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AGE-AT-DEATH DISPARITY AND SEX GAP IN AVERAGE LENGTH OF LIFE AMONG THE *OLDEST-OLD* IN ITALY. AN ANALYSIS OF ITALIAN COHORTS BORN BETWEEN 1890 AND 1919

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ABSTRACT

The increase in life expectancy is generally associated with greater lifespan equality, but this is not always true, especially among the elderly, as seen in the case we are examining. This is what we show in this research through the reconstruction of a series of life tables for the oldest-old from the cohorts born between 1890 and 1919, starting from age 85. To date, only the 1890–1911 cohorts can be considered extinct, so the study of the sex-based mortality gap focuses on these cohorts, while all cohorts are considered in the study of age-at-death disparities. Both absolute and relative standard statistical measures were used as indicators, highlighting inconsistencies between absolute and relative measures in this final section of cohort life tables. Our findings reveal an evolution of mortality characterized by cohort effects, especially among older cohorts and to an overall decline in the mortality occurring in the context of two different ageing patterns for the two sexes. The outcomes not only show an increasing mortality male excess in, but also significant disparities in the average inter-individual differences in lifespan.

KEYWORDS: *1890-1919 cohort life tables, Gompertz model, average length of life after age 85, standard statistical measures of dispersion, sex gap in mortality, inter-individual inequality in length of life.*

INTRODUCTION

The life tables are the principal research instrument used to analyze the different risks of death by gender (Chiang, 1984) and allow us to examine the mortality conditions for men and women during a time interval. In demography, two types of life tables reflect different time interval characteristics: period (or calendar-year) life tables, and cohort (or generation) life tables. The first type, the period life table, is the best known and most frequently used: in Italy, for instance, this type of life table refers to the calendar year (Istat, 2002), thereby providing a general picture of the current mortality conditions and synthesis of all the age-specific death rates for the same year. A cohort life table, on the other hand, describes the whole mortality process for a given cohort or adjacent cohorts, again based on specific death rates, but over an extended time interval. A practical challenge arises because compiling all the data needed to construct such a table requires access to historical records spanning many years, as well as comprehensive and continuous statistical documentation.

These instruments may appear identical in structure but serve different purposes. Both can be used to identify differences in mortality, though from completely different perspectives: period life tables assume that current mortality conditions remain constant, although this hypothetical situation is updated annually; cohort life tables, on the other hand, describe the actual situation of each cohort and, as in our case, illustrate how mortality differences evolve over time.

To examine this aspect, we constructed the final sections of a series of cohort life tables from 1890 onwards, based on the period life tables published by the Italian National Institute of Statistics[1] - Istat starting with the table of 1975 (*demo.istat.it*) and extending to 2023. The segment considered focuses on of the *oldest-old*, namely those aged 85 and over. Age is crucial factor that has a massive impact on mortality and this age group is considered the most "fragile" since people at this age generally suffer from several degenerative diseases, creating a precarious balance between good health and poor health, autonomy and dependence. Their health also depends on the availability of resources, social support, and healthcare access. This age group faces a heightened risk of institutionalisation, hospitalisation and death (Meslé and Vallin, 2021).

According to recent population data in Italy (January 1, 2023), older adults (those aged 65 and over) accounted for 24% of the population, with the most fragile component of this subset – the *oldest-old* – constituted 15.8%, globally almost 2.25 million individuals Among them, men represented only one-third of this group.

When studying this particular age group, one must take into account key hypotheses and empirical analytical approaches regarding the long-term relationship between early-life conditions – experienced in childhood or even before birth – and mortality in old age. These conditions are known as "cohort factors".

There are other factors with long-term effects, known as "period factors", that have significantly influenced the evolution of mortality rates and greatly contributed to increased longevity. These include, for example, the role of the Italian National Healthcare Service (introduced in 1978) whose impact is clearly reflected in our results, just as, in this specific case, improved pensions as the result of the 1969 reform which also introduced a "social" old-age pension for all citizens aged over 65 without financial resources (Amato and Graziosi, 2013).

Differences between genders and within each gender regarding age at death give rise to a fundamental form of social inequality, which we aim to analyze using the indicators in these life tables. Our goal is to measure both the sex-based mortality gap and inter-individual inequality in lifespan. Among the indicators used are the mean remaining lifetime of each cohort (e^*_{85}) at age 85, along with its standard deviation (σ^*_{85}) and coefficient of variation (CV_{85}) – well known measures of dispersion. Additionally, we include the Gini coefficient as a life table function (G_{85}) – the index of concentration in the distribution of life spans – and the inter-quartile range (IQR_{85}), which is a a valuable measure of inequality in age at death (Meslé, 2001; Shkolnikov, Andreev, 2014). These statistical measures fall into two categories: absolute measures (standard deviation and inter-quartile range) and relat ive measures (coefficient of variation and Gini coefficient). While these measures are typically consistent when applied to complete tables in a time series context, using them for a final section of a cohort life table can yield conflicting results between absolute and relative measures – as observed in our case[2].

The analysis of e^*_{85}, σ^*_{85}, the coefficient of variation and the Gini coefficient is only feasible for extinct cohorts[3] (currently those born between 1890 and 1911). By adopting this time frame we can highlight the presence of the cohort effect (Bengtsson, 2019). Additionally, we perform a comparative analysis of e^*_{85} against the corresponding period life expectancy at age 85, e_{85}, from the period life tables.

[1] Also the Human Mortality Database (https://mortality.org) provides a series of cohort life tables for various countries, including Italy; however this source warns that the tables for the years up to 1906 may be inaccurate.
[2] See the conclusion in the following Section at this regard.
[3] A cohort is considered "extinct" when the number of survivors in the life tables is between 0 and 1, which generally happens after age 110 or 111, depending on whether a man or a woman.

Another key indicator, i.e. the inter-quartile range (IQR_{85}), plays a role in identifying changes in the mortality selection process; thanks to our methodology, we have been able to include a greater number of cohorts, extending up to the cohort born in 1919.

