

How Cause-Specific Mortality Contributes to Sex Differences in Life Expectancy over Time. Trends in Utah and Denmark

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How Cause-Specific Mortality Contributes to Sex Differences in Life Expectancy over Time

Trends in Utah and Denmark

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ABSTRACT

Female life expectancy exceeds male life expectancy. To better understand the basis for this discrepancy, we use Arriaga decomposition methods to examine differences in life expectancy by exploring age-specific and cause-specific mortality differences between the sexes and how these differences arise over time and between two distinct populations, Denmark and Utah (U.S.). Our focus is on how specific causes point to shifts in secular circumstances and behavioral factors that help to explain female-male differences in mortality across these two populations. Our findings point to the prominence of cardiovascular mortality as a key contributor to the female advantage in life expectancy for both Denmark and Utah but its influence is waning. External causes of mortality, including suicide and homicides along with motor vehicle accidents, also have large effects for both locales despite differences in lifestyles and policies. Cancer mortality in younger reproductive ages slightly subtract from the female life expectancy advantage. Overall, if the objective is to reduce sex differences in life expectancy, achieving this goal can be aided by promoting policies that reduce overall mortality but also mortality from key causes of death that are the basis for these discrepancies.

Keywords: Sex differences, Life expectancy, Causes of death, Decomposition

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1 INTRODUCTION

The survival advantage of females over males is nearly universal for most Western nations and it has been growing for many decades in the 20th century. This advantage is now seen throughout most countries in the world (Barford et al., 2006; Sauerberg et al., 2023) though the pattern varies by time and place (Bergeron-Boucher et al., 2022). Females enjoy this advantage for many reasons due to differences in biology (Austad, 2006), genetics (Eskes & Haanen, 2007; Moller et al., 2009) and behavioral/social factors (Kalben, 2000).

In previous work that explored sex differences in survival between three distinct populations (Utah, Sweden and Denmark), the female advantage in cohort life expectancy was found to be similar between these populations (Lindahl-Jacobsen et al., 2013). On one hand, the authors reported that over time the sex differences in the *improvement in life expectancy* was two years smaller for those identified as members of the Church of Jesus Christ of Latter-day Saints than in Denmark and Sweden. This finding suggests that shifts in lifestyle (for Denmark and Sweden, where the *improvement* was larger) are likely a reason for the overall change seen in cohort life expectancy. On the other hand, sex differences in cohort life expectancy at the age of 50 years were similar for individuals actively affiliated with the Church of Jesus Christ of Latter-day Saints and for Denmark and Sweden. Given that those affiliated with the Utah-based church are proscribed from using tobacco and alcohol, two key behaviors affecting disease risk, these results also point to possible biological mechanisms or other unknown risk factors.

1.2 AIMS

In this paper, we examine differences in life expectancy by exploring age-specific and cause-specific mortality differences between males and females and how these differences contribute to the female survival advantage. We investigate how these differences arise over time and between two distinct populations, Denmark and Utah in the United States. Our focus is on how specific causes of death contribute to sex differences in life expectancy over time and how these causes point to shifts in secular circumstances and behavioral factors that help to explain female-male differences in mortality. By looking at sex differences in life expectancy over many decades and across populations, we are able to observe how shifting cause-specific mortality patterns drive alterations in the patterns underlying these changes in life expectancy. Identifying these cause-specific patterns also helps to identify how previous changes in policies or social trends contributed to the male disadvantage and potentially nominate targets for intervention that may serve to increase life expectancy for both sexes and minimizing differences between them.

This paper includes data on Utah and Danish mortality and survival. Utah provides a useful comparison to Denmark that should serve to reveal differences in the distribution of causes of death that may align with differences in social patterns between the two populations. Utah's population has the largest percentage of individuals and families affiliated with the Church of Jesus Christ of Latter-day Saints. Those who are active adherents to the religion not only experience overall survival benefits but may have lower risks of specific types of death given that they are discouraged from using alcohol and tobacco, encouraged to fast once a month, and engage more often in church and related social activities compared to others (Mineau et al., 2004). Among states in the contemporary U.S., Utah has the lowest rate of adult tobacco usage (6.7%) whereas Denmark has rates 2.5 times higher. With respect to alcohol, per capita annual consumption is the lowest in Utah among all states (4.65 liters) and Denmark's is 10.4 liters.

