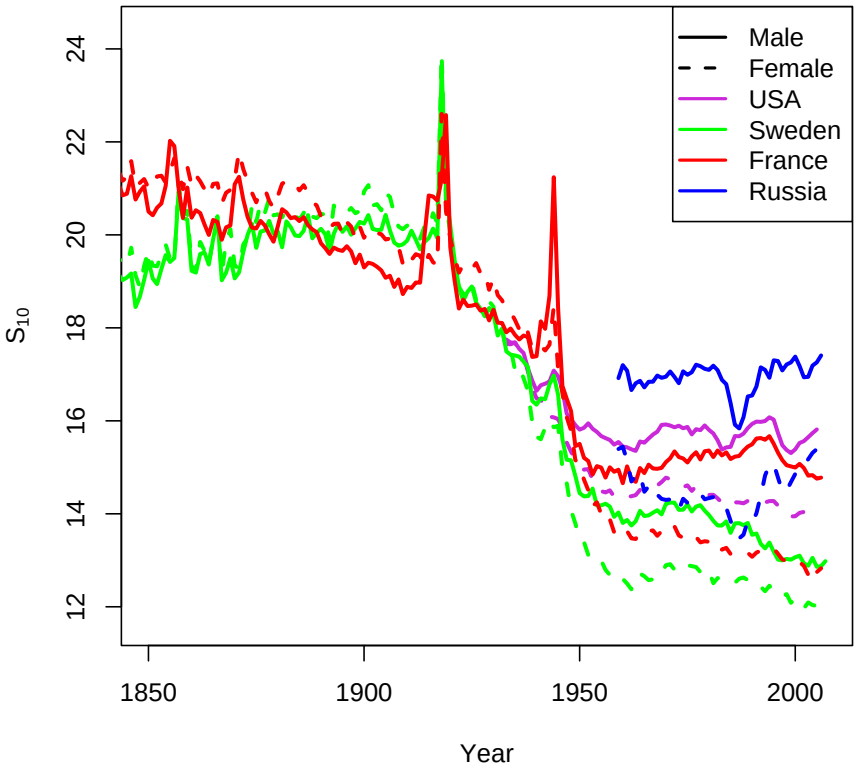


Figure 1: Trends in variability of age at death (as measured by S_{10}) for France, Russia, Sweden, and the United States. Solid lines represent male country-specific trends, and broken lines represent female country-specific trends.