Because of these discrepancies between the two cohort groups, the results of e^*_{85} and the IQR_{85} had to be analyzed separately. In any case, our findings cannot be applied as a general rule to the earlier ages.

THE GENERAL MORTALITY CONTEXT AMONG 1890-1911 COHORTS

In this Section and the next one we examine the set of "extinct" cohorts since they are the only ones among all those considered for which we can actually analyze the sex gap in mortality.

Mathematical mortality models offer a valuable approach to examine general changes in mortality and we can understand initially this by means of the Gompertz's Law – the most common function used to describe oldest old mortality patterns (Olshansky and Carnes, 1997). The Gompertz model suggests an exponentially increasing mortality in early old age and characterizes the force of mortality $\mu(x)$ (for $x \geq 0$) (Chiang, 1984) at age x by two parameters

$$\mu(x) = \lambda e^{\gamma x} \qquad (1)$$

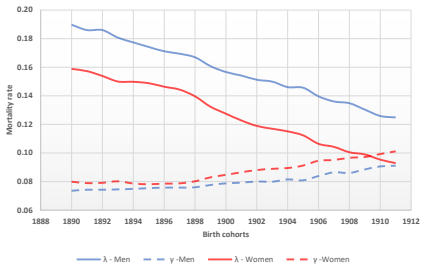
where $\mu(x)$ is known as instantaneous mortality rate. Here, $x \geq 85$, with $\lambda, \gamma > 0$; λ represents the mortality rate at age 85 (also called the scale parameter), while γ denotes the rate at which mortality increases with age, commonly referred to as the Gompertz slope.

The decline of initial mortality is unsurprising, as advancements in healthcare over the past 40 years have significantly improved health outcomes, even in advanced age. Figure 2.1 illustrates the pronounced male–female differences in their starting levels (λ), which, as demonstrated later, have played a key role in the widening gender gap in mortality.

Figure 2.1 also illustrates the so-called "inverse relationship" between initial death rate (λ) and mortality coefficient (γ), that is, in this case, high initial mortality is offset by a lower mortality coefficient (Gavrilov, L. A., 1984).

Regarding the role of gamma in equation (1), the Gompertz model effectively captures the mortality pattern across all ages for each women's cohorts. However, for men, this holds true only up to the 1904 cohort. In later cohorts, mortality rates between ages 99 and 104 increase at a slower pace than the expected exponential trajectory, after which a compensatory effect appears, likely due to a delayed increase in mortality at more advanced ages.

Figure 2.1: Scale parameter (λ) and slope (γ) values for the Gompertz model fitted* to the oldest segment (i. e., age 85+) of the 1890-1911 birth cohorts. Men and women



*We estimated the two parameters by fitting regression of log mortality hazards (instantaneous mortality rates) on age.
Source: Our calculations, based on Istat data.

These deviations from the exponential increase at very old ages can be seen as caused by stochastic effects, which play a significant role even when relatively few individuals contribute to mortality (Horiuchi & Coale, 1990). It is also important to consider Istat's observations regarding the final ages in life tables, specifically: *"Since deaths among individuals over 95 are rare"* (especially in the case of men), *"as we approach the threshold of extreme survival, we should rely on a demographic model capable of describing a regular trend in the probability of dying"* (Istat, 2002, p. 14).

Additionally, cohort heterogeneity can also explain some of the conflicting observations in late-life mortality trends. Just as mortality selection can cause an acceleration in death rates, it can also lead to deceleration, as mentioned earlier (Wrigley-Field, 2014; Steinsaltz and Wachter, 2006).

Given these complexities, our analysis focuses on age 85. Assuming that Gompertz's Law (1) holds, an alternative interpretation of γ is that it represents an inverse function of lifespan disparities (Zhang & Vaupel, 2009); thus, Figure 2.1 already suggests that the mortality sex gap is also characterized by greater disparities in men's age at death. Moreover, as

we will discuss further, male cohorts –particularly older ones – tend to be more heterogeneous than female cohorts in terms of educational background.

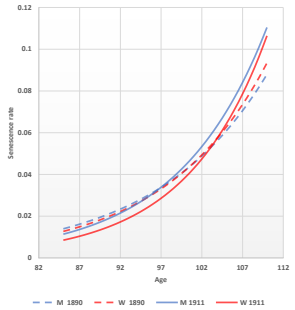
Aging and senescence are closely linked to accelerating mortality, and here too, the γ parameter plays a crucial role in constructing the so-called senescence rate η(x), which reflects the rate at which mortality accelerates with aging (Koopman *et al*., 2015). Since death rates measure the speed at which deaths occur within a given population or cohort over time, we derive equation (1) to determine the senescence rate:

$$\eta(x) = \frac{d\mu(x)}{dx} = \gamma \lambda e^{\gamma x} \qquad (2)$$

A single measure, such as the senescence rate, cannot fully capture the complexity of ageing and senescence, which involve a progressive increase in disease risk, functional impairment, and mortality. In our case, η(x) serves as a proxy indicator, providing a measure of aging by quantifying the increase in mortality rate per year of age in later life. Figure 2.2 illustrates the pattern of senescent mortality for the earliest and latest cohorts under observation.

In the earliest cohorts, the trajectories of η(x) were closely aligned and consistently crossed around age 100, with men exhibiting higher rates than women up to this age, but lower rates thereafter. In later cohorts, however, the gap between the sexes widened progressively, as depicted in Figure 2.2, and with higher rates of senescence towards age 100 in both sexes than in previous cohorts.

Figure 2.2: Senescence rates estimated from Gompertz model parameters; 1890 and 1911 cohorts. Men and women



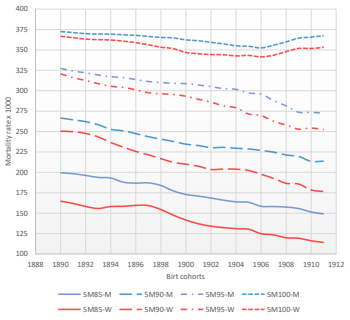
Source: Our calculations, based on Istat data.

