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Research Article

Mortality shifts and mortality compression in period and cohort life tables

Nico Keilman

Contents

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Nico Keilman[1]

Abstract

BACKGROUND
When age-specific mortality falls, period life tables give a distorted view of the life expectancy (LE) and the degree of mortality compression in birth cohorts.

OBJECTIVE
To derive mathematical expressions for the link between LEs and compression in period life tables on the one hand and corresponding variables in birth cohorts on the other hand.

METHODS
We analyse the age at death distribution (AADD) computed from the life table's $d(x)$-column. We derive general expressions for the moments of this distribution in a series of annual period life tables, written as functions of the moments in the AADD of cohorts.

RESULTS
We use data for Norwegian men and women to illustrate simple versions of the new expressions. The LE increases twice as fast across cohorts compared to what period life tables suggest under this model. Compression in Norwegian mortality, expressed in terms of decreasing variance of the AADDs, is approximately 40% slower in period than in cohort mortality.

CONCLUSIONS
We show how one can analyse the amount of distortion in period LEs compared to cohort LEs. In addition, we show how period compression is determined by cohort compression, together with both period and cohort LEs.

CONTRIBUTION
We derive new expressions that link the period AADD to the cohort AADD. Under a simple linear model, we show why compression in the period AADD often goes together with increases in the period LE.

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1. Introduction

Mortality trends in a certain country are often characterized using the development of the life expectancy (LE) at birth over time. For a given year, the period LE reflects the expected life length of a newborn child if age-specific mortality rates would remain constant for many years into the future. It relates to the behaviour of many different birth cohorts during just one calendar year. However, this is a hypothetical situation, and real people do not live this way. They are members of only one birth cohort, and most of them live their lives during many years. When mortality changes over time, the period LE gives a distorted view of longevity in birth cohorts, i.e., cohort LE. The aim of this paper is to derive and illustrate mathematical expressions that link period measures for the mean length of life and for its variance to mortality measures of real cohorts. These expressions help us to understand why, for instance, compression of period mortality around the mean age often goes together with an increase in the period LE.

When age-specific mortality is falling, LE may increase faster than period life tables suggest. For instance, 'best practice' cohort LEs for women born between 1870 and 1920 increased by 0.43 years of age per calendar year (Shkolnikov et al. 2011) – almost twice as fast as the improvement in best practice period LE for women since 1840 (0.24 years of age per calendar year). Here, best practice LE refers to the maximum LE observed among national populations in a given year or for a given birth cohort. Wilmoth (2005) shows that LEs of Swedish cohorts born in 1880 and later increase faster than period LEs do after that year. Stoeldraijer, Van Duin, and Janssen (2012) use data from the mortality forecasts computed by Statistics Netherlands and note the same effect in Dutch birth cohorts born between 1900 and 1960. Goldstein and Wachter (2006) use estimates and projections of female LE from Sweden and the United States. They find that period LEs are approximately equal to cohort LEs for cohorts born 40–50 years earlier. They note also that this so-called LE lag (the number of years it takes for a period LE to reach the current level of the cohort LE) lengthens as mortality improves. The LE gap (the difference between the LE for a cohort born in a particular year, and the period LE for that year) has risen and then fallen over time. Canudas-Romo and Schoen (2005) analyse the Siler model of age-specific mortality combined with constant rates of mortality decline and find qualitatively similar effects for the lag. Gaps were about one-ninth to one-tenth of the lags. Missov and Lenart (2011) assume a Gompertz model for age-specific mortality and the same yearly rate of improvement in mortality at all ages. Under these conditions, the temporal change in period LE is approximately proportional to the change in cohort LE. If period LE improves by two years of age per decade, cohort LE would improve by 2.5 years per decade. Wilmoth (2005) assumes that distributions of deaths by age change over time in

accordance with a linear shift model. Under this model, he also finds that cohort LEs increase faster than period LEs. Like Missov and Lenart, he establishes a simple linear relationship between period and cohort LEs.

One other consequence of the distorted view that period life tables give in times of changing mortality concerns the compression of mortality around the mean age or the modal age of death. Ouellette and Bourbeau (2011) show an ongoing process of mortality compression in Canada, France, Japan, and the United States. Deaths tend to be progressively concentrated near the mean or the modal age at death. Janssen and de Beer (2019) analyse period mortality in 26 European countries, Japan, and the United States since 1950. They find a transition from a period in which LE increased mainly caused by mortality compression, to a period with gains in LE mainly due to mortality delay. Women in the United States and in Northern and Western Europe experienced the transition early, i.e., between 1950 and 1970. Generally, the transition occurred among men about ten years later than among women. Canudas-Romo (2008) studies the period standard deviation from the modal age at death for the Siler model and the Gompertz model. The period standard deviation is constant under the Gompertz model but falls regularly under the Siler model. Ediev (2013) discusses the theoretical link between period and cohort compression measures. He concludes that mortality compression in periods may very well go together with mortality expansion in cohorts. Tuljapurkar and Edwards (2011) show that the variance in the age at death is inversely related to the Gompertz slope of log mortality.

The contribution of this paper is twofold. First, it is complementary to work that studied the link between period and cohort LEs using purely analytical models for mortality dynamics (Canudas-Romo and Schoen 2005; Missov and Lenart 2011; Wilmoth 2005). Instead, we assume an empirical relationship between period and cohort probabilities. Second, we derive new expressions that link the period age at death distribution (abbreviated as AADD henceforth) to the cohort AADD. These can be used for all moments of these distributions, although we analyse only the first and the second moment. One important result is that under a simple linear model, we show why compression in the period AADD often goes together with increases in the period LE.

We focus on the statistical distribution of the age at death. This is the column in the life table conventionally expressed as $d[x]$, which becomes a proper distribution when the life table radix $l[0]$ is set to one. For a given age x, $d[x]$ is the unconditional probability that a newborn child will die at that age, given the mortality regime underlying the life table. We write $d[x,t]$ with $x = 0,1,2,\ldots$ for the AADD in the period life table of year t. Similarly, for varying values of x, $\delta[x,g+x]$ is the AADD for the cohort born in year g, where $g = t - x$. The advantage of focusing on the AADD is that its first two moments have a straightforward interpretation. The first moment is the LE,

while the second moment reflects variation in the distribution around the mean/LE (not around the modal age at death, as in some prior analyses). Hence, one may use the second moment in analyses of mortality expansion and compression.

We use an empirical regularity to link cohort probabilities $\delta[x,t]$ to period probabilities $d[x,t]$. Then we derive expressions for the first moment of the period AADD, which is the period LE. Next, the variation across the lifespan, reflected in the second moment of the period AADD, gives expressions for the variance and the standard deviation of the period AADD.

The structure of this paper is as follows: In Section 2, we specify the data for ten countries that we used. Section 3 gives a descriptive analysis of LEs and standard deviations in periods and birth cohorts of Norwegian men and women since the mid-1800s. In Section 4, we derive the equations for period and cohort measures of mortality. We use empirical data for Norwegian men and women to illustrate in detail the new expressions (Section 5), whereas some summary findings for the other nine countries are in the Supplementary Material. Section 6 discusses, among others, the link with Ryder's formulae for period and cohort measures for fertility. Conclusions are in Section 7.

2. Data for period and cohort life tables

The Human Mortality Database (HMD) contains cohort life tables for ten countries: Denmark, England and Wales, Finland, France, Iceland, Italy, the Netherlands, Norway, Sweden, and Switzerland; see HMD (2017). The periods and birth cohorts covered differ substantially across countries; see Table 1.

Table 1: Periods and cohorts covered by data from the HMD, selected countries

	Period life tables	Cohort life tables
Denmark	1835–2014	1835–1923
England and Wales	1841–2013	1841–1922
Finland	1878–2015	1878–1924
France	1816–2015	1816–1924
Iceland	1838–2013	1838–1922
Italy	1872–2014	1872–1923
Netherlands	1850–2014	1850–1923
Norway	1846–2014	1846–1923
Sweden	1751–2014	1751–1923
Switzerland	1876–2014	1876–1923

We extracted data from cohort and period life tables for men and women in the form of one-year age groups and single calendar years. All life tables stretch to age 110. Death probabilities at ages beyond 90 for cohorts born in 1904–1923 are based on extrapolated mortality rates using the Kannisto method (Wilmoth et al. 2017). Given the focus on the AADD, we selected the column labelled "dx" in the HMD data. This column gives numbers of deaths by age in the life table population.

The primary aim of the paper is to derive expressions for mortality shifts (changes in the LE) and mortality compression or expansion (changes in the standard deviation of the AADD). Therefore, we focus on mortality for adults and elderly persons, and we ignore mortality of children and young adults. Throughout this paper, we restrict ourselves to the AADDs for ages 30 and onwards. This choice of a cut-off age equal to 30 years is somewhat arbitrary, and it can be criticized (Robine 2001). In Section 6 we report empirical findings by Robine (2001) for France, by Engelman, Canudas-Romo, and Agree (2010) for 23 developed countries and by Permanyer and Scholl (2019) for high-income countries. There we conclude that using a cut-off age different from 30 years will have an impact on our findings, but that the patterns will be qualitatively similar as long as this age is not higher than approximately 40 years.

We rescaled age-specific data on the AADD for ages 30+ such that the sum over all ages from 30 onwards equalled one for each calendar year (period life table) and each birth cohort (cohort life table). Thus, the AADDs represent the probability of dying at a certain age $x \geq 30$, conditional upon survival until age 30.

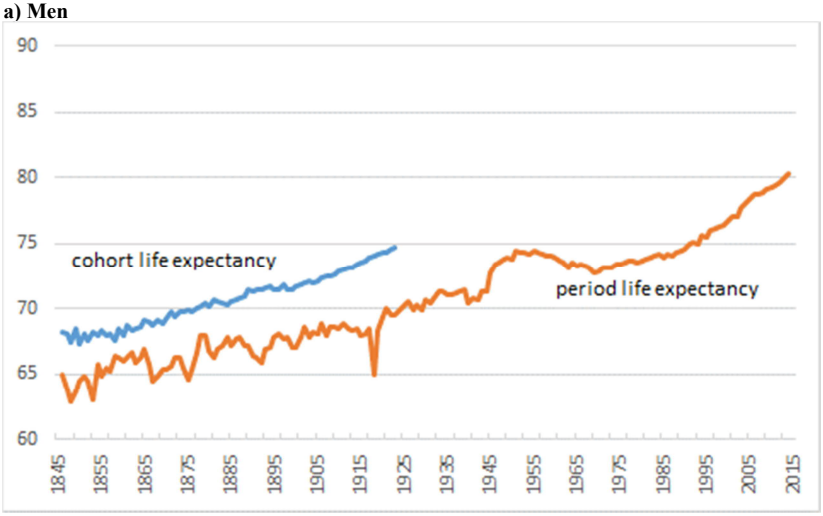
3. Period and cohort mortality – descriptive findings

Figure 1 plots period and cohort LEs of Norwegian men and women. Each LE is computed as the mean of the AADD, with ages from 30 to 110.