Utahns have had and continue to have some of the highest fertility rates in the U.S. (Bean et al., 1990) which is likely to alter the risks of certain causes of death such as breast cancer (Lima et al., 2021). Early and frequent childbirth reduces the risk of incident breast cancer and lowers the risk of breast cancer mortality. By comparison, Denmark has significantly lower fertility rates with a total fertility rate of 1.46 child per women in 2024 (Statistics Denmark, 2025).

2 DATA

2.1 DENMARK

Death counts by primary causes of death were obtained from the Danish Register of Causes of Death (Helweg-Larsen, 2011; Juel & Helweg-Larsen, 1999) for one-year age and time period intervals from 1952 to 2015 for all citizens with an address in Denmark. The Danish causes of death registry data are based on the death certificates completed by medical doctors for all Danish decedents since 1952. Causes of death were classified according to four-digit International Classification of Diseases versions 6, 7, 8 and 10 in 1951–1957, 1958–1968, 1969–1993, and 1994–1998, respectively and were bridge-coded (Jacobsen et al., 2006; Janssen & Kunst, 2004) into 18 causes of death and by cancer specific deaths. This classification was applied to the Utah deaths as well. Person-year time by sex and one-year age and period groups was retrieved from the human mortality database (Max Planck Institute for Demographic Research, 2015). Deaths in Denmark for this study total 3,355,720.

2.2 UTAH

The Utah Population Database (UPDB) is the source for data on survival and causes of death in Utah. UPDB is a comprehensive database comprising linked demographic, medical, and genealogical individual and family-level data spanning the Utah population for the last two centuries (Smith et al., 2022; Smith & Mineau, 2021). UPDB holds considerable data on medical outcomes, including causes of death, for all decedents in the state since 1904. UPDB comprises data on families with and without an affiliation to the Church of Jesus Christ of Latter-day Saints.

The Utah data for this analysis are based on several inclusion criteria. To be part of the analysis, individuals must be Utah born between 1847 (the arrival of church members into the Utah territory) and 2015. We observe these individuals up to their last follow-up year (including the year of death) between 1904–2015. Since we compute a series of decade-specific *period life tables* and we need to observe causes of death, individuals must have the opportunity to be followed until 90 years of age. The official Utah vital record system was established in 1904 where Utah death certificates were issued that contain primary (and all other) cause of death information. In calculating *period life expectancy* from individual life histories, person-years were calculated from birth to death (in Utah) or through the year in which the individual was last seen alive in Utah. Given the broad historic coverage of UPDB, we are aware of deaths inside and outside Utah but that lack coded causes of death. These deaths are included for the purposes of generating life tables but their causes are in the "unknown" category. Because death certification did not begin until 1904, we start with the 1940–1949 period life table since it is well past 1904 and for persons born in 1847, they could have reached age 92. With these restrictions, the number of deaths totals 365,448.

2.3 DIFFERENCES IN DATA REGISTRATION

The data are structured differently for the two populations. For Denmark, the period life tables are based on age-sex counts in a given year, and death certificates are issued regarding decedents that same year. If a Danish resident leaves the country, no longer has an address in the country, and dies outside Denmark, they are not included in the estimation.

For the Utah analysis, the data are at the individual-level over their life time. If a person is alive and then dies in a given year within Utah, this person is counted in the person-year and death calculations when estimating mortality rates. If instead they leave Utah in a given year and we observe their death outside Utah, they are included in the person-year calculation for all preceding time points but are censored at the year they leave Utah. The UPDB often records these out-of-state deaths as well as the year of out-migration but the UPDB does not collect non-Utah death certificates where causes of death are recorded. In the end, the full life history data of individuals are formatted to derive the usual period life table person-year and death counts by calendar year and sex.