In this context of persistent disparities, the following Sections examine mortality patterns among men and women, exploring how – despite undeniable progress in reducing mortality – these improvements have not been equally accessible to all. Both the sex-based mortality gap and inequalities in age at death remain significant challenges.

TRENDS IN IMPROVEMENTS IN LATE AGE MORTALITY OF OLDEST-OLD BY SEX WITHIN 1890-1911 COHORTS

An important step in this approach is the initial assessment of differences in age-specific death rates. To estimate the extent of the gender gap by age, we first analyzed the age-specific death rates (${}_5M_x$) for the following age groups: 85-89, 90-94, 95-99 and 100-104. The positive trend – especially in the 85–89, 90–94, and 95–99 age groups (Figure 3.1) – confirms that females have experienced greater improvements in survival compared to males; in fact, while the linear decreasing trends in ${}_5M_{85}$ of men and women is nearly parallel – aligning with the λ outcome of the model (Figure 2.1) – the reduction in female mortality becomes increasingly pronounced in the 90–94 and 95–99 age groups (Figure 3.1). However, in the case of ${}_5M_{100}$, the decline in mortality ceases with the 1906 cohort for both sexes. As mentioned earlier, late-life mortality trends can also result from variations in cohort robustness, particularly among those with a significant number of survivors at older ages and these differences in the rate of mortality decline between sexes are the main driver of the increasing divergence in age-at-death variability. It will therefore be interesting to track future developments in these historical data series, especially as demographic aging continues to increase the number of deaths at extreme ages.

Figure 3.1: Evolution of the death rates ($_5M_{85}$, $_5M_{90}$, $_5M_{95}$, $_5M_{100}$) of the *oldest-old* in the 1890–1910 cohorts. Men and women



Source: Our calculations, based on Istat data.

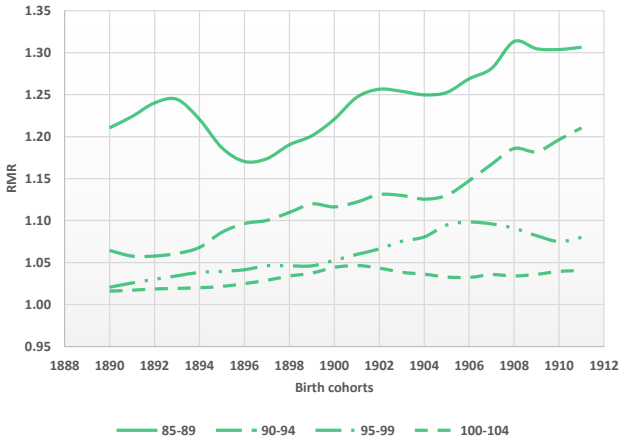
Figure 3.1 also reveals two distinct phases in the $_5M_{85}$ trend for the 85–89 age group: an initial decline, followed by a notable interruption for male cohorts born in 1896–1897 and female cohorts born in 1894–1897. Mortality rates then resume their decline – at an accelerated pace, particularly for women – without further interruptions (Figure 3.1).

These trends can be interpreted as a consequence of the impact of World War I (1915–1918) on the cohorts born between 1890 and 1898. Men born between 1874 and 1900 were conscripted in phases, though only the youngest served in active combat. This was especially true for the 1896–1898 cohorts, who were caught in the war's most devastating moments before the Armistice of Villa Giusti on November 3, 1918.

With approximately 5 million men enlisted**,** women were called upon to replace them in agriculture and industry. Like all civilians, women endured food rationing and declining purchasing power due to rising wartime taxation. Moreover, their access to healthcare was severely limited, as most able-bodied medical professionals were deployed to the front lines, and many modern medical treatments had yet to be introduced. This crisis was exacerbated by a series of natural disasters that began in 1915 (Schaumann, 1993; Maccheroni, 2016).

At this point, it is relevant to consider the evolutionary theory of aging (Williams, 1957), as mentioned in the introduction. According to this theory, individuals who experience hardship and adverse environments during childhood and young adulthood tend to have poorer health in old age, leading to higher late-life mortality rates (Horiuchi, 1983; Gurven & Fenelon, 2009). The decline in mortality observed in cohorts born after 1897 may thus reflect the disappearance of the adverse conditions that had initially disrupted the trend (Figure 3.1).

Figure 3.2: Ratio in Mortality Rates (RMR) for ages 85-89, 90-94, 95-99 and 100-104. 1890–1911 cohorts



Source: Our calculations, based on Istat data.

A measure of these sex gaps in mortality can be expressed through the Ratio of Excess Mortality (RMR) − in this case, the ratio between $_5M_x$ (Figure 3.2) for men and women. This ratio highlights the relative risk of death for men compared to women at different ages (Maccheroni, 2014).

As shown in Figure 3.2, RMR trends increase across successive cohorts for the 85–89 and 90–94 age groups and up to the 1906 cohort for ages 95–99. As we shall see, women's greater progress in reducing ${}_5M_{85}$ and ${}_5M_{90}$ has been the main driver behind improvements in their median age at death – the age at which 50% of a birth cohort is expected to survive.

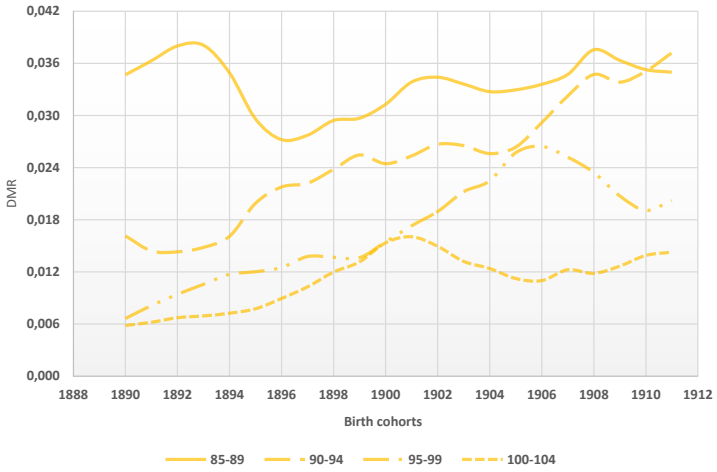
Moreover, applying Pressat's (1985) comparative approach – which measures the contribution of survival improvements at different ages to increases in e^*_{85}– reveals that for men, 95.5% of these gains came from survival improvements between ages 85 and 94, while for women, this figure was 85.7% and the remaining 14.3% among the subsequent age groups.