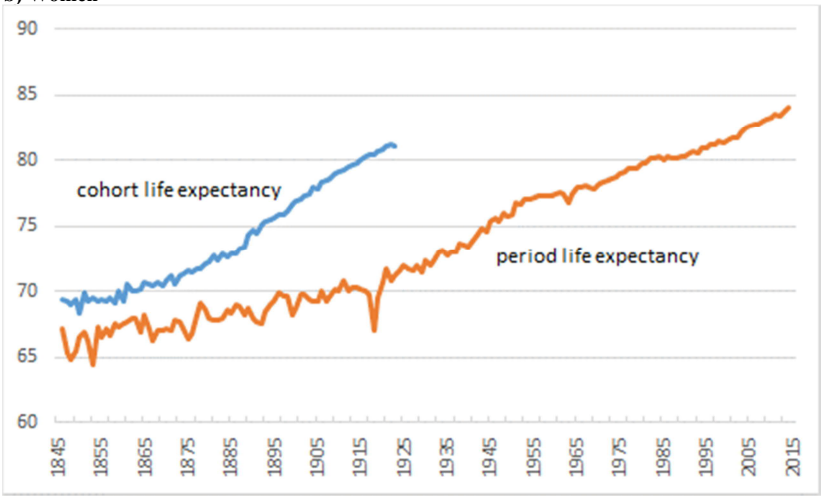
Trends in cohort LEs are smoother than those in period LEs. For women born after 1880 the trend is close to a straight line. For men, the pattern is a bit more irregular. Period LEs increase more or less in tandem with cohort LEs, with several exceptions. In 1918, the year of the Spanish Influenza, period LEs dropped by two to three years. Temporary low values are also visible for the years 1940–1945, especially for men. After World War II, period LEs of Norwegian men first rose more quickly than suggested by trends before 1940, next stagnated and even fell until the end of the 1960s, after which the pre-war upward trend was slowly picked up again.

Figure 1: Cohort LEs for cohorts born 1846–1923, and period LEs for the years 1846–2014, Norway. Each LE is conditional upon survival to age 30.

a) Men



b) Women



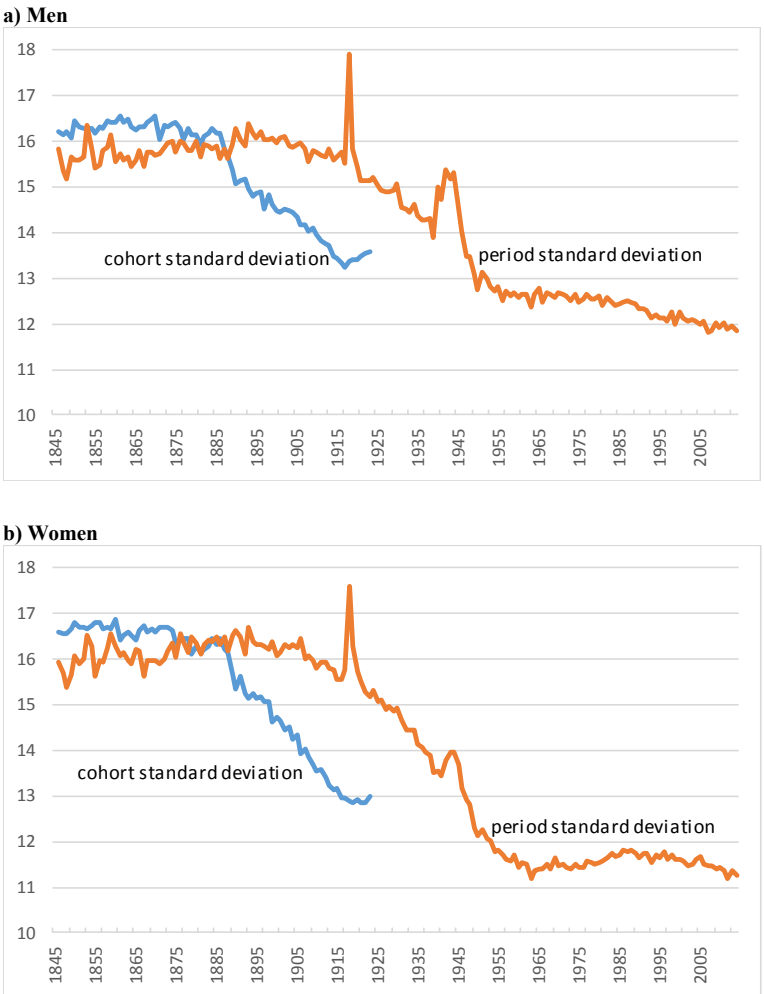
Male populations in a number of Western countries have experienced similar developments (Meslé and Vallin 2011; Luy 2015; Beltrán-Sánchez, Finch, and Crimmins 2015). Male LEs stagnated not only in Norway in the 1950s and 1960s but also in other countries such as Denmark, Finland, Netherlands, Belgium, New Zealand, and Australia. One possible explanation for the structural breaks in male mortality after World War II is the progression of the tobacco epidemic. Several studies find that smoking has had a distorting effect on trends in male mortality (e.g., Vollset, Tverdal, and Gjessing 2006; Beltrán-Sánchez, Finch, and Crimmins 2015; Janssen, Rousson, and Paccaud 2015; Peters, Mackenbach, and Nusselder 2016). For instance, Beltrán-Sánchez, Finch, and Crimmins (2015) show that smoking-attributable deaths account for about 30% of excess male mortality (compared to female mortality) at ages 50–70 for cohorts born in 1900–1935 in 13 developed countries. However, even after accounting for smoking, they find that excess male mortality at ages 50–70 remained, particularly from cardiovascular diseases. These findings suggest that different changes in mortality regimes for different age groups have led to changes in the age pattern of mortality. More generally, as Meslé and Vallin (2011) note, the trend shifts for males were caused by a change in major causes of death for middle-aged men, from infectious diseases before the war to cardiovascular deaths after the war.

The LE is a measure of *location* of the AADD. A measure for the *variability* in the AADD is the standard deviation of the distribution. Figure 2 plots the cohort standard deviation and the period standard deviation for the AADDs of Norwegian men and women. Standard deviations for cohorts born between 1846 and the end of the 1880s show very little variation. For later cohorts there is a systematic decline (and a slight increase for men born 1918–1923). The patterns for period standard deviations are very different, with a slight increase until the beginning of the 1900s and next a falling tendency until the beginning of the 1950s. In the second half of the 20th century, patterns for men and women are different, with a slight decline for men, and a more or less stable line for women. Period standard deviations peak in 1918, the year of the Spanish Influenza. The humps for men and women correspond to the years of World War II.

The patterns that we see in Figures 1 and 2 are related to changes in the age pattern of Norwegian mortality. Initially, childhood and reproductive-age mortality declined. Since we restricted the AADDs to ages 30 and over, we see no effects on LEs of men, and a weak effect on cohort LEs of women born in the 19th century. Also, period and cohort standard deviations for ages 30 and over are more or less constant. A second stage started around 1900, in which causes of death that operate in mid and later life were reduced. As a result, deaths became more concentrated at higher ages, and the standard deviations declined. In recent decades, much of the gain in LE is due to mortality reductions in old ages. Indeed, one may speak of "the advancing front of old-

age human survival" (Zuo et al. 2018). However, one should be prepared for growing inequalities in later life and a stagnation in the decline of the standard deviation; in future years, we might even see an increase.

Figure 2: Standard deviations (in years) of the AADD for cohorts born 1846–1923, and for calendar years 1846–2014, men and women, Norway. Ages 30–110



The link between period and cohort mortality that we will establish in Section 4 starts from an assumed relationship between the period probability $d[x,t]$ at age x in year t and the cohort probability $\delta[x,t]$ for the same age in the same year $t = g + x$, as defined in Section 1. Although the two probabilities refer to the same year and the same age, they are different, for two reasons. First, by the construction of the life table, the period probability $d[x,t]$ for any age $x > 0$ is a function of a series of age-specific death rates $m[0,t]$, $m[2,t]$, $m[3,t]$ … $m[x–2,t]$, $m[x–1,t]$ for the same year t. However, the cohort probability $\delta[x,t]$ depends of a different set of death rates, namely $m[0,t–x]$, $m[1,t–x+1]$, $m[2,t–x+2]$, … $m[x–2,t–2]$, $m[x–1,t–1]$ for the cohort born in year $(t–x)$. When mortality changes over time, death rates for year t and ages from 0 to x-1 differ from the set of rates for cohort $g = t–x$, ages from 0 to x–1. The second reason is the fact that we restrict the analyses to ages 30 and over. As described in Section 2, we normalize period probabilities for ages 30 and over from the HMD by dividing them by the sum of period probabilities for ages 30 and over for year t, whereas cohort probabilities are normalized by dividing them by a different sum, namely the sum of cohort probabilities ages 30+ for the cohort born in year $g = t–x$.[2]

Figure 3 gives scattergrams for $d[x,t]$ and $\delta[x,t]$ for selected ages x between 30 and 90 years for men and women in Norway. We used data from period life tables for the years 1876–2013, and life tables for cohorts born in 1846–1923. The Lexis diagram in Figure 4 illustrates the data situation. Each dot in the scattergrams of Figure 3 represents the pair$\{\delta[x,t], d[x,t]\}$ for one particular year t. For age 30, the years are 1876, 1877, …, 1953. For age 90, the years are 1936–2013. With a few exceptions, the plots suggest a linear relationship between the two variables. A straight line fits the data less well around age 80. We often find the modal age at death – which varies between 76 and 81 years for Norwegian men born between 1846 and 1923 – in this age interval. For young cohorts and ages between 75 and 80, say, the cohort probability $\delta[x,t]$ is located at the upward part of the slope of the AADD because age x is below the modal age. The period probability $d[x,t]$ for the same year and the same age may very well come from a life table with relatively low modal age at death. In that case, the latter probability is located at the downward part of the slope, beyond the modal age. When longevity improves, AADD curves shift to the right. The cohort mortality $\delta[x,t]$ moves down the slope, while the period probability $d[x,t]$ moves up. This continues until we reach a year in which the modal age of the period AADD exceeds x. Therefore, chances that the two probabilities move together are lower for ages around 80 than for higher or lower ages.

[2] An alternative normalization starts from a life table radix $l[30]$ equal to one and requires new life table calculations for periods and cohorts. In that case $d[30,t] = \delta[30,t]$, but the probabilities for higher ages still differ. However, for reasons of consistency we prefer to use the HMD life tables. This leads to different values of the two probabilities also for age 30.