3 METHODS — ARRIAGA DECOMPOSITION

The study of sex differences in causes of death permits an analysis of the differences in mortality from two different perspectives. First, there is the estimation of how each cause of death affects the *sex difference in mortality for each age group*. This is followed by a second estimation, where we determine the contribution of each cause of death to *sex differences in life expectancy* (Arriaga, 1984, 1989).

Each of these stages of estimation is summarized here. The contribution of differences in life expectancy attributable to sex differences in all-cause mortality at each age is calculated first. This means that we estimate the years added or subtracted from life expectancy attributable to differences (above or below) in the mortality rates in the period life table. Then, the influence of mortality differences by causes of death on life expectancies is calculated. This estimation requires an assumption: the contribution to *sex differences in life expectancy* by causes of death *for each age group* is proportional to the *sex differences in the total mortality rate* due to the sex differences in cause-specific mortality in the same age group. For example, if the contribution to the sex difference (female minus male) in **life expectancy** for ages 50–54 was 1.2 years, and the sex difference in cardiovascular **mortality rates** for ages 50–54 represented 30% of the sex difference in mortality rates of all causes for ages 50–54, then the contribution of cardiovascular mortality for ages 50–54 to sex differences in **life expectancy** would be $(1.2 \text{ years} \times 0.30) = 0.36 \text{ years}$.

The following is a general presentation of these calculations adapted from Arriaga (1989), though other closely related methods give similar results (Beltrán-Sánchez & Preston, 2007). For each age group j , the influence on life expectancy attributable to sex differences in mortality for a given age group is Age - Specific Difference in Life Expectancy or $ASD_j(e_x)$, which is a function of the mortality difference for each age group between males and females ($m_j^{male} - m_j^{female}$). We define $D_j = f(m_j^{male} - m_j^{female})$ to represent the total mortality sex difference for age j . Now, this is expanded to represent each age-specific mortality difference for cause of death c : $cD_j = c m_j^{Male} - c m_j^{Female}$. Accordingly, if you sum all the cD_j across all causes you get D_j . This simply means that the difference in total mortality between groups for a given age equals the sum of all differences by cause in that age group.

The aforementioned assumption means that the influence on life expectancy attributable to a sex difference for a cause of death c is proportional: $cASD_j(e_x) = ASD_j(e_x) \times (cD_j/D_j)$. Estimation of the Arriaga decomposition was done using R (Auger et al., 2014).

4 RESULTS

Both figures for Utah and Denmark (Figures 1 and 2, respectively) have age on the x-axis and a y-axis showing the difference in years of life expectancy (LE) between males and females. Each age-specific bar is "stacked" comprising the contribution that each cause of death (the colored components or $cASD_j(e_x)$) provides toward the sex difference in LE at a given age. As males generally have higher mortality rates than females at all ages, the stacked bars show positive values indicating that most causes of death contribute to the female advantage in LE. Negative values occur for some ages where a specific cause of death *reduces* the sex differences in LE that generally favor females. The net height of an age-specific stacked bar (adding all positive and negative values) is the overall contribution, at that age, to the sex difference in LE.

To establish how we interpret the decompositions, we first provide an example assessment for one age- and time-specific results in a single decade (1970–1979) to setup how we interpret the decompositions of sex differences in life expectancy. For this decade at age 65 in Utah, the largest contribution to the female advantage is attributable to diseases of the circulatory system (component). Indeed, it is the differential effects of mortality by cardiovascular disease that is most responsible for the female advantage for ages 45–85.

Several key trends arise by period, age and causes of death that serve to reveal how shifts in the female life expectancy advantage have occurred for both populations. With respect to historical periods, the size of the overall female advantage is smaller in the early decades but then grows substantially in the ensuing years and then shrinks starting in the 1990s.