More recently, an alternative approach has been proposed: the Difference in Mortality Rates (DMR), which measures the absolute difference between male and female death rate**s** (Wisser & Vaupel, 2014). Unlike RMR, which highlights which sex benefited less from mortality improvements, DMR (Figure 3.3) is not affected by general mortality decline but instead reflects the presence of specific risk factors. Comparing these two indicators can therefore provide insights into the underlying causes of mortality differences.

Since the cohorts analyzed were born over a century ago, our assessment is inevitably limited to general mortality rates (${}_5M_x$) rather than cause-specific mortality (Figure 3.3). However, we observe strikingly similar trends in historical RMR and DMR patterns across age groups (Figures 3.2 and 3.3), with Pearson correlation coefficients of 0.73 for ages 85–89 and $r > 0.99$ for the subsequent three age groups.

The impact of World War I on male mortality is more pronounced in the DMR profile for the 85–89 age group. Both RMR and DMR reach an initial peak in the 1893 cohort, followed by a low point in the 1896 cohort, before rising again in the 1900 cohort due to the faster decline in female mortality compared to male mortality (Figures 3.2 and 3.3).

Figure 3.3: Difference between Mortality Rates (DMR) for ages 85-89, 90-94, 95-99 and 100-104. 1890–1911 cohorts



Source: Our calculations, based on Istat data.

All these findings confirm that aging trajectories differ significantly by sex, leading to substantial effects on both mean remaining lifetime at age 85 (e*85), longevity, and age-at-death variability.

THE AVERAGE LENGTH OF LIFE AFTER AGE 85 FOR COHORTS BORN IN 1890–1911, COMPARISONS WITH PERIOD LIFE EXPECTANCY. COMPARISON OF INDICES OF VARIABILITY: σ^*_{85} ,CV_{85} , G_{85}.

Period life expectancy at birth (e_0) is the most widely used indicator of current mortality conditions in a country; indeed, it is a common practice to synthesize the mortality evolution based on the e_0 general trend. In recent years period life expectancy in old age has gained greatly, especially e_{65}: this is now one of the benchmarks for all modern pension systems (Maccheroni, 2012; Palmer, 2019), given that the increasing proportion of older people in modern society and their longer lifespan are having an bigger a bigger effect in the economy of the welfare state. However, mortality trends are now more strongly influenced by gains and losses in old-age survival.

In our case, the life tables used for this study capture the complete mortality conditions for each cohort throughout their lifespan. and e^*_{85}, the mean remaining lifetime at age 85, indicates the average time that the cohort went "extinct"; this historical analysis therefore synthesizes the evolution of the aging process in this age group.

The biometric function d_x (i.e. the number of deaths between the ages x and x+1 in the cohort life table) has been used to calculate e_{85}*; for every extinct cohort, we can use these data to construct the age-at-death distribution after 85 years (X_{85}), and then find the distribution of remaining lifetime (Z_{85}) through variable transformation:

$$Z_{85} = X_{85} - 85 \quad (3)$$

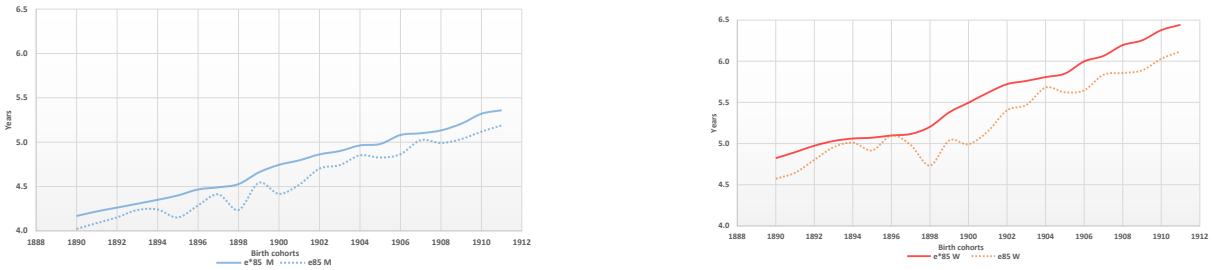
where e^*_{85} represents the mean of Z_{85} and $Var(Z_{85})$ denotes its variance (2). Since Z_{85} is the result of a linear transformation of X_{85}, it has the same dispersion as X_{85} (Cifarelli, 2003) so that:

$$Var(Z_{85}) = Var(X_{85}) \quad (4)$$

In other words, not only do both distributions have the same variance and therefore the same standard deviation, as well as the same inter-quartile range (IQR), which is also a measure of dispersion and so we focus on both X_{85} and Z_{85}.

Figure. 4.1: Mean remaining lifetime at age 85 years (e^*_{85}) of the 1890–1911 cohorts and the corresponding period life expectancy (e_{85}) on the basis of the current life tables (1975 to 1995) when those born between 1890 and 1911 reach the age of 85 years. Men and Women

Men Women



Source: Our calculations, based on Istat data.

Figure 4.1 shows there were two separate phases in e^*_{85} growth: first, a slow increase for the 1890–1897 cohorts, and then a much faster increase for those born between 1898 and 1911 (Figure 4.1); in relative terms, e^*_{85} for men increased by 12% among the first cohorts, and by 17.5% among the later ones; in contrast, the increases for women were 6% and 23% respectively. These more recent mortality dynamics have been driven by environmental changes in behavioral and lifestyle factors resulting from the "Italian miracle" of the 1960s.