Figure 3: **Scattergrams for the relationship between cohort probabilities δ[*x*,*t*] (horizontal axis) and period probabilities *d*[*x*,*t*] (vertical axis). Solid red lines are lines with slope 1, passing through the origin. Ages 30(10)90; cohorts 1846–1923; years 1876–2013, men and women, Norway**

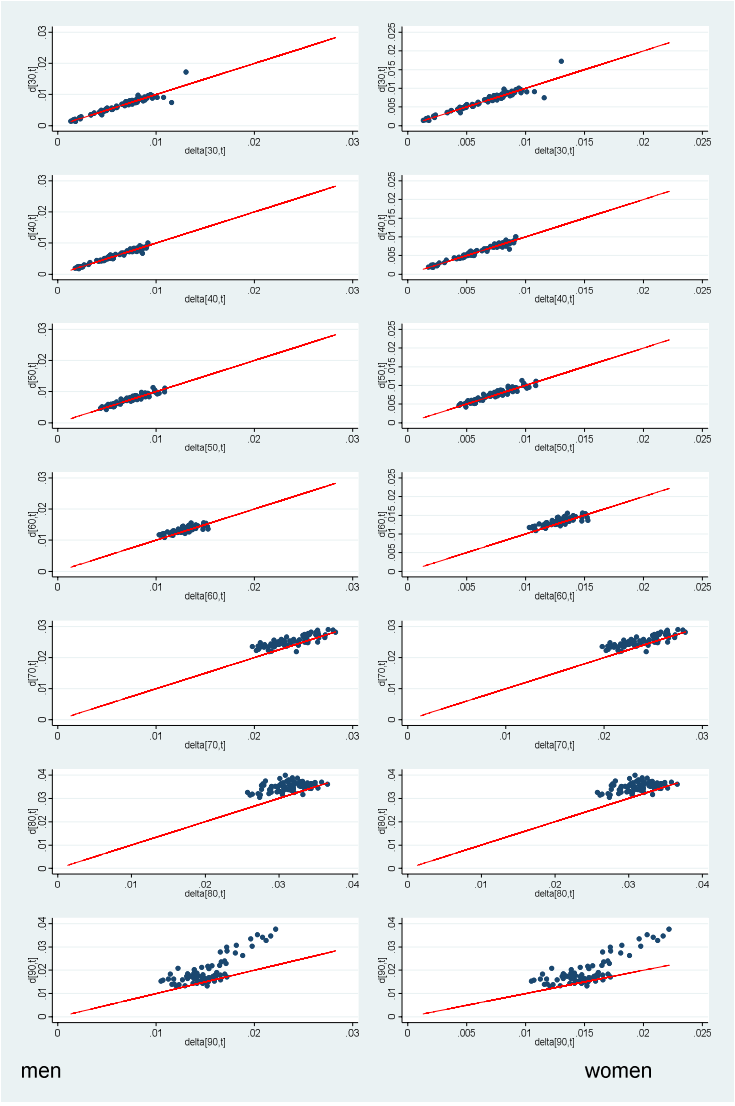
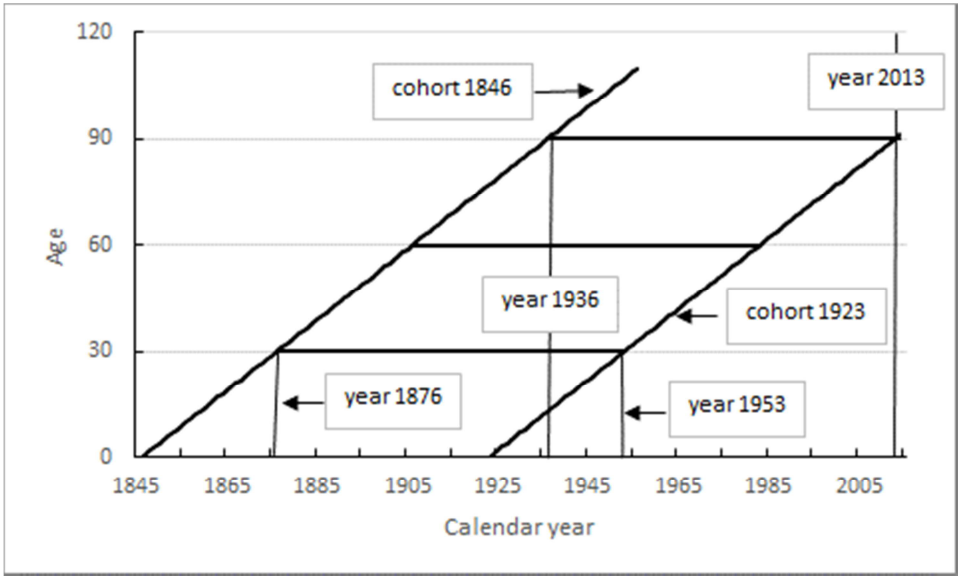


Figure 4: Lexis diagram illustrating data situation for Figure 3



An alternative explanation for the strong deviations from a straight line for age 80 is as follows: For ages 30, 40, and 50, chances of dying at these relatively young ages have fallen over time, both for period and for cohort AADDs. High values of both the cohort probabilities $\delta[x,t]$ and the period probabilities $d[x,t]$ for these ages were common in the late 19th century and the beginning of the 20th century, and less common in the second half of the 20th century. However, for age 90, high values of both indicators are more frequent at the end of the period than at the beginning; both the cohort probability $\delta[90,t]$ and the period probability $d[90,t]$ move upwards along the regression line for increasing t. For intermediate ages, illustrated by 80 in Figure 3, it is unclear whether the probabilities move in the same direction when time unfolds, or in opposite directions.

The red lines in Figure 3 are <u>not</u> regression lines. Rather, they represent strict equality between cohort and period probabilities ($\delta[x,t] = d[x,t]$) and are plotted as a reference. Note how the dots gradually drift away from the red lines when age increases. Yet a linear relationship between cohort and period probabilities is reasonable (with the exception of ages around 80, for reasons explained above).

4. The new expressions

Based on our findings for Norway (Figure 3) and other countries (Figure A-1 of the Supplementary Material), we assume a linear relationship $d[x,t] = a_x + b_x.\delta[x,t]$ between cohort and period probabilities. Denote the *k*-th period moment of the period-AADD by $V_k[t] = \Sigma x^k.d[x,t]$, for $k = 1, 2, 3, \ldots$. Similarly, the *k*-th cohort moment of the cohort distribution is $W_k[g] = \Sigma x^k.\delta[x,g+x]$. We derive a general expression for the period moments $V_k[t]$ as a function of the cohort moments $W_k[g]$ and their time derivatives. First we use Taylor series expansion to write the cohort probability $\delta[x,t]$ for a fixed age *x* as a series of terms that include its first and higher-order time derivatives. Next, we use the assumed linear relationship between cohort and period probabilities to link the Taylor series to the period probability $d[x,t]$. Multiplying by x^k and taking the sum over all ages, we find, after some algebra, the link between cohort moments and period moments. The derivation goes as follows:

$\delta[x,g+x]$ is the probability of dying at age *x* for the members of the cohort born in year *g*. Take the cohort born in year $g = t–x$; then $\delta[x,g+x] = \delta[x,t]$. A Taylor series expansion of $\delta[x,t]$ about $t+x$ gives

$$\delta[x,t+x-x] = \delta[x,t+x] - x.\delta'[x,t+x] + \frac{x^2}{2!}.\delta''[x,t+x] - \frac{x^3}{3!}.\delta'''[x,t+x] + \cdots$$

or

$$\delta[x,t] = \sum_{i=0}^{\infty} \frac{(-x)^i}{i!} \delta^{(i)}[x,t+x].$$

Differentiation applies to time. Use the assumed relationship $d[x,t] = a_x + b_x.\delta[x,g+x] = a_x + b_x.\delta[x,t]$ to find

$$d[x,t] = a_x + b_x \sum_{i=0}^{\infty} \frac{(-x)^i}{i!} \delta^{(i)}[x,t+x].$$

Multiplying by x^k and taking the sum over *x* gives

$$\sum_{x=0}^{\omega} x^k.d[x,t] = \sum_{x=0}^{\omega} x^k \left\{ a_x + b_x \sum_{i=0}^{\infty} \frac{(-x)^i}{i!} \delta^{(i)}[x,t+x] \right\}$$

or

$$V_k[t] = A_k + \sum_{x=0}^{\omega} x^k \, b_x \sum_{i=0}^{\infty} \frac{(-x)^i}{i!} \delta^{(i)}[x, t+x]$$

where $A_k = \Sigma x^k.a_x$. By the Mean Value Theorem we can find a constant $B_k \neq 0$ such that

$$V_k[t] = A_k + B_k \sum_{x=0}^{\omega} x^k \sum_{i=0}^{\infty} \frac{(-x)^i}{i!} \delta^{(i)}[x, t+x].$$

This leads to

$$V_k[t] = A_k + B_k \sum_{i=0}^{\infty} \frac{(-1)^i}{i!} W_{k+i}^{(i)}[t]. \quad (1)$$

Expression (1) gives the general relationship between the moments $V_k[t]$ of the period distribution for the year t and the moments $W_k[t]$ of the cohort distribution of the cohort born in year t. We will use expression (1) to analyse two cases of interest: $k = 1$ for the mean age/LE, and $k = 2$ for the variance of the age distribution.

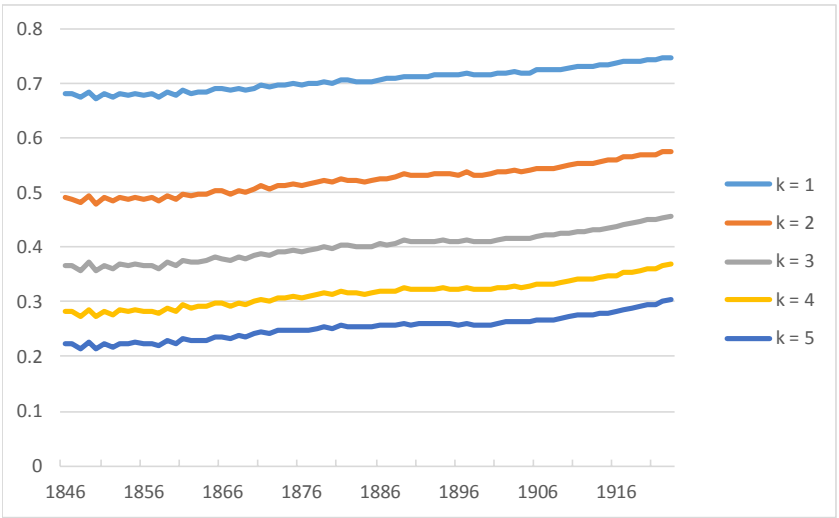
4.1 Mean age ($k = 1$)

The right side of expression (1) has infinitely many terms. This is not practical in empirical applications. For the remainder of this paper we will assume that we can approximate all cohort moments $W_k[t]$ by linear functions of time. Figure 5 shows that this is a realistic assumption for moments of orders 1–5 of Norwegian women born 1880–1923, and for men born 1846–1923. Figure A-2 in the Supplementary Material suggests (with the exception of Danish women) linear trends in these moments for men and women in the other nine countries who were born in two to three decades up to the 1920s.[3]

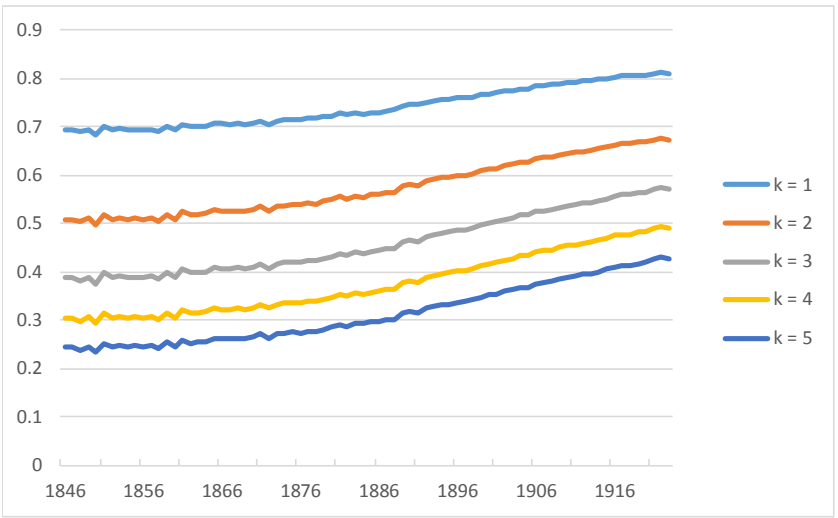
[3] Note that the assumed linearity for the moments concerns a linear trend over time. However, the linear relationships in Figure 3 concern the link between cohort and period probabilities. These probabilities do not need to be linear functions of time. For each age, the time trend in the period probability is merely a linear transformation of the time trend in the cohort probability.