Figure 1 *Arriaga decomposition of period life expectancy by decade, age and cause of death for males and females in Utah*

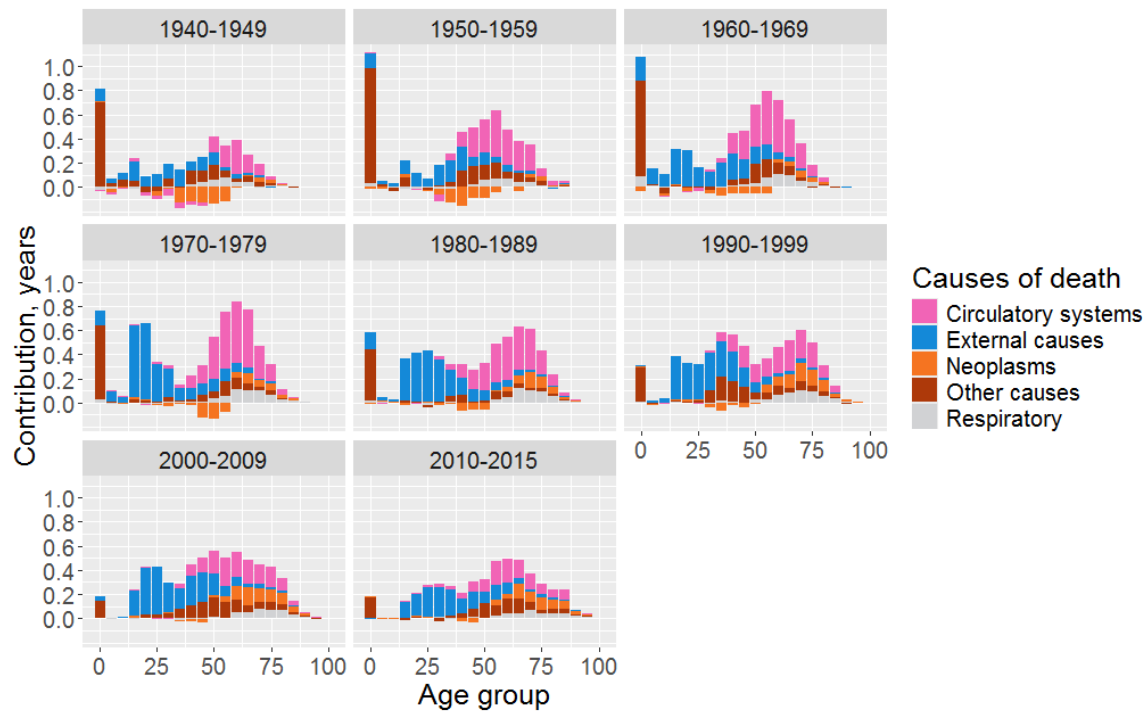
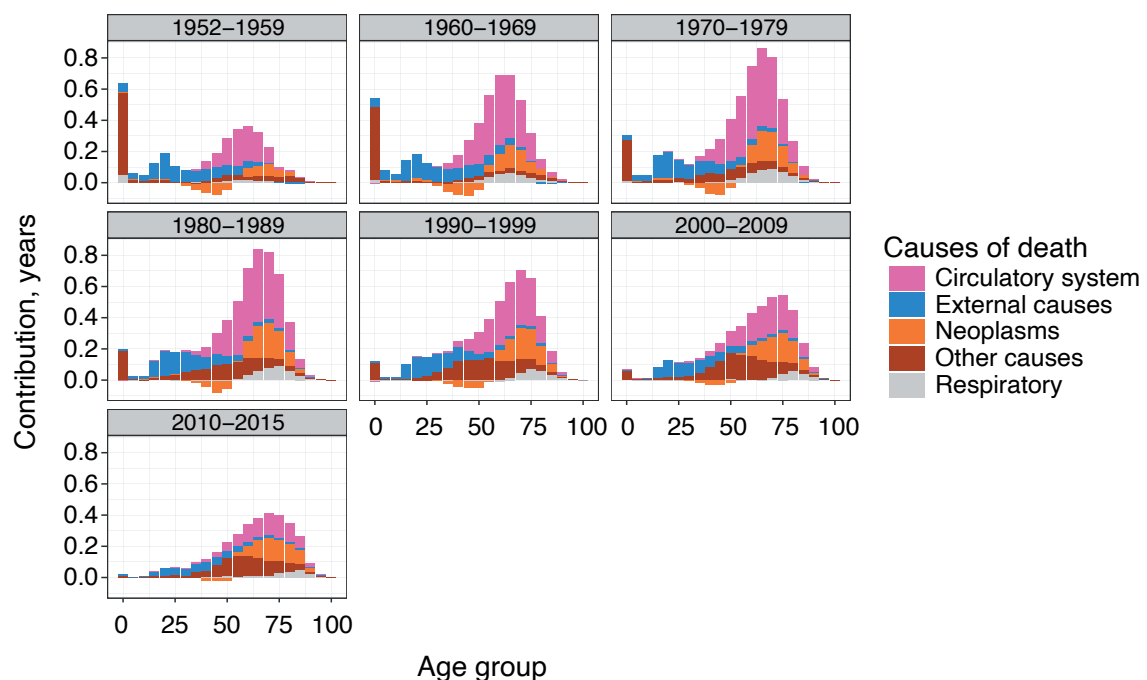