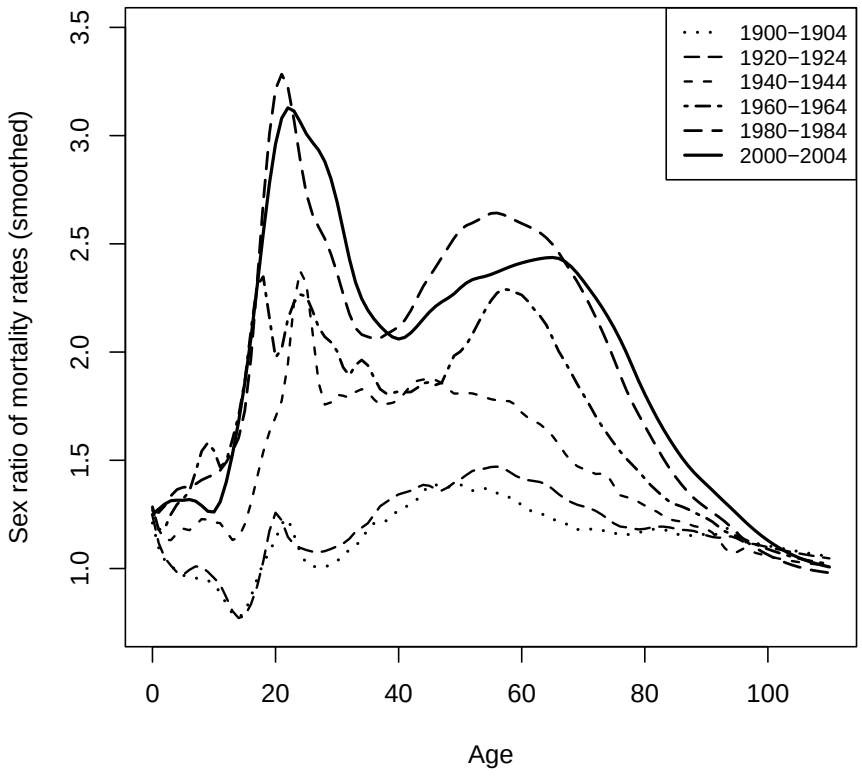
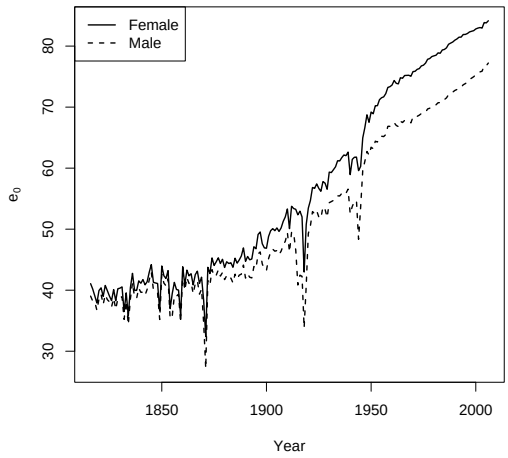
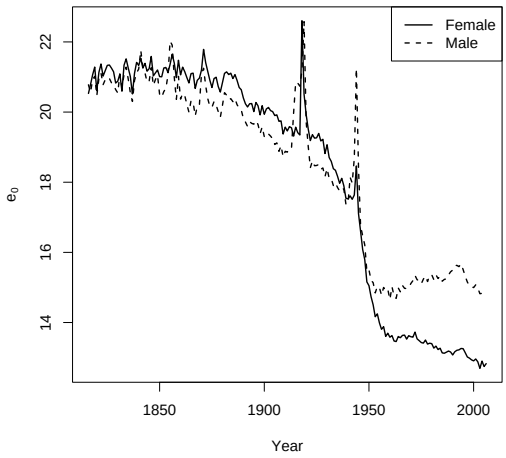


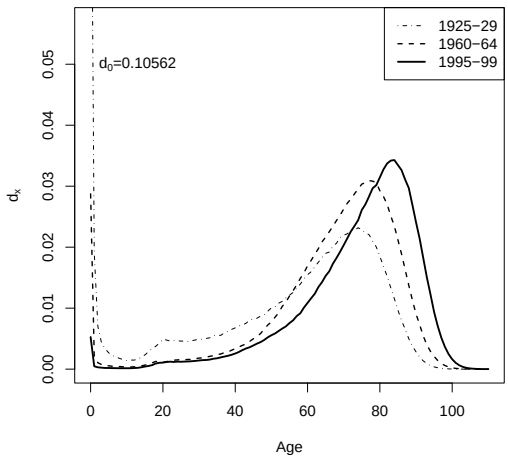
Figure 2: Ratio of male m_x to female m_x, France. Data source: Human Mortality Database.