Figure 5: Moments of orders one ($k = 1$) to five ($k = 5$) of the AADD for cohorts born 1846–1923, Norway

a) Men



b) Women



Note: When computing the moment of order k, we rescaled age x as $x/100$.

Using this assumption, and writing $e_0^p[t]$ and $e_0^c[g]$ for the period and the cohort LEs, respectively, expression (1) leads to

$$e_0^p[t] = A_1 + B_1\{e_0^c[t] - W_2'\}. \quad (2)$$

The period LE in year *t* is a linear function of the cohort LE of persons born in year *t*. Over time, the period LE follows a straight line, with a constant term $(A_1 - B_1 W_2')$ and a coefficient B_1. When period and cohort AADDs do not change over time, $A_1 = 0$ and $B_1 = 1$. The period LE increases by $e_0^p[t]' = B_1 . e_0^{c\prime}$, where $e_0^{c\prime}$ is the annual increase in the cohort LE, which is constant by assumption. Thus, the period LE may increase faster (B_1>1) or slower (0<B_1<1) than the cohort LE. The term $(-W_2')$ reflects a change over time in the age pattern of the cohort AADD. When the slope of the second moment is positive ($W_2' > 0$), this in itself has a downward effect on the period LE (as long as B_1 is positive).

In principle, one could use expression (2) to estimate the parameters A_1 and B_1 by linear regression. However, the mortality experience of the cohort born in year *t* has little in common with the period mortality for the synthetic cohort of year *t*. Therefore, we opted for a different approach.

Given our assumption of linear cohort LEs, we can write $e_0^c[t - \lambda] = e_0^c[t] - \lambda e_0^{c\prime}$ for a cohort that was born λ years before *t*. Eliminating $e_0^c[t]$ from expression (2) gives

$$e_0^p[t] = A_1 + B_1\{e_0^c[t - \lambda] + \lambda e_0^{c\prime} - W_2'\}. \quad (3)$$

For instance, assuming a lag λ equal to 75 years (the cohort LE of Norwegian women born around the middle of the period 1846–1923; see Figure 1), gives $e_0^p[g + 75] = A_1 + B_1\{e_0^c[g] + 75e_0^{c\prime} - W_2'\}$. The period LE in a certain year is a linear function of the cohort LE for the cohort born 75 years earlier. Expression (3) forms the basis for the regression analysis of the link between period and cohort LEs in Section 5.

λ is the time interval between the year *g* when the cohort was born, and the year $t = g + \lambda$ for which we compute the period life table ('forward-looking lag'). Consider the year *t* in which cohort *g* reaches an age equal to its own LE.[4] How large is the period LE in that year? Insert $\lambda = e_0^c[g]$ in expression (3) to find

$$e_0^p[t] = A_1 - B_1 . W_2' + B_1 . (1 + e_0^{c\prime}) . e_0^c[g], \quad (4)$$

[4] In Section 6, we discuss the link with the adjusted period LE of Bongaarts and Feeney (2002, 2003) and the cross sectional average length of life (CAL) of Brouard (1986) and Guillot (2003).

where year t equals $g + e_0^c[g]$. Expression (4) gives the period LE for the year t in which cohort g reaches its own LE.

Expression (3) applies to the general case with an unspecified lag of λ years. Consider the period LE in a certain year t. How large is the lag that leads to an equally large cohort LE for a cohort born in year t–λ ('backward-looking lag')? In other words, how many years does it take a period LE to reach the current level of cohort LE? Substitute $e_0^p[g + \lambda] = e_0^c[g]$ in (3) and solve for λ to find

$$\lambda = \lambda[g] = \frac{(1-B_1).e_0^c[g]+B_1W_2'-A_1}{B_1 e_0^{c\prime}}. \quad (5)$$

In general, the period LE in year t is different from the LE of the cohort born in year t. The gap $\gamma[t] = e_0^c[t] - e_0^p[t]$ between these two (cf. Goldstein and Wachter 2006) equals $(1 - B_1)e_0^c[t] + (B_1.W_2' - A_1)$, which, after some algebra, is the same as $\lambda[g].B_1.e_0^{c\prime} = \lambda[g].e_0^{p\prime}$. The gap is positive as long as $e_0^c[t] > (A_1 - B_1.W_2')/(1 - B_1)$.

4.2 Variance in the age at death (k = 2)

Changes in the variance and the standard deviation of the AADD signal a possible compression or expansion of mortality. A number of indicators for compression and expansion have been proposed, but many of them are strongly correlated (Wilmoth and Horiuchi 1999). Hence, they are likely to move in the same direction.

The variance of any distribution equals its second moment minus its first moment squared. We write S^2 for the period variance, and σ^2 for the cohort variance. As before, we assume linear cohort moments. Expression (1) gives

$$V_2[t] = A_2 + B_2\{W_2[t] - W_3'\}. \quad (6)$$

Under the assumptions stated, the second period moment is a linear function of time. Writing (6) in terms of variances gives

$$S^2[t] = A_2 + B_2\{\sigma^2[t] + (e_0^c[t])^2 - W_3'\} - \left(e_0^p[t]\right)^2. \quad (7)$$

The period variance is a sum of three time-dependent functions. The cohort variance σ^2 and the cohort LE squared $(e_0^c[t])^2$ drive the period variance up (provided that $B_2 > 0$; see below). The period LE squared $\left(e_0^p[t]\right)^2$ presses it down. Whether the period variance decreases (signalling compression of mortality around the mean) or

increases (expansion) can be determined by taking first derivatives of both sides of expression (7). This gives

$$\frac{\partial}{\partial t}S^2[t] = B_2 W_2' - 2.e_0^{p\prime}.e_0^p[t]. \quad (8)$$

Compression of mortality, in the sense of decreasing period variance, occurs when $e_0^p[t] > B_2 W_2'/2e_0^{p\prime}$. These expressions help to shed light on the question why, in a period perspective, increases in longevity often go together with less variability in the age at death (Canudas-Romo 2008; Ouellette and Bourbeau 2011; Muszyńska and Janssen 2016; Németh 2017).

Similar to the case of the LE, we can write for the second cohort moment lagged by λ years before *t*

$$V_2[g+\lambda] = A_2 + B_2\{W_2[g] + \lambda W_2' - W_3'\}. \quad (9)$$

Expression (9) forms the basis for the empirical analysis of the link between period and cohort variances and standard deviations in Section 5.2.

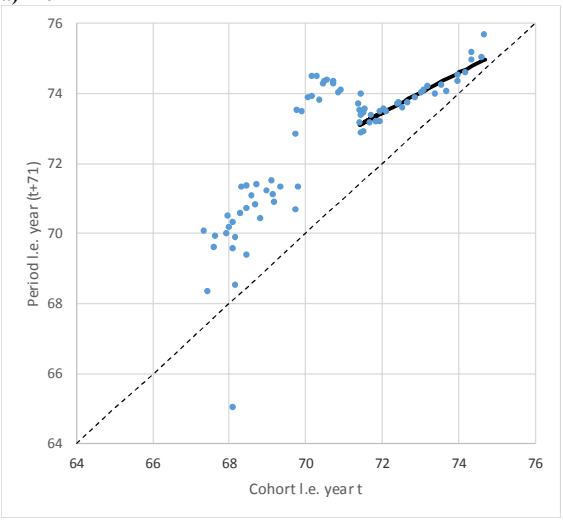
5. Application to mortality in Norway

5.1 Life expectancies

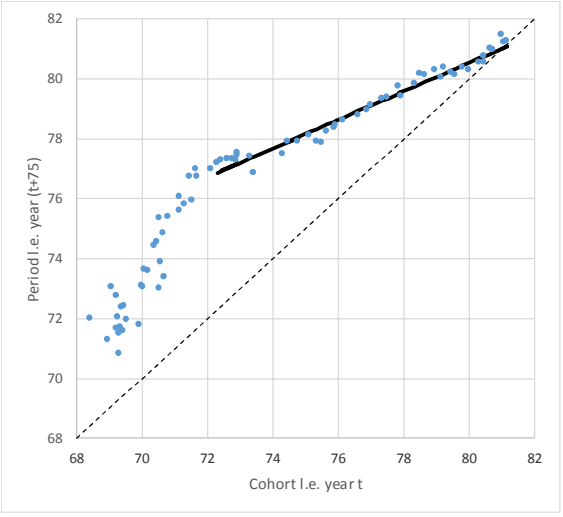
Figure 6 is a scattergram for period and cohort LEs of men and women in Norway. Cohort LEs are lagged by 71 years for men and by 75 years for women. The lags represent cohort LE values of men and women born around the middle of the period 1846–1923.