Figure 2 *Arriaga decomposition of period life expectancy by decade, age and cause of death for males and females in Denmark*



The decomposition plots peak around ages 60 to 70 where the mortality rates of males and females on an absolute scale is large but when there are still sufficient years of potential life left to live such that differences can arise. The right tail of the plots has very low values naturally as the number of years of life remaining is falling quickly for both sexes.

With respect to the effects of cardiovascular mortality on sex differences, in the 1940–1949 period for Utah, its contribution to the female advantage arises around age 50 and is the dominant cause of death for all subsequent ages except for age 90 or older. This general pattern persists for all periods though its absolute magnitude declines as the absolute difference in life expectancy between men and

women declines. It is likely that the substantially larger effects of cardiovascular mortality (pink), and to a smaller extent death attributable to respiratory diseases (grey), during the years spanning 1940 to 1990 coincides with high smoking prevalence rates among men compared to women. While smoking tobacco played a role, it would be less significant in Utah where tobacco usage is the lowest in the U.S. It is likely that the rising rates of unhealthy weight from the 1960's onward contributed to more cardiovascular deaths for men than women (Parikh et al., 2007).

For Denmark, the prominence of cardiovascular mortality is also present in the first (partial) decade (1952–1959) in accounting for large sex differences in life expectancy. This pattern continues to grow in prominence though its absolute magnitude declines in the 1990s as the sex differential in life expectancy declines here as in Utah. Denmark's and Utah's patterns are also similar in terms of the age at which cardiovascular mortality first contributes to the sex difference occurs, in their forties.

The contribution of neoplasms (i.e., cancer) to sex differences in life expectancy is dissimilar between Utah and Denmark. For Utah, cancers serve to *reduce* the female longevity advantage during the early adult and middle age years. This finding first arose in the 1940's and continued until the new millennium. Around age 60, male cancer mortality, like nearly all other causes, adds to the male deficit in life expectancy.

Denmark generally reveals a similar cancer pattern except the magnitude is considerably larger. Cancer again serves to subtract from the female advantage in life expectancy in the early and middle adult years but this penalty is larger than it is for Utah. After age 60, cancer mortality adds to the male disadvantage but these additions are larger and extend further into advanced ages in comparison to Utah.

The role of external causes of death, including accidents, homicides, and suicides, has considerable effects on life expectancy generally as these deaths tend to occur earlier in life but they also contribute to sex differences since this broad class of death arises disproportionately among males. While the effects of external causes of death of sex differences in life expectancy are present for all decades, it has an outsized effect starting around 1970 with more homicides among men being a major contributor (O'Brien, 2023). This phenomenon accelerated during the 1970's through the 1990's and then declined slightly. While homicides are an important component, the 1960's marked the rise of automobile ownership and access to firearms which led to more motor vehicle accidents and suicides, all of which affected males more than females. For Danes, where miles travelled by car are lower than in Utah and access to firearms is illegal (unless you are hunter), external causes of death consistently add to the male disadvantage where it is concentrated in the younger and middle ages. For the entire period, the largest contributor of most external causes of death in Denmark are male suicides and motor vehicles accidents.

The very large spike occurring during the first years of life reflect the well-known excess risk of infant and childhood mortality for males. Large contributors to this male excess arise from death due to birth defects, prematurity, and infections. For both populations, the magnitude of these early deaths toward the male disadvantage has declined considerably as infant mortality rates overall have declined in the latter half of the 20th century and into the 21st century.