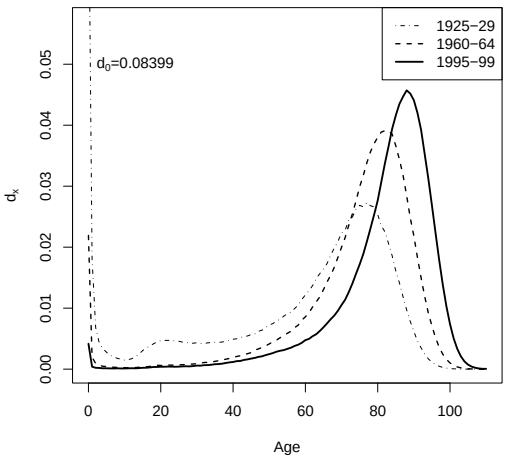
(a) e_0



(b) S_{10}

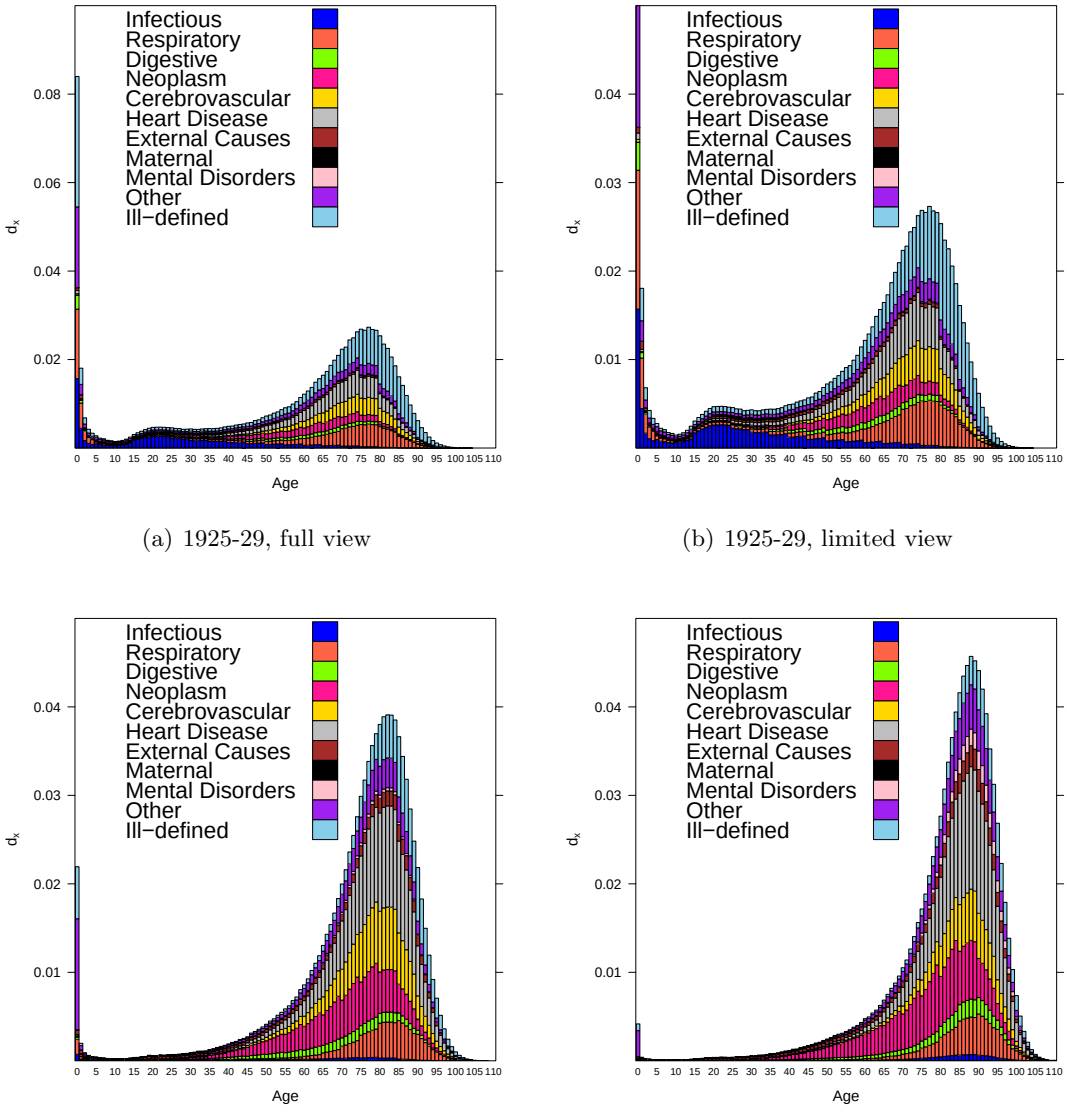


(c) d_x, Male



(d) d_x, Female

Figure 3: Sex-specific trends in life expectancy, variability of age at death, and the distribution of life tables deaths, France. Data source: Human Mortality Database.