Figure 6: Scattergram for cohort LEs for cohorts born 1846–1923 and period LEs 71 years (men) and 75 years (women) later, Norway

a) Men



b) Women



For women born after 1880 (LEs 72 years or more), the relationship is close to a straight line. The cohort LE of these women increases twice as fast as the period LE. For men, there seem to be three regimes. The first one is for cohort LEs up to 70 years (cohorts 1846–1877). The period LEs increase more or less in tandem with cohort LEs, although the pattern is quite irregular. The outlier (period LE of 65.0 years) is 1918, the year of the Spanish Influenza. Another regime is for cohort LEs from about 71.5 years and beyond (cohorts born 1890 and later), with period LEs increasing rather regularly. For these men, cohort LEs increase approximately twice as fast as period LEs (similar to the case of women). In between are years that correspond with cohort LEs between 70 and 71.5 years (cohorts born 1877–1890). As noted before, after World War II, period LEs of Norwegian men first rose more quickly than suggested by trends before 1940, next stagnated and even fell until the end of the 1960s, after which the pre-war upward trend was slowly picked up again. Why does this irregular pattern in the period LE of men occur? Note that third and higher cohort moments do not follow a strict straight line for cohorts born 1877–1910; see Figure 5a. The shape of the cohort AADD changed in various ways. The mean age at death shifted regularly to higher ages, and the standard deviation became smaller, signalling stronger compression. In addition, the tail to the left of the AADD (ages 30–60) became more pronounced for cohorts born 1846–1890, reflected in a skewness (normalized third central moment) that became more and more negative. After 1890, the trend reversed, signalling that the tail at the left of the distribution became less important.[5]

Based on expression (3), we assumed the following model for period and cohort LEs:

$$e_0^p[t] = \alpha_1 + \beta_1 . e_0^c[t - \lambda] \; + \; \varepsilon_1[t], \tag{10}$$

where $\alpha_1 = A_1 + B_1(\lambda e_0^{c\prime} - W_2')$ and $\beta_1 = B_1$ are parameters to be estimated. $\varepsilon_1[t]$ is an error term. We performed linear regressions with cohort data for men born 1890–1923 (period data 1961–1994, lag λ equal to 71 years) and for women born 1880–1923 (period data 1955–1998, $\lambda = 75$ years).[6] Table 2 gives the results. The parameter estimates are strongly significant, and the R^2-values are close to 1. The solid black lines in Figure 6 represent the regressions. The β_1-estimates confirm earlier findings: When the cohort LE increases by one year, the period LE 71 (75) years later increases by 0.573 (0.476) years. In other words, the increase in period LEs of Norwegian men and women is only about half the increase in cohort LEs.

[5] The skewness (normalized third central moment) of the distribution fell regularly from –0.56 for men born in 1846 to –0.77 for cohort 1880. It fluctuated between –0.8 and –0.9 for cohorts 1881–1913, with a lowest value of –0.90 for cohort 1890. For cohorts born in 1914–1923, the skewness changed from –0.77 to –0.56.

[6] Alternative lags are discussed in Section 6.

Table 2: Regression estimates for LEs in expression (10)

	Men (λ = 71)	Women (λ = 75)
α_1	32.200	42.441
Standard error	2.926	0.997
β_1	0.573	0.476
Standard error	0.040	0.013
R^2	0.929	0.985
N	34	44

Under the conditions stated earlier, the estimates imply that the cohort LE of those born in a certain year is larger than the period LE 71 (men) or 75 (women) years later, for cohort LEs that exceed 32.200/(1–0.573) = 75.4 years of age for men, and 42.441/(1–0.476) = 81.0 years of age for women. Norwegian women born in 1921 are expected to reach LE levels that high (see Figures 1 and 5). Norwegian men seem to be close to that tipping point. Assuming an increase in cohort LE of one year per decade (the average slope of the LE for cohorts born 1890–1923), men born around 1930 are likely to reach LEs of 75.4 years or more.

Expression (4) gives the relationship between cohort and period LE for the year t in which cohort g reaches its own LE. Using the estimates from Table 2, we find $e_0^p[t] = 24.73 + 0.63e_0^c[g]$ for men and $e_0^p[t] = 27.25 + 0.57e_0^c[g]$ for women. With this particular lag, the cohort LE exceeds the period LE for male cohort LEs above 24.733/(1–0.630) = 66.84 years, and for female cohort LEs above 27.250/(1–0.573) = 63.82 years. These values are lower than the lowest cohort LEs in our data set. Thus, for men born in 1890 or later, and for women born in 1880 or later, the cohort LE is always larger than the period LE in the year when the cohort reaches its own LE.

For the backward-looking lag in expression (5), we first note that Norwegian men born between 1890 and 1923 and Norwegian women born between 1880 and 1923 increased their LEs by approximately one year and two years of age per decade on average, respectively. Using $e_0^{c\prime} = 0.1$ for men and $e_0^{c\prime} = 0.2$ for women, together with the parameter estimates from Table 2, we find a predicted backward-looking lag $\lambda[g]$ equal to $7.49e_0^c[g] - 493.68$ years for men, and $5.43e_0^c[g] - 365.06$ years for women. The lags widen rapidly, by approximately five and a half to seven and a half years for every one-year increase of the cohort LE. For a cohort LE of 71 years (men born 1890) or 75 years (women born 1892), the predicted lags are 38.3 and 42.3 years, respectively. The lags are 65.8 years (men) and 75.0 years (women) for the most recent cohort for which we have enough data, i.e., those born in 1923. Canudas-Romo and Schoen (2005) demonstrate a qualitatively similar effect for the so-called Siler model of age-

specific mortality. Our results concur with those of Goldstein and Wachter (2006), who find a lag of about 40–50 years using data from the United States and Sweden. They also note that the lag lengthens as mortality improves.

As noted in Section 4, the gap is positive for $e_0^c[t] > \alpha_1/(1-\beta_1)$, which amounts to 65.9 years for men and 67.2 years for women. These values are lower than the earliest cohort LEs; see Figure 1. The gap will become wider as longevity improves. Our model suggests that it will grow by $(1–\beta_1)$ years, or by roughly half a year for every one-year increase in cohort LE.

5.2 Standard deviations

Figure 7 shows, for the case of Norway, the relationship between period second moments in a certain year and cohort second moments 71 years (men) and 75 years (women) earlier. As before, we note that the relationship is approximately linear for men born 1890–1923 ($W_2[g] > 5300$) and for women born 1880–1923 ($W_2[g] > 5500$). A linear regression for these cohorts and lags λ equal to 71 and 75 years was based on model (11) below.

$$V_2[t] = \alpha_2 + \beta_2 . W_2[t-\lambda] + \varepsilon_2[t], \tag{11}$$

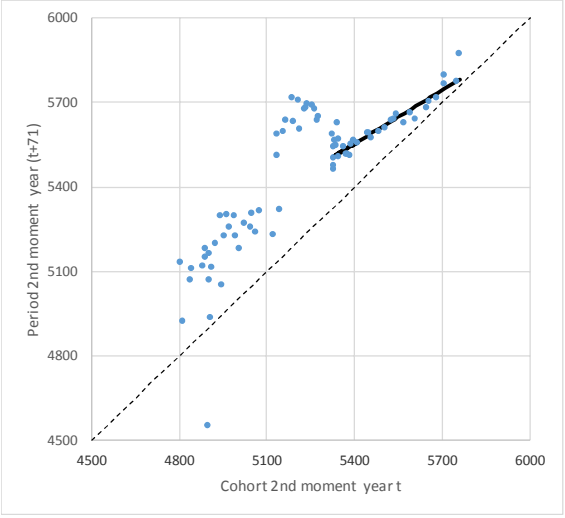
where $\alpha_2 = A_2 + B_2(\lambda W_2' - W_3')$ and $\beta_2 = B_2$ are parameters to be estimated. Table 3 gives the results.

Again, the parameter estimates are strongly significant, and the R^2-values are close to 1. See also the solid black lines in Figure 7. Given these estimates, period variances $S^2[t]$ are

$S^2[t] = 2117.3 + 0.636(\sigma^2[t-71] + (e_0^c[t-71])^2) - \left(e_0^p[t]\right)^2$ for men, and
$S^2[t] = 3081.7 + 0.539(\sigma^2[t-75] + (e_0^c[t-75])^2) - \left(e_0^p[t]\right)^2$ for women.

Figure 7: Scattergram for cohort second moments for cohorts born 1846–1923, and period second moments 71 years (men) and 75 years (women) later, Norway

a) Men



b) Women

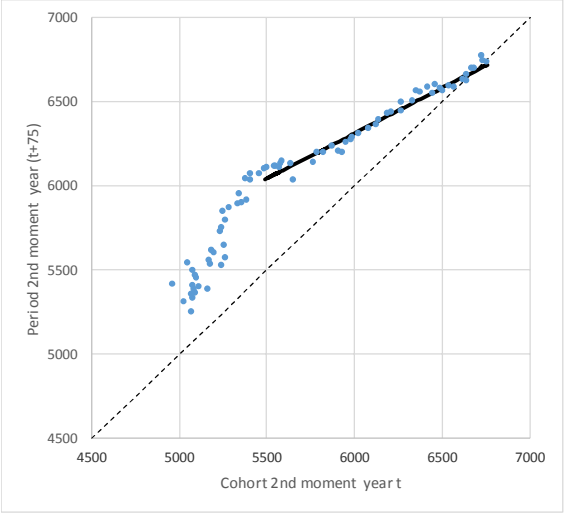


Table 3: Regression estimates for second moments in expression (11)

	Men (λ = 71)	Women (λ = 75)
α_2	2117.3	3081.7
Standard error	226.2	89.6
β_2	0.636	0.539
Standard error	0.041	0.015
R^2	0.939	0.985
N	34	44

When the period LE increases by two years, the period variance falls by four years squared (years^2). However, for every two-year increase in the cohort LE, the period variance increases by 0.636*4 = 2.5 years^2 for men and by 0.539*4 = 2.2 years^2 for women. In addition, the period variance changes are not as fast as the cohort variance changes; the speed reduction is 36 % for men and 46 % for women[7]. The period variance falls over time because the condition that $e_0^p[t] > B_2W_2'/2e_0^{p'} = \beta_2W_2'/2e_0^{p'}$ is fulfilled for both men ($e_0^p[t] > 42.4$ years; $e_0^{p'} = 0.1$ and $W_2' = 13.328$) and women ($e_0^p[t] > 38.83$ years; $e_0^{p'} = 0.2$ and $W_2' = 28.816$). Thus, there is compression of mortality around the mean of the period AADD for both sexes, although this process seems to have stagnated for women since the 1960s; see Figure 2. One technical explanation is that the slope of the period LE of Norwegian women has varied a great deal since 1950. Period LEs increased much more slowly in the 1960s (0.8 years per decade) and 1980s (0.5 years per decade) than in other decades after 1950 (between 1.3 and 1.6 years per decade); see Figure 1. Other things being the same, a strong increase in $e_0^p[t]$ leads to a steep fall in $S^2[t]$ and vice versa. A similar stagnation in mortality compression is visible for men and women in Denmark, Finland, and Sweden, and for men in England and Wales, France, and Switzerland; see Supplementary Material Figure A-3. In addition to this technical point, we can refer to the different stages of the epidemiological transition, discussed in Section 3. Countries that experience a gain in LE particularly due to mortality reductions in old ages might show growing inequalities in later life and a stagnating compression, or even expansion. Other countries that have not yet entered this stage might show a steady compression of mortality.

Tuljapurkar and Edwards (2011) plot the historical trend in the period standard deviation above age 10 for both sexes combined in the United States in the period

[7] When we express dynamics in lifespan inequality in terms of changes in the *standard deviation*, the speed reduction is less than 1-B_2. This is because $\partial S/\partial\sigma = B_2(\sigma/S)$ by the chain rule of differentiation, together with the empirical fact that $\sigma > S$ with lags equal to 71 years for men and 75 years for women; see Figure 2.