5 DISCUSSION

Sex differences in life expectancy have long favored females but this advantage is not constant over time or across populations. In this paper, we have investigated the underpinnings of this differential as reflected in the contribution that specific causes of death make that give rise to the fact that females outlive males. The populations in question, Denmark and Utah in the United States, share commonalities as both are economically large Western societies (indeed, only certain governments collect and maintain cause of death information for their citizens for many decades) but there are also differences in terms of factors such as lifestyle, family size and access to health care. The years examined in these data also span numerous decades where public health and demographic shifts arise, as reflected in the use of tobacco, access to firearms, and fertility rates.

Despite the differences in the two populations, one of the main conclusions drawn from these analyses is the undeniable influence of cardiovascular mortality. It is fundamental to contributing to the almost impervious female life expectancy advantage in a similar fashion for both Utah and Denmark. Not surprisingly, as mortality from cardiovascular diseases is the leading cause of death in both locales,

this source of mortality is the largest contributor to the female advantage despite differences in the two populations and secular changes over the past 60 to 80 years (Hoyert, 2012). While rates of cardiovascular mortality have declined for both sexes, there have been persistently higher rates for men than women (Dimala et al., 2024; Haunsø et al., 2020). While heart and circulatory diseases have been responsive to public interventions (e.g. anti-tobacco ad campaigns, taxation on cigarettes) that have led to lower disease rates, our results nonetheless underline the strong biological forces that persistently increase the risk of cardiovascular mortality for men. Indeed, if this biological liability for men could be eliminated, it would likely do more to narrow the sex life expectancy gap. In a manner, this is happening already because cardiovascular mortality rates are declining for everyone, which means that the dominance of this source of female advantage is steadily diminishing.

While cardiovascular mortality is fundamental to understanding the dynamics of sex differences in the length of life, external causes of death are also central as they occur more often at younger ages where the years forfeited are greater. To the extent that males at younger ages take more risks, often starting around puberty (Owens, 2002), there are again biological bases for external causes of death as contributors to the male disadvantage. This large contribution of external causes is observed in both populations. Our analyses treat all external causes of death as a single category, a group that is quite heterogeneous that includes homicides, suicides and a number of unintentional causes (e.g., falls, fires and burns, drowning, poisoning by gases and vapors, and firearms). Nonetheless, what is striking is the similar contribution that external causes make for both Denmark and Utah in adding to the male disadvantage. This is noteworthy because the socio-environmental conditions are very different between the two populations in terms of drug and alcohol use, access to firearms, family size, and government support for health care (Pedersen et al., 2005). While the two sets of decomposition plots share similarities by time and age, it is the case that a more detailed analysis of specific types of external causes of death, which is beyond the scope here, should reveal how the different social contexts yield different types of accidental mortality.

The role of cancer in generating sex differences in life expectancy is different from the other leading causes. We have shown that during the reproductive years, cancer mortality serves to reduce the female advantage, a pattern found in both populations. As cancer is generally presented as a disease closely aligned with aging (Berben et al., 2021), some cancers are more lethal at younger ages such as breast cancer (Gnerlich et al., 2008).

The remarkable decline in infant and child deaths throughout the decades considered here not only served to elevate life expectancy over all but helped to diminish a large male penalty seen in the earlier decades. Indeed, the contribution of any infant cause of death toward a greater female advantage in life span has largely disappeared by the 21st century. Certainly more work is needed to further elucidate how mortality selection (or "culling") of more males at younger ages may serve to graduate more robust males to older ages where their mortality would be lower, and hence more similar to female mortality risks for many potential causes of death (Bruckner & Catalano, 2007; Griffin et al., 2018; van Dijk et al., 2019).

In sum, based on this comparison of Denmark and Utah, the longevity differential favoring females will likely continue but diminish in size. Gains in prevention and treatment of cardiovascular (and likely respiratory) diseases and cancer will contribute to the trend of increasing parity in life expectancy between the sexes. Additional social and public health policies will be needed to address persistent sex differences in external causes of death.

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