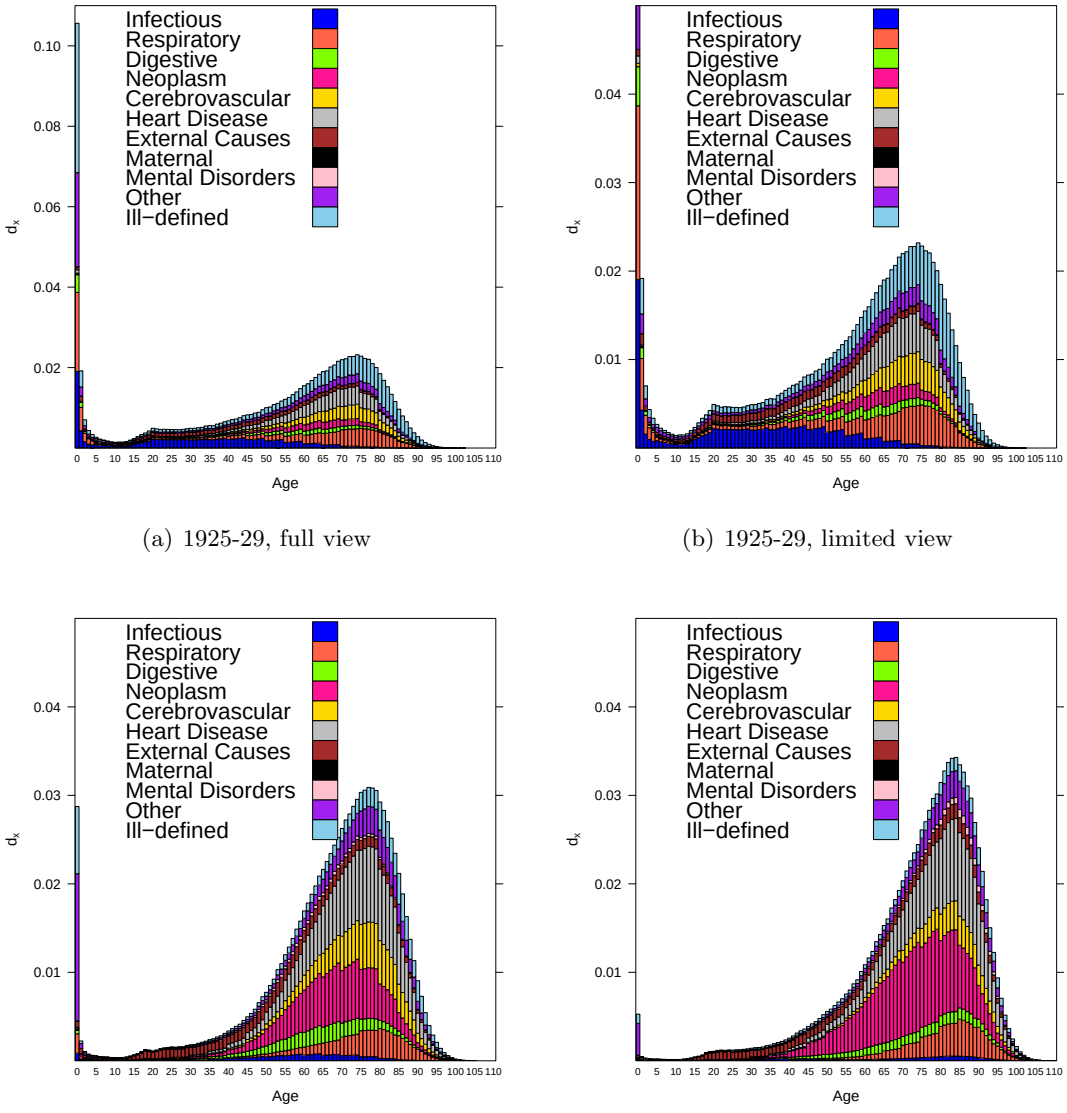
(a) 1925-29, full view

(b) 1925-29, limited view

(c) 1960-64

(d) 1995-99

Figure 4: Period life table death distributions for French females broken down by cause-of-death. Data sources: Human Mortality Database, French Cause-of-Death Series.