1959–2005, and the predicted trend using Lee–Carter-based mortality projections to 2050. While the historical pattern fluctuates between 15.0 and 15.8 years, the standard deviation is clearly sloping downwards to 2050. Canudas-Romo (2008) analyses a related dispersion indicator of the distribution of deaths, namely the standard deviation around the *modal* age. His standard deviations for six developed countries for both sexes combined show a much slower fall during the second half of the century than in the first half. This agrees with the pattern for Norway in Figure 2.[8]

The fact that there is compression in Norwegian cohort mortality, too, is also visible in Figure 5. The first derivative with respect to time of cohort variance $\sigma^2[t]$ equals $W_2' - 2e_0^{c\prime}e_0^c[t]$. The curves for the first and the second cohort moments in Figure 5 are approximately parallel to each other. Since we have scaled age x as x/100, Figure 5 suggests that $W_2' \approx 100e_0^{c\prime}$. Thus, cohort variance falls as soon as cohort LE is larger than approximately 50 years, which is the case for all Norwegian cohorts in our data set (as well as the cohorts in the other nine countries). The compression in Norwegian cohort mortality is one of the factors that contributes to the compression in period mortality; cf. expression (7).

6. Discussion

The issue that this paper addresses is how cohort measures of mortality affect period measures. For fertility, this has been studied extensively. Ryder (1956, 1964, 1980) was the first to analyse the link between cohort and period fertility in times when the level and the age pattern of fertility changes, and who coined the term 'translation analysis.' Although the translation approach in this paper bears resemblance to fertility translation, there are two important differences. First, for fertility, one distinguishes between the level (Total Fertility Rate or TFR for periods, Completed Cohort Fertility or CCF for birth cohorts) and the age pattern (Mean Age at Childbearing, variance of the age schedule defined by age-specific fertility rates). For mortality, the level is one (death per person), since everyone dies. Hence we have restricted the analysis to moments of orders one and higher. For fertility, one uses the moment of order zero to analyse the link between TFR and CCF (e.g., Ryder 1964: 76). Second, the fertility rate $f(x,t)$ for age x in year t is part of both the TFR (for year t) and of the CCF (for the

[8] The standard deviations of Canudas-Romo are much larger (around 40 years in 1900, falling to 15–20 years in 2000) than those for Norway in Figure 2 (declining from 16 to 11–12 years over the same period). There are two explanations. First, he did not condition on survival to age 30, but included all ages in the standard deviation. Second, he computed the standard deviation from the modal age at death, not the mean age at death. This in itself increases the standard deviation: for any probability distribution, the variance around the mean μ is smaller than the variance around the mode M by an amount of $(\mu\text{-M})^2$.

cohort born $t–x$). However, the period probability $d[x,t]$ for age x in year t is different from the cohort probability $\delta[x,g+x]$ for the same age and year ($g+ x = t$). We assumed a linear relationship between period probabilities $d[x,t]$ and cohort probabilities $\delta[x,g+x]$. In the hypothetical case that the two are equal for all x and t, A_k in expression (1) is zero and B_k is one. In that case, equation (1) reduces to Ryder's general formula (adapting his notation to ours)

$$V_k[t] = \sum_{i=0}^{\infty} \frac{(-1)^i}{i!} W_{k+i}^{(i)}[t]\,;$$

see Ryder (1964: 76).

We have used Norwegian data to illustrate the link between period and cohort mortality. Expression (1) covers the relationship fully, but one has to use an approximation in empirical applications. The graphs in the Supplementary Material suggest that the periods and cohorts for which there is a clear linear relationship between period and cohort moments can differ from one country to another. The reason might be that different countries have experienced the changes in the age pattern of mortality during different periods. Note the period standard deviations of Iceland and Italy in Figure A-3: They show a more or less systematic decline, as opposed to the patterns for the other countries, where we often see a stagnation in recent years (for men in England and even, for Wales, an increase). As discussed in Section 3 for Norway, this stagnation might be related to a very recent stage in the dynamics of the AADD, in which mortality decline is faster at old than at young ages. Iceland and Italy seem not to have entered this stage yet. Other countries that have developed more recently, such as Spain or Japan, may be qualitatively similar to Iceland and Italy when it comes to the time trend in the standard deviation. On the other hand, in countries in which mortality decline started early, we may expect, in the future, a positive correlation between the LE and the standard deviation.

We have restricted ourselves to the AADDs for ages 30 and onwards. Our choice of a cut-off age equal to 30 years is somewhat arbitrary, and it can be criticized (Robine 2001). For the mean age at death, selecting a cut-off age of 20, or 30, or 40 years clearly will have an impact on its value, but not very much on its qualitative behaviour over time (increase, decrease, fluctuations). Permanyer and Scholl (2019) analysed global trends in the mean age at death between 1950–1955 and 2010–2015. They considered cut-offs at age 15 and at age 65 and computed mean ages at death μ_{15} and μ_{65} with these cut-off ages. For high income countries, both values increased regularly from 1950–1955 to 2010–2015. In 1950–1955, μ_{65} was higher than μ_{15} by 7.8 years. The gap diminished regularly over time; in 2010–2015 it was 4.9 years. Because the pattern is very regular, we expect smaller gaps, also diminishing over time, when comparing

mean ages based on cut-offs at ages 20 and 30, or 30 and 40. However, the choice for a particular cut-off age is likely to have an effect on the degree of mortality compression and its development over time. Robine (2001) computed the standard deviation of the AADD for men and women in France between the periods 1890–1894 and 1990–1994, as a function of the cut-off age. He found that the standard deviation fell over time when using cut-off ages up to 45 years, approximately. On the other hand, the standard deviation increased when using cut-off ages between 45 and 90 years. He explained his findings by pointing out that although mortality fell at all ages during these 100 years, the decrease in child mortality came first (as a consequence of the decline in infectious diseases), whereas mortality started to decline at adult ages, including among the very old, starting in the 1950s in Northern and Western Europe, North America, and Japan. The findings of Engelman, Canudas-Romo, and Agree (2010) and Permanyer and Scholl (2019) support those of Robine (2001) for France. Engelman, Canudas-Romo, and Agree (2010) found that standard deviations after age 10 fell by approximately 20%–40% between 1900 and 2006 in 12 European countries (including New Zealand). In 11 other developed countries during the period 1950–2005/2006, they fell by 10%–30% (except for men in the United States, where the standard deviation after age 10 was the same in 1950 as in 2005). However, when they analysed the standard deviation with a cut-off age of 50 years instead of 10, they found an increase over time in 32 of 46 cases. Using a cut-off age of 75 years, the standard deviations increased in all cases. Permanyer and Scholl (2019) reported the variance in the AADD based on cut-off ages of 15 and 65 years. They showed that the variance in the AADD for ages 15+ for high income countries declined between 1950–1954 and 2010–2015 (although not regularly), while the variance for ages 65+ increased systematically. Based on these findings, we conclude that using a cut-off age different from 30 years will have an impact on our findings, but that the patterns will be qualitatively similar as long as this age is not higher than approximately 40 years.

We assumed a linear relationship $d[x,t] = a_x + b_x.\delta[x,t]$ in the derivation of expression (3). Note that this is not a necessary condition to hold for (3) to be true. The scattergrams in Figure 6 suggest that expression (3) is plausible (for selected cohorts), even if we would not have seen plots for the relationship between $d[x,t]$ and $\delta[x,t]$. In other words, linearity in the age-specific period and cohort probabilities does not need to exist for the expressions to work.

On the other hand, assume that Figure 3 suggests a nonlinear relationship between cohort probabilities $\delta[x,t]$ and period probabilities $d[x,t]$. What are the consequences for the simple expressions (2) and (6) that we found with linear relationships? There is no general answer. The simplest case is a quadratic relationship for each x and t, between the period probability d and the cohort probability δ: $d[x,t] = a_x + b_x .\delta[x,t] + c_x .\delta^2[x,t]$. Then we need an extra term $C_k \sum x^k \delta^2[x,t]$ in expression (1), where the sum is over ages

x. C_k is a parameter to be estimated. Independently from this quadratic assumption, Figures 5 and A-2 show that the cohort moments up to $k = 5$ are approximately linear functions of time. Thus, for the first moment we still have expression (2), but with an extra term $C_1 \sum x\delta^2[x,t]$. Note that the sum is over ages x, for a fixed year t. For that year, this term is constant (given an estimate of C_1). Because the sum $\sum x\delta^2[x,t]$ includes δ^2, it is much smaller than an LE value. Attempts to simplify the term $C_1 \sum x\delta^2[x,t]$ further were unsuccessful. Developing $\delta^2[x,t]$ in a product of two Taylor series of $\delta[x,t+x]$ and its time derivatives leads to a complicated expression that does not give much insight. Nor does a Taylor series of $\delta^2[x,t+x]$ and its time derivatives. The conclusion must be that when $d[x,t]$ is a polynomial in $\delta[x,t]$ rather than a linear function, expressions (2) and (6) do not hold any longer: They have to be complemented with one (third-degree polynomial) or more (higher order polynomial) additional terms. The additional terms will be different for each moment, but whether they have a large or a small impact on the period LE or the period variance is an empirical issue.

We assumed that cohort moments are linear functions of time. Figures 5 and A-2 (Supplementary Material) suggest that this is a reasonable assumption for moments of orders 1–5 for the countries for which we have data. However, when one or more cohort moments are not linear, one has to include first and higher order time derivatives for the nonlinear moment(s). Expression (1) tells us how this can be done in principle, but the resulting expressions will soon be difficult to handle in empirical applications.

We selected lags of 71 years (men) and 75 years (women) when estimating model (3). Alternatively, we could have assumed a lag equal to the cohort LE in expression (4). This requires interpolation of period LEs between subsequent years (or rounding the year $g + e_0^c[g]$ to the nearest integer). Alternatively, one could select a lag that results in the best fit. We have experimented with lags between 71 and 79 years. For women, the estimates of B_1 were very similar. For men, the B_1-estimate changed from 0.57 (lag 71 years) to 0.72 (73 years) and 0.86 (75 years). Further inspection revealed that, contrary to our assumption, the slope of the second moment for men is a bit erratic but tends to increase slightly between cohorts 1890 and 1923. (For women, the slope is more or less constant.) Thus, one may include a quadratic second cohort moment for men instead of a straight line. When we did that, the fit became worse ($R^2 = 0.51$), and the B_1-estimate became negative ($p = 0.002$). This is difficult to interpret. For pragmatic reasons, we prefer the simple model (3) with lags based on visual inspection.

Expression (4) gives the link between the cohort LE $e_0^c[g]$ for a cohort born in year g, and the period LE $e_0^c[g]$ years later. This is very similar to two other notions developed earlier, namely (i) the adjusted period LE $e_0^*[t]$ introduced by Bongaarts and Feeney (2002, 2003) and (ii) the cross-sectional average length of life (*CAL*) of Brouard (1986) and Guillot (2003). Goldstein (2006) studies the case in which the age schedule

of period mortality rates is progressively shifted to higher ages – the so-called Linear Shift Model. He shows two things: First, under these conditions, $e_0^*[t]$ equals $CAL[t]$; second, the cohort LE in year t ($e_0^c[t]$) equals the CAL in calendar year $\tau = (t + e_0^c[t])$, i.e., in the year in which cohort t reaches an age equal to its own LE. Under this model, expression (4) gives us the possibility to link the adjusted period LE in year τ to the LE in that year, and hence also to the cohort LE of those born in year t. We have not done so because an important assumption behind the Linear Shift Model is that the shape of the mortality age pattern does not change over time – it just shifts to higher ages. Figure 5 for Norway and Figure A-2 in the Supplementary Material for the other countries show that second and higher-order moments increase over time. Hence, the constant shape assumption is not realistic for the ten countries for which we have data.