However, women had higher initial e^*_{85} values, and after the 1897 cohort, the sex gap widened significantly. Initially stable at around 8 months, this gap grew so that by the 1911 cohort, women's mean remaining lifetime exceeded that of men by 13 months.

This difference may appear modest, but it took twelve years for the first cohort of men (born in 1902) to reach the same e^*_{85} level as women born in 1890. Notably, even after age 85, remaining life expectancy (e^*_{85} for x = 86, 87, … 104) continued to increase, although at a slower rate with advancing age. Sex-based differences were most pronounced in the 90–94 age group, consistent with previous findings on mortality rate comparisons.

We can also make a more current comparison – between e^*_{85} and e_{85} – using the data in Figure 4.1. We find that each cohort has actually lived longer than envisaged by the period life tables from 1975 to 1995 for the 1890–1911 cohorts on turning 85, and the life expectancy tends to increase as e^*_{85} increases (Canudas-Romo and Schoen, 2005). It is worth noting, however, is the fact that the relative increase of e^*_{85} for the 1911 cohort compared to that for the 1890 cohort showed the same magnitude as the increase in e_{85} between 1975 and 1995: approximately 28% for men and 32% for women.

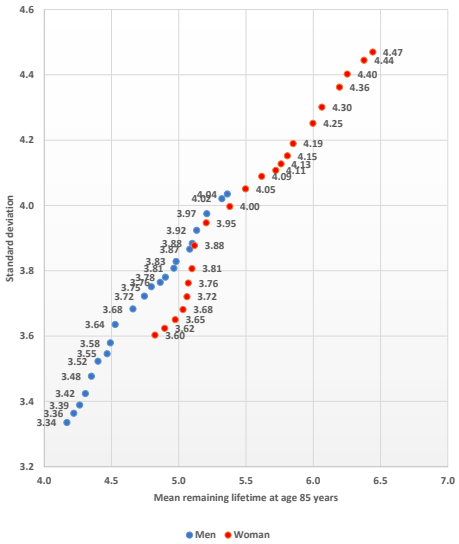
The evolution profiles of these two indicators are, nonetheless, quite different. Given that the levels of e_{85} are influenced by current mortality conditions, they often fluctuate, as can be seen in Figure 4.1. More specifically: negative health events affecting the entire population regardless of age or within a certain age group (and so involving several cohorts at the same time) will cause a general rise in mortality, although the intensity of such a rise will vary with age and health conditions. It follows that period life expectancy e_{85} decreases; following this selection process, mortality rates will fall down and e_{85} will rise.

Indeed, Figure 4.1 clearly shows a sharp decline in e_{85} in 1983, particularly among women (5% decrease for women, 3.9% for men), due to a heatwave that caused a substantial increase in mortality, primarily attributable to cardiovascular and cerebrovascular diseases (Centers for Disease Control and Prevention (CDC), 1984; Istat, 2011). This sex-based mortality gap is also due to the fact that women, who generally live longer, are more prone to chronic diseases and

disabilities. They tend to be more fragile, so when an adverse event occurs, the resulting mortality increase is more pronounced for women**.**

On the other hand, the e^*_{85} trend does not reflect the severe impact of the 1983 heatwave on mortality risk. For males, the e^*_{85} of the 1898 cohort remained largely unchanged, aligning with the levels of the 1897 cohort, while for females**,** there was a slight increase (Figure 4.1). This is because the consequences of the 1983 event were only reflected in a single year for each affected cohort in this event, i.e. with increases in mortality in the 1890 cohort at the age of 93, in the 1891 cohort at the age of 92 and so on up to the 1898 cohort at the age of 85.

Figure. 4.2: Standard deviation versus average length of life after age 85 for the 1890–1911 cohorts. Men and Women

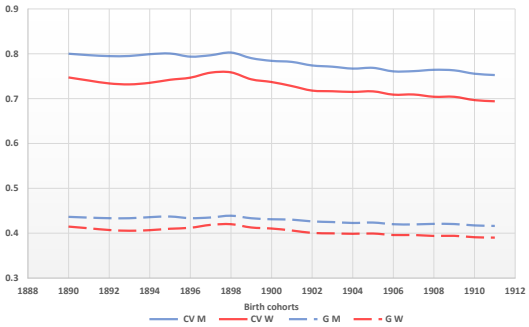


Source: Our calculations, based on Istat data.

As already mentioned, there was a steady increase in the cohort's average length of life from one cohort to the next and it is often said that the higher the level of life expectancy, the lower the level of inequalities in age at death. This observation was made on the basis of numerous studies (Shkolnikov, Andreev, 2014), though standard statistical measures of variability – such as standard deviation (σ) and inter-quartile range (IQR) which are absolute measures – but applied to a final section of a life table do not always provide these results.

For these now extinct cohorts, lifespan disparity has been measured using standard deviation (σ^*_{85}) of Z_{85} (4). The results are shown in Figure 4.2, where the trends of σ^*_{85} are expressed as a function of e^*_{85}, and it is evident that these indices (i. e. e^*_{85} and σ^*_{85}) are closely related to each other: the Pearson correlation coefficient is, in fact, 0.99 for both men and women. Now, if we compare the standard deviations of males and females for each cohort, we notice greater disparity in women's age at death, as they exhibited more significant mortality improvements compared to men. However, if we consider the σ^*_{85} values for both sexes at the same average length of life (Figure 4.2), we reach the opposite conclusion. These latest results thus confirm the importance of the differences between e^*_{85} for the two sexes. If we standardise (Baudish, 2011) the remaining lifetime distribution Z_{85} (3), and denote the standardized version as $Z_{s,85}$ (where $Z_{s,85} = X_{85}/e^*_{85}$), we obtain the standard deviation of $Z_{s,85}$, which corresponds to the coefficient of variation (CV_{85}; $0 \leq CV_{85} \leq 1$) of Z_{85}.