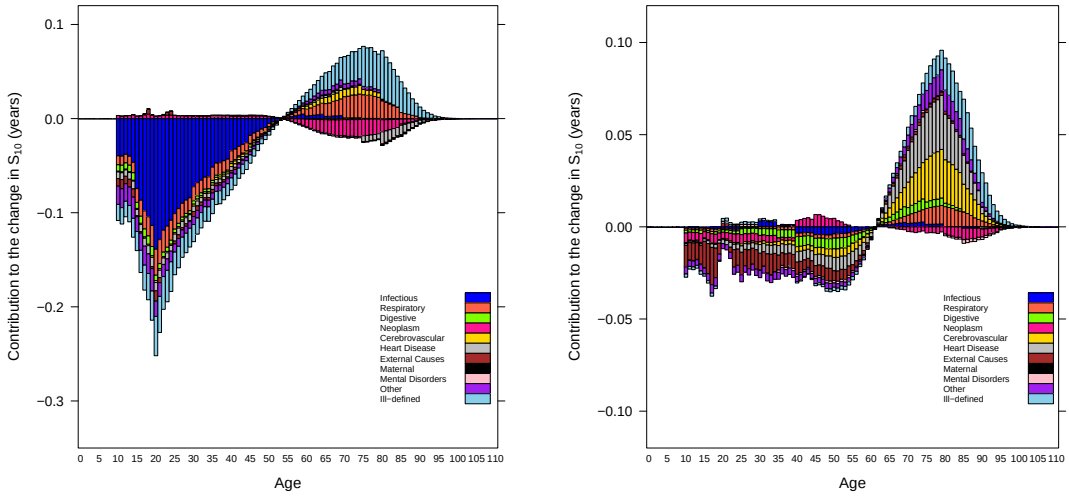
(a) 1925-29, full view

(b) 1925-29, limited view

(c) 1960-64

(d) 1995-99

Figure 5: Period life table death distributions for French males broken down by cause-of-death. Data sources: Human Mortality Database, French Cause-of-Death Series.