A consistent finding in the literature is that the cohort LE increases faster than the period LE. In our model, a necessary and sufficient condition is that $B_1 < 1$, which turned out to be the case for Norway. We assume that one will always find a stronger increase in the cohort LE than in the period LE as long as age-specific mortality falls over a long time interval, but a general proof does not exist, to the best of our knowledge. For the special case in which age-specific death rates decrease by the same annual amount k for each age and over a long period, the proof is straightforward. Compared to a reference situation with an LE equal to e_0 and small k, the relative increase in the LE $\Delta e_0/e_0$ is approximately $\bar{x}$, where $\bar{x}$ is the mean age of the stationary population associated with the reference life table (Keyfitz and Caswell 2005: 79). Consider relative changes in period LE between years t and t+1, and in cohort LE for cohorts born in these two years. They increase by factors approximately equal to $k\bar{x}_p$ and $k\bar{x}_c$, respectively. Now $\bar{x}_c > \bar{x}_p$ because the survival function $l_c(x)$ of the cohorts born in year t is better, for all ages $x > 0$, than that of the synthetic cohort of year t with $l_p(x)$. The ratio of absolute increases in cohort LE and period LE $e_0^{c\prime}/e_0^{p\prime} \approx (\bar{x}_c e_0^c/\bar{x}_p e_0^p)$, which is larger than 1. Hence, cohort LE increases faster than period LE, for two reasons: The area under the survival curve (e_0) is larger, and the form of the survival curve is more rectangular (as reflected in a high mean age $\bar{x}$).

7. Conclusions

We have analysed the age at death distribution (AADD) resulting from life table calculations, conventionally known as the $d(x)$-column of the life table. For a long series of period and cohort life tables, we have derived analytical expressions for the moments of the period distribution, written as a function of the cohort moments. The relationship is based on an empirical regularity that we found in observed period and cohort mortality data for ten countries for which the HMD contains cohort life tables:

Denmark, England and Wales, Finland, France, Iceland, Italy, the Netherlands, Norway, Sweden, and Switzerland. We noticed that for many ages x above 30, the relationship between the cohort-$d(x)$ – written as $\delta(x)$ in this paper – and the period-$d(x)$ resembles a straight line, with some exceptions.

We restricted ourselves to mortality beyond age 30 and analysed the trends in the period LE and in the variance/standard deviation in the period AADD. We gave detailed illustrations based on Norwegian data. The Supplementary Material contains summary results for nine additional countries.

We assumed that cohort LEs of Norwegian men and women are linear functions of time. This is a reasonable assumption for men born 1890–1923 and women born 1880–1923. We studied the link between period and cohort LEs. In one case, we compared each birth cohort with the calendar year in which that cohort would reach an age equal to its own LE ('forward-looking lag'). For Norway, the cohort LE always exceeds the period LE with this specific lag. In a second case, we analysed the 'backward-looking' lag, i.e., the number of years it takes a period LE to reach the LE of a cohort born in a particular year. Consistent with the findings of others, our expressions imply that the lags for Norwegian men and women increase as mortality improves. The backward-looking lags are 70 years (men) and 75 years (women) for the most recent Norwegian cohort for which we have enough data, i.e., those born in 1923. Finally, the stagnation of the period LE of men in Norway and in many other Western countries in the 1950s and 1960s goes together with a change in the shape of the AADD of cohorts born in the first half of the 20th century. The first moment (the LE as a measure of location) and the second moment (the variance plus the LE squared as a measure of spread) develop very regularly over time. We suspect that the distortion is caused by trend shifts in the third moment (which reflects skewness) or higher order moments (reflecting other features of the AADD of cohorts). Underlying this change in the period AADD is a change in major causes of death for middle-aged men, from infectious diseases before World War II to cardiovascular diseases after the war.

For a number of industrialized countries, the improvement in longevity in the 20th century went together with a regular compression of mortality around an ever-increasing average age at death. We analysed the link between the variances of the cohort and the period AADDs (ages 30 and over) as measures of expansion/compression. The data for Norway shows that compression in period mortality, expressed in terms of time changes in the variance of the AADDs, is approximately 40% slower than in cohort mortality. An important new result for compression in the period AADD is that the cohort variance and the cohort LE drive the period variance up, while the period LE presses it down. Whether the period variance will increase or decrease over time is an empirical question. The mortality age distributions of Norwegian men born after 1890 and Norwegian women born after 1880

fulfil the conditions for decreasing variance in the period AADDs, and hence we observe ongoing compression.

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Supplementary Material: Period and cohort mortality in nine European countries

Figure A-1 below shows scattergrams for the relationship between period probabilities $d(x)$ and cohort probabilities $\delta(x)$ of men and women in nine countries. The red lines represent strict equality between period and cohort probabilities ($\delta(x) = d(x)$) and are plotted as reference. Figure 3 in the main text gives the same plot for Norway. The graphs suggest that the relationship between the two variables is linear, although the relationship is not always linear. For ages 70 and 80, the patterns deviate more strongly from a straight line than for the other ages, for reasons explained in the main text. Large populations (England and Wales, Italy) show patterns closer to a straight line than small populations (Iceland). The fit to a straight line is better for women than for men (e.g., Denmark, France, and Netherlands).

Figure A-2 plots moments of orders one to five of the cohort AADD for men and women in nine countries (cf. Figure 5 for Norway). All moments show an upward trend, although the curves are somewhat less regular for men than for women. We assume that World War I caused the dip in cohort LE for English, French, and Italian men born 1885–1890, and that the Finnish Civil War of 1917 caused a similar dip for men born in Finland around 1888. With the exception of Danish women, the graphs suggest linear trends in the moments for birth cohorts born in two to three decades up to the 1920s. Lindahl-Jacobsen et al. (2016) analyse the stagnation of the LE of Danish women born between WW1 and WW2. Smoking was more prevalent in these generations than in cohorts born before and after the two wars. As a result, period LEs rose quickly from the mid-1990s because the survivors from the interwar generations were a selected group with relatively low death rates, compared to later generations at that age.

Figure A-3 shows LEs and standard deviations for men and women by birth cohorts (thick lines) and by calendar years (thin lines). Period effects caused by the two world wars are clearly visible for men in England, France, and Italy. The Spanish Influenza had a strong impact on all countries (possibly with the exception of Denmark): LEs fell abruptly in 1918, whereas standard deviations were higher than the trend suggested. In Denmark, the Spanish Influenza had a small impact on the LEs. Excess mortality due to the pandemic was relatively low: According to data presented by Johnson and Mueller (2004), Denmark is fourth among 17 European countries ranged from low to high excess mortality. The three countries with even lower excess mortality (Austria, Germany, and Russia) are not included in our data set.

Figure A-1: Scattergrams for the relationship between cohort probabilities δ[*x*,*t*] (horizontal axis) and period probabilities *d*[*x*,*t*] (vertical axis). Solid red lines are lines with slope 1, passing through the origin. Ages 30(10)90; cohorts 1835–1923; years 1875–2013. Men and women, Denmark

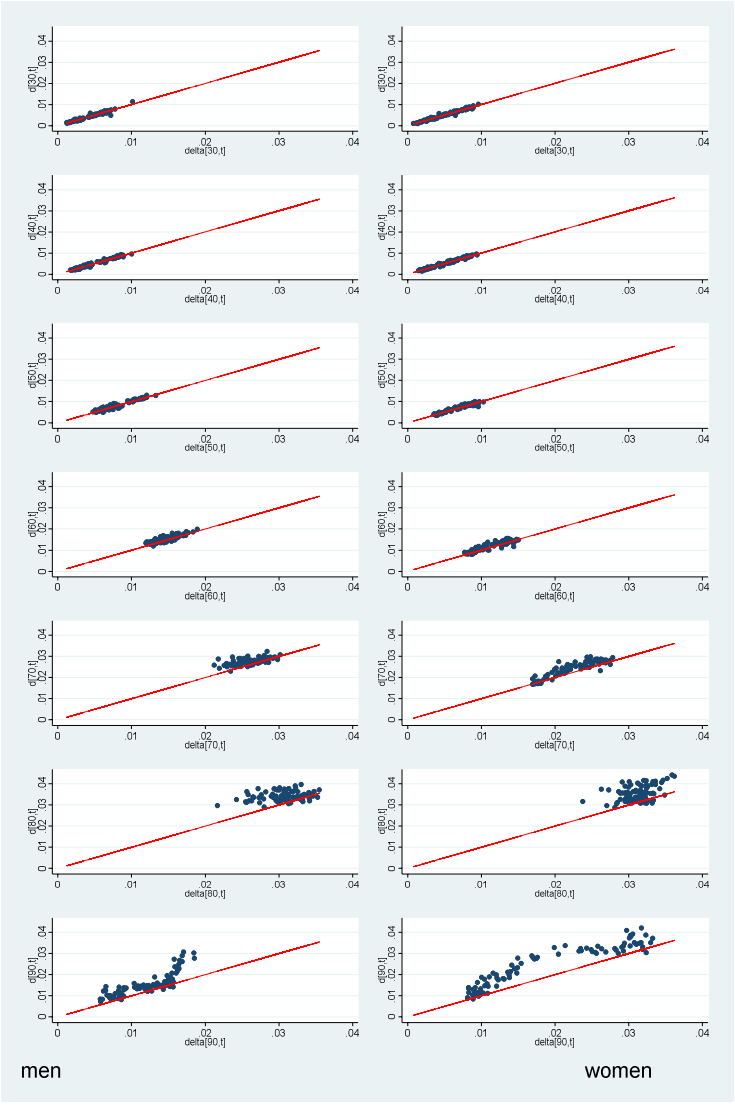


Figure A-1: (Continued) Scattergrams for the relationship between cohort probabilities δ[*x*,*t*] (horizontal axis) and period probabilities *d*[*x*,*t*] (vertical axis). Solid red lines are lines with slope 1, passing through the origin. Ages 30(10)90; cohorts 1841–1922; years 1871–2012. Men and women, England and Wales