Figure 4.3: Evolution of the coefficient of variation (CV_{85}) and Gini coefficient (G_{85}) for the 1890–1911 cohorts. Men and Women



Source: Our calculations, based on Istat data.

Not only that, but if we also consider the Gini coefficient (G_{85}; $0 \leq G_{85} \leq 1$) as a life-table dispersion measure (Shkolnikov, Andreev, 2014), and examine this index alongside the coefficient of variation (CV_{85}; $0 \leq CV_{85} \leq 1$), we find that (Figure 4.3) both indices are higher for males than for females. However, while they share the same general trend, they differ magnitude (Figure 4.3), suggesting that the sex-specific trends in lifespan inequality follow similar general patterns.

The differentiated process of delaying older deaths resulted in a decreasing concentration of ages at death and G, also known as the concentration index, highlighted this reduction starting from the 1898 cohorts (Figure 4.3). The G_{85} profiles and gender differences are influenced by the varying concentrations of deaths around the modal age of death, which, notably, shifts to older ages across generations only in the case of women; this will be explored further in the next Section.

EVOLUTION OF AGE-AT-DEATH DISPERSION IN THE 1890–1919 COHORTS ACCORDING TO THE RESULT OF THE COHORT LIFE TABLE INTER-QUARTILE RANGE

This index has been used in various applications, mainly for studying the rectangularization process of the survival curve (Nusselder, Mackenbach, 1996) and, as a consequence, the variability in age at death (Ouellette N. and R. Bourbeau, 2011). These applications have typically relied on period life tables and have primarily focused on the entire lifespan; therefore, direct comparisons with our findings are not possible.

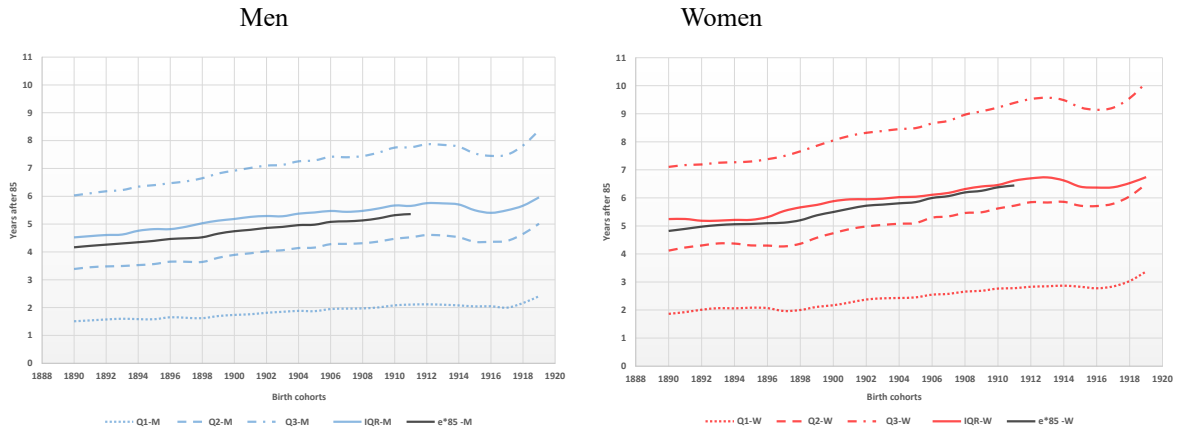
The life table inter-quartile range (IQR) has also been recognized as an absolute measure of disparity in lifespan (Shkolnikov, Andreev, 2014) and is calculated on quartiles – measures used to describe a dataset structured according to a statistical distribution. More specifically, quartiles summarize the distribution using three values, Q_1, Q_2 and Q_3, which divide the dataset into four equally sized intervals. Since we can calculate the IQR for more recent cohorts beyond the extinct generations, the available dataset is larger: we have, in fact, extended our analysis to the cohort born in 1919, which means that our analysis can cover a span of 30 generations.

To maintain the continuity with the results of e^*_{85}, we construct IQR_{85} using the distribution Z_{85} (3) with quartiles obtained from its cumulative frequency. Therefore, the IQR is:

$$IQR = Q_3 - Q_1 \qquad (5)$$

where Q_1, the first quartile, represents the value of Z_{85} below which 25% of the remaining lifetimes fall, while 75% exceed it. Likewise, Q_3, the third quartile, marks the value of Z_{85} below which 75% of the remaining lifetimes fall and 25% exceed it. Between these two quartiles is Q_2, the second quartile, or the median age at death. The IQR captures the central interval of this distribution, containing 50% of the remaining lifetime or ages-at-death for cohort members – namely those closest to the median, The latter also indicates the time span within which a cohort halves. As previously observed with e^*_{85}, this sex-based gap has widened across cohorts: whereas in the 1890 cohort the median lifespan of females was just over 10 months longer, by the 1919 cohort, this difference has reached almost a year and a half more.

Figure 5.1: Quartiles (Q_1, Q_2 and Q_3), inter-quartile range (IQR) for the 1890–1919 cohorts, and average length of life (e^*_{85}) of the 1890–1910 cohorts. Men and Women



Source: Our calculations, based on Istat data.

Given our understanding of the relationship between Z_{85} and X_{85}, we can graphically compare IQR_{85} with the quartiles and e^*_{85} (Figure 5.1). As shown in the figure, IQR_{85} has been gradually increasing due to the increase of all quartiles, particularly Q_3, which – in both sexes – has grown, on average, twice as fast as Q_1. However, for women, Q_3 starts at a much higher age, causing the gap between the two IQRs to progressively widen.

For cohorts born between 1890 and 1911, Figure 5.1 also shows that the increasing trends of IQR_{85}, e^*_{85} and Q_2 are largely parallel for men. In contrast, for women, the trends of e^*_{85} and IQR_{85} tend to converge, with the e^*_{85} of the latest cohorts flattening closer to IQR_{85}. In both cases, the variability in the distribution of ages at death, as measured by IQR_{85}, increased as mortality declined – more so for women than for men. Specifically, for men, IQR_{85} and e^*_{85} increased at nearly the same rate from one cohort to the next, whereas for women, e^*_{85} sometimes grew even faster. This direct relationship between IQR_{85} and e^*_{85} brings us back to the considerations in the previous Section on absolute and relative measures. It is the relative measures that we will use when assessing disparities in age at death.