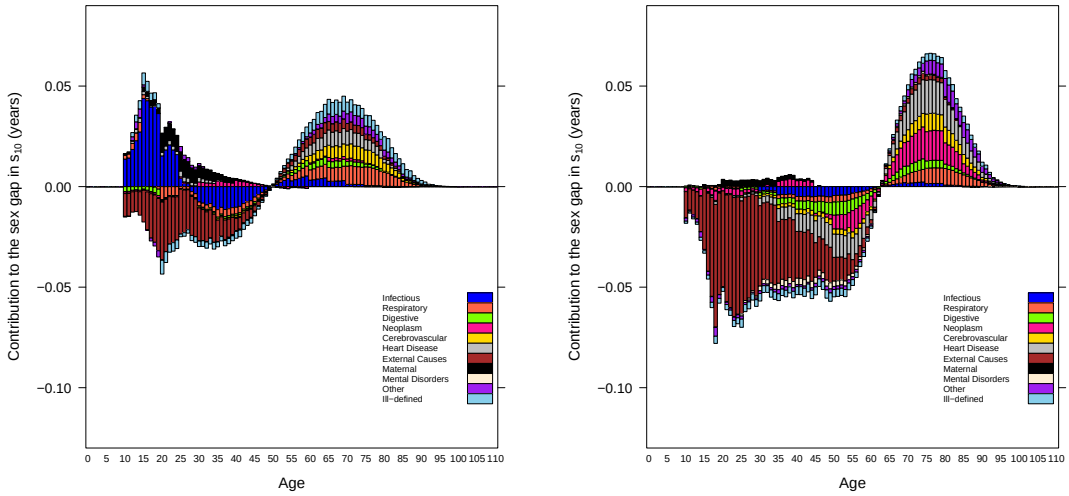
(a) 1925-29 to 1960-64, Male

(b) 1960-64 to 1995-99, Male

(c) 1925-29 to 1960-64, Female

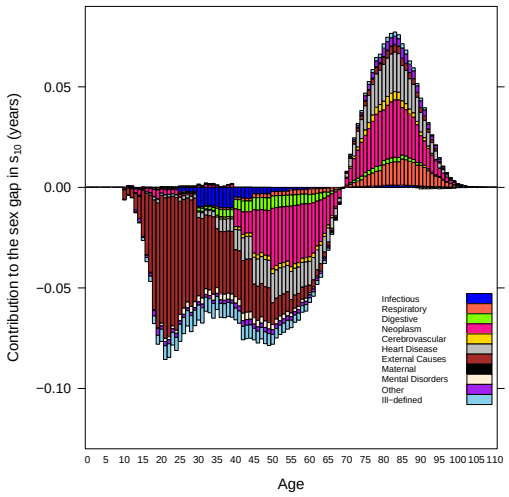
(d) 1960-64 to 1995-99, Female

Figure 6: Contributions of changes in age-and cause-specific mortality rates to changes in S_{10} over time, France.



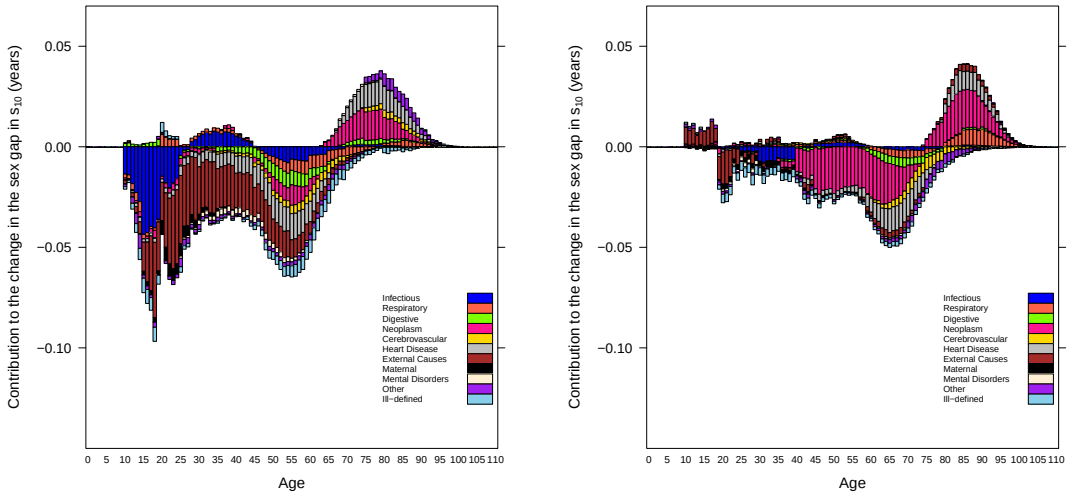
(a) 1925-29, S_{10}

(b) 1960-64, S_{10}



(c) 1995-99, S_{10}

Figure 7: Contributions of differences in age-and cause-specific mortality rates to differences in S_{10} between males and females at fixed points in time, France



(a) 1925-29 to 1960-64, S_{10}

(b) 1960-64 to 1995-99, S_{10}

Figure 8: Contributions of changes in age-and cause-specific mortality rates to changes between time points in the difference in S_{10} between males and females, France