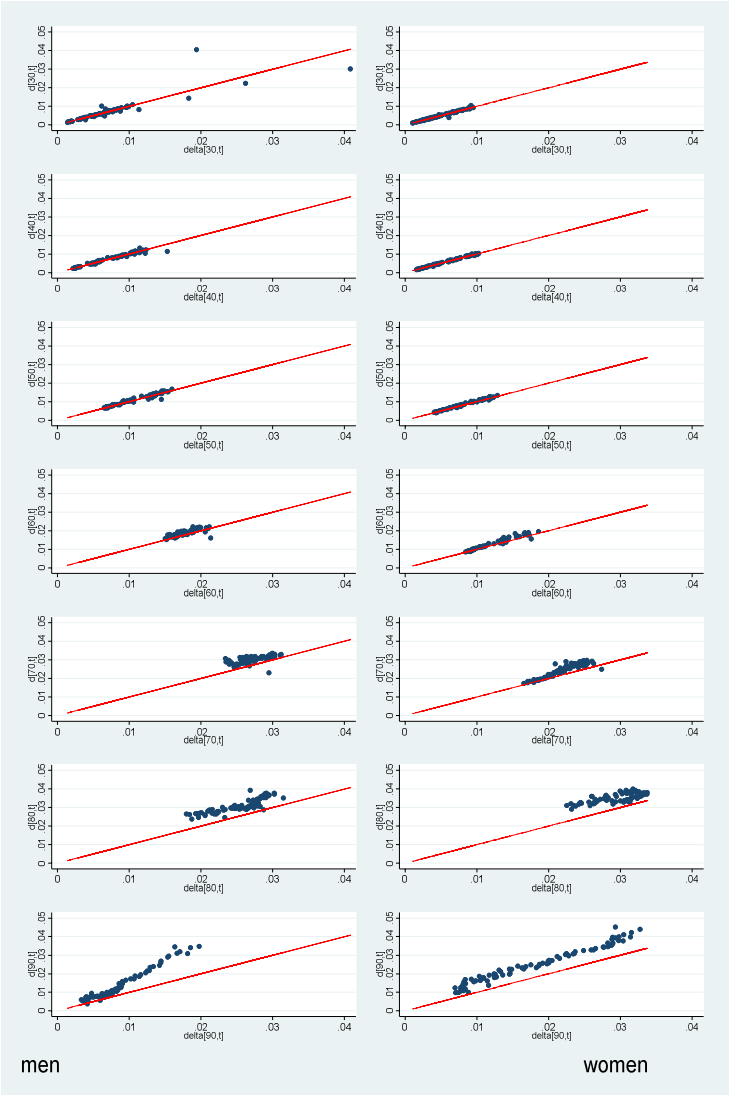


Figure A-1: (Continued) Scattergrams for the relationship between cohort probabilities δ[*x*,*t*] (horizontal axis) and period probabilities *d*[*x*,*t*] (vertical axis). Solid red lines are lines with slope 1, passing through the origin. Ages 30(10)90; cohorts 1878–1924; years 1908–2014. Men and women, Finland

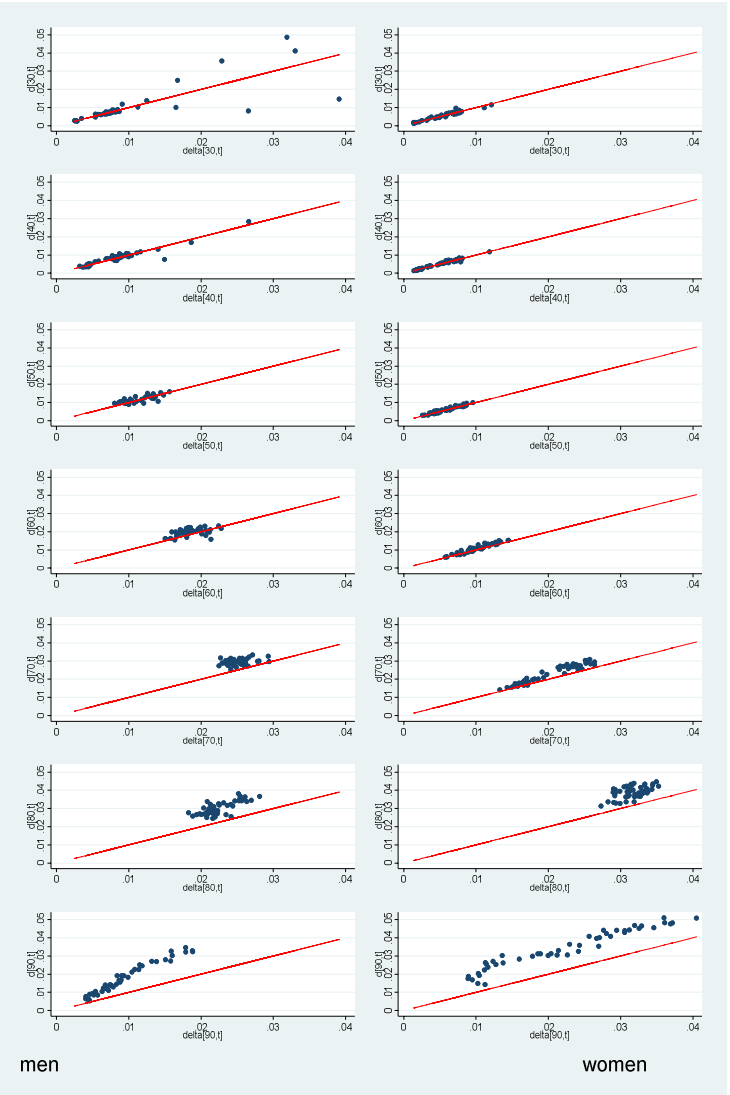


Figure A-1: (Continued) Scattergrams for the relationship between cohort probabilities δ[*x*,*t*] (horizontal axis) and period probabilities *d*[*x*,*t*] (vertical axis). Solid red lines are lines with slope 1, passing through the origin. Ages 30(10)90; cohorts 1816–1924; years 1846–2014. Men and women, France

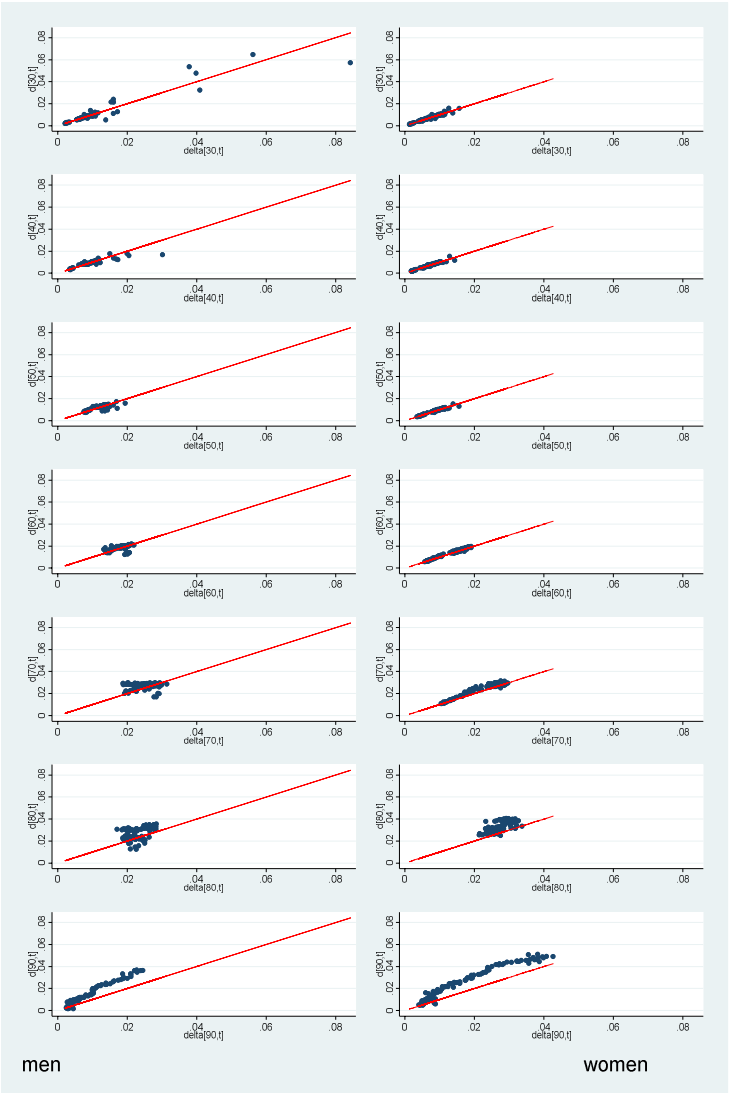


Figure A-1: (Continued) Scattergrams for the relationship between cohort probabilities δ[*x*,*t*] (horizontal axis) and period probabilities *d*[*x*,*t*] (vertical axis). Solid red lines are lines with slope 1, passing through the origin. Ages 30(10)90; cohorts 1838–1922; years 1868–2012. Men and women, Iceland

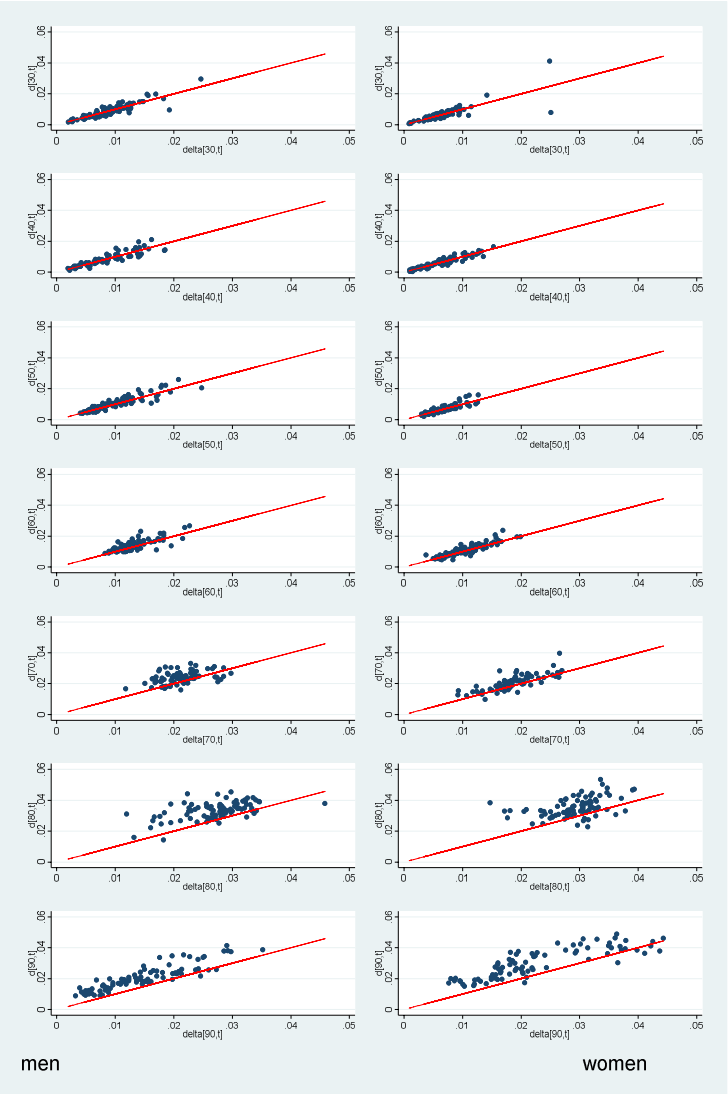


Figure A-1: (Continued) Scattergrams for the relationship between cohort probabilities δ[*x*,*t*] (horizontal axis) and period probabilities *d*[*x*,*t*] (vertical axis). Solid red lines are lines with slope 1, passing through the origin. Ages 30(10)90; cohorts 1872–1923; years 1902–2013. Men and women, Italy