Additionally, as recently pointed out, IQR may have a certain limitation: "it is only sensitive to transfers between quartiles and not to transfers within quartiles" (van Raalte & Caswell, p. 13, 2013). This issue arises in some contiguous female cohorts due to a shift in the modal age of the age-at-death distribution. To address this**,** we will focus on variations in the average remaining lifetime at the first and last quartiles.

The trends of IQR_{85} (Figure 5.1) reflect changes in the profiles of male and female age-at-death distributions, resulting from the two distinct aging patterns of mortality decline. As anticipated in Section 3, this is particularly evident in mortality rates for ages 85–89 and 90–94 (Figure 3.1). For men, the profile of the distribution of X_{85} remains unchanged, with the mode staying around the age of 85 until the 1916 cohort, after which it fluctuates between 85 and 86 years. For women, however, the mode serves as an indicator that anticipates the era of longevity extension for these cohorts. It shifts from the initial age group to the next in the 1894 cohort, then to 87 years in the 1911 cohort, and finally to the 88–89 age group in later cohorts.

This shift had significant repercussions on the mean remaining lifetime at both the lower (Q_1) and upper (Q_3) quartile limits for men and women. It is therefore interesting to examine to what extent this longevity advantage is accessible to all individuals. In the case of the 85 year old people in the 1890 cohort, whose age at death was less than or equal to Q_1 – i.e. the most fragile component within their cohort –, the maximum age gained was 1 year and 6 months for men (36% of e^*_{85}) and 1 year and 10 months for women (39% of e^*_{85}). By the 1911 cohort, these values increased to 2 years and 1 month (39% of e^*_{85}) for men and 2 years and 7 months (43% of e^*_{85}) for women.

Over the years, a combination of factors has gradually improved living standards and survival conditions for the oldest-old, particularly the most vulnerable. However, these improvements have primarily benefited those who already had a lower risk of dying. By the time members of the 1890 cohort reached the age corresponding to Q3, they had already lived at least 6 years (men) and 7 years and 1 month (women) – four times as long for men and 3.8 times longer for women compared to earlier ages. For the 1911 cohort, these values rose to 7 years and 9 months for men and 9 years and 5 months for women (3.8 times longer for men, 3.2 for women).

Moreover, the mean remaining lifetime at the third quartile has continued to rise, though there are signs of a slowdown starting from the 1902 cohort – particularly for men (Gavrilov & Gavrilova, 2019). These findings further support previous evidence that the disparity in men's age-at-death and the male-female mortality gap has increased.

Furthermore, Figure 5.1 shows a temporary stagnation in the IQR trend, as well as in all quartiles, for the 1912 cohort (men) and the 1913 cohort (women). This is followed by a steep decline until the 1916 and 1917 cohorts, respectively. This apparent reversal is due to an increase in death rates caused by period-specific conditions and a possible cohort effect.

These cohorts experienced severe external shocks. Specifically, they lived through the devastating heat wave that struck Italy in the summer of 2003, which had a tragic impact on the health of the elderly (D'ippoliti *et al*., 2010), affecting an increasingly younger age group: 91 years for those born in 1912 to 86 years for those born in 1917. Additionally, another surge in mortality occurred in 2015 among the 1905–1917 cohorts (aged 98-110), linked to a severe flu season, exacerbated by a dramatic decline in vaccination rates during autumn and winter (Istat[b], 2020).

Another possible cause for the rise in mortality seen in the 1916–1917 cohorts is the "fetal origins hypothesis" (Barker *et al*., 1993; Barker and Lackland 2002), which aligns with the evolutionary theory of aging mentioned above. According to the "fetal origins hypothesis", malnutrition in the womb during late pregnancy and in very early infancy may increase the risk of death in adulthood and old age. More broadly, this perspective suggests that the aging process itself might be "programmed" by events during childhood (Sayer *et al.*, 1998). What we can conclude here is that many of those born in 1916–1917 endured severe hardship. The year 1917 was not only a pivotal year for the outcome of World War I but also the worst year in living conditions and food shortages for the civilian population.

CONCLUDING REMARKS

The discussion of the oldest-old born over a century ago naturally requires consideration of various historical factors that may have affected the evolution of their mortality – commonly referred to as "cohort factors". Regarding the long-term negative impact of the First World War on our earliest cohorts, the results provided by e^*_{85} and the quartiles (Figures 3.1 and 4.1) do not necessarily indicate a direct causal link to this event, although they align with patterns observed in other countries involved in the war (Horiuchi, 1983: Horiuchi and Coale, 1990). The cohort effect is actually the outcome of multiple interacting factors. For older cohorts in particular, aging was often perceived as an inevitable decline, shaped by a cyclical view of time and life typical of traditional societies. Moreover many of these either did not benefit from the support of a National Health Service or only received partial assistance**,** forcing them to endure both the long-term effects of past hardships and the diseases associated with old age.

The debate also remains open regarding the negative impact in old age of conditions experienced at birth – commonly referred to as the "fetal origins hypothesis"; a notable example of this ongoing discussion is the conflicting opinions (Doblhammer et al., 2011) surrounding the study of the Great Finnish Famine of 1866–1868 (Kannisto et al., 1997), which questioned the validity of the fetal origins hypothesis. However, this theory was later supported in the case of individuals exposed to the prolonged siege of Leningrad from 1941 to 1944 (Sparén et al., 2004). This suggests a stronger mortality gradient during specific periods of life, whereby individuals who experienced poor early-life conditions were later subject to higher-than-average mortality. Consequently, if the primary drivers of mortality trends – particularly in older age — were (and still are) linked to specific historical periods (Kannisto, 1996), then it would appear that death rates in period life tables do not always exclusively reflect contemporary conditions.

The set of extinct cohorts (1890–1911) reached old age between the late 1970s and the 1990s, a period characterized by significant economic, social, and medical advancements, as well as improvements in health and safety standards (Istat, 2016). As these older cohorts passed, the societal perception of aging gradually evolved. Little by little the image of old age changes as the older cohorts pass. Often dictated by the cultural and economic backwardness of the country, this image in people's memory and experience is gradually being replaced by new models adopted by more and more people in subsequent cohorts, thus leading to genuine social transformation. Since the 1980s, the steady rise in the number of the oldest-old has also contributed to their growing importance in social and economic terms (European Commission, 2018; Osservatorio Sulla Spesa Pubblica E Sulle Entrate, 2020) and has created a growing interest in aging not just from a biomedical perspective, but also from a psychosocial point of view, or both; the view of longevity has dramatically changed and the emphasis is moved from "successful aging" to "active aging" (Vergani, 1997; Istat[a], 2020). The most recent cohorts within our study have begun to reap the benefits of health improvements as they age, leading to a decline in mortality – although this trend remains gender-dependent and varies within each sex, as previously noted.

In this regard, the only available data that offer at least partial insight into the social conditions of the *oldest-old* come from the Italian census records for 1971, 1981, 1991 and 2001, which provide details on educational attainment. In our case the age-disaggregated data refer to the 75-84 age group, (i. e. the 1890, 1900, 1910 and 1920 cohorts), who were 81 years old at the time of each respective census – an age close to the 85 thresholds used in our analysis. As shown in Table 5.1, educational levels among these cohorts were generally very low, putting many of the oldest-old at significant risk of social exclusion. In fact, a substantial portion of the population had little to no formal education, particularly among women: in the 1890 cohort, over 50% of women lacked any schooling, while for all subsequent cohorts, the most common level of education attained was primary school.

The significant differences in the type of education received by women and men (Table 5.1) – especially in higher educational levels – stem from traditional understanding of gender roles. These differences are just one aspect – albeit a partial one – of the profound social heterogeneity of these cohorts, which has also contributed to the rise in social differentials in mortality. Although this cultural gap among sexes has begun to narrow in more recent cohorts, these results demonstrate (Table 5.1) – even if indirectly – that men have historically enjoyed a better social status and higher economic power within society at the time (Istat, 2016 ; Chen *et al*, 2021). This was partly because there was a longstanding lack of legal reforms aimed at restructuring social relations between the sexes.

Family care, informal care, intergenerational solidarity and, more broadly, the structure of social relationships and networks also played a decisive role in the lives of these people. There is no doubt, however, that higher education and income increased access and use of health care, as well as enabled better lifestyle choices, all of which contributed to a better quality of life and reduced vulnerability to risk factors (Cutler and Lleras-Muney, 2010). However, this special social advantage for men was limited to only a small number in the older cohorts and by a distinct minority thereafter (Table 5.1). Paradoxically, although women have historically exhibited higher overall disease rates than men, they have also consistently shown lower mortality rates (Colchero *et al.* 2016).

Table 5.1: Population aged 75-84 years, by educational qualification and gender. Italian census records for 1971, 1981, 1991 and 2001. Percentage data

Educational qualification	1971	1981	1991	2001
			Men	
No title	43.3	40.9	36.4	20.8
Primary school diploma	45.9	45.6	47.1	50.8
Lower secondary school diploma	5.1	7.0	9.5	7.0
High school diploma	3.3	3.8	4.1	12.9
University degree	2.4	2.7	2.9	8.4
Total	100.0	100.0	100.0	100.0
Entropy (*relative disparity index)*	*0.667*	*0.704*	*0.732*	*0.827*
			Women	
No title	51.2	42.3	41.8	30.8
Primary school diploma	42.4	47.1	46.6	51.2
Lower secondary school diploma	3.6	5.5	8.2	3.2
High school diploma	2.6	4.5	2.9	8.7
University degree	0.2	0.6	0.4	6.0
Total	100.0	100.0	100.0	100.0
Entropy (*relative disparity index)*	*0.580*	*0.652*	*0.654*	*0.744*

Source: Our calculations, based on Istat data. (H. Theil, Economics and Information Theory, 1967).

Regarding women in these cohorts, although there were notable local differences across Italy, their family responsibilities and broader social roles generally served to protect them from typically male risk factors, such as physically demanding workloads, alcoholism and smoking, and their associated consequences (Diderichsen *et al.*, 2009; Rogers *et al.*, 2010). This protective effect also enabled many women to gain greater awareness changing lifestyles[4] and to generally adopt better health behavior. The earlier rise in the modal age-at-death for women, already evident in the 1894 cohort, compared to the relatively stagnant trend for men, indicates a different aging process.

Over time, however, bot lifestyles and the prevalence of risk factors have changed, with clear distinctions between the two sexes. A relatively recent study (Beltrán-Sánchez *et al*., 2015) examines the emergence of mortality differences using data from Japan, New Zealand, the USA and five European countries (not including Italy). Beyond smoking – a major factor responsible for excess deaths among men – socio-economic transformations at the turn of the 20th century may have further undermined men's ability to manage their health, potentially heightening their biological vulnerability.
Various other studies have emphasized the influence of genetic factors and hereditary longevity investigating how the distinct genetic makeup of men and women contribute to more deaths among men (Crimmins et al., 2019; Wisser and Vaupel, 2014). However, such genetic influences appear to be relatively minor.

On one hand no policy can change the biological makeup of the population, on the other hand the sex gap in mortality and age-at-death disparities do not seem to be perceived as problems in many social and political fields, partly because they are often masked by population heterogeneity. Interventions in social factors and behavioral characteristics that are the key factors to solving those instances of social inequality are therefore not yet adequate.

[4] However, the results we have seen in the precedent Sections call to mind those written by Vaupel (2010, p. 537) : …" Mortality is by far the most readily and reliably measured index of health."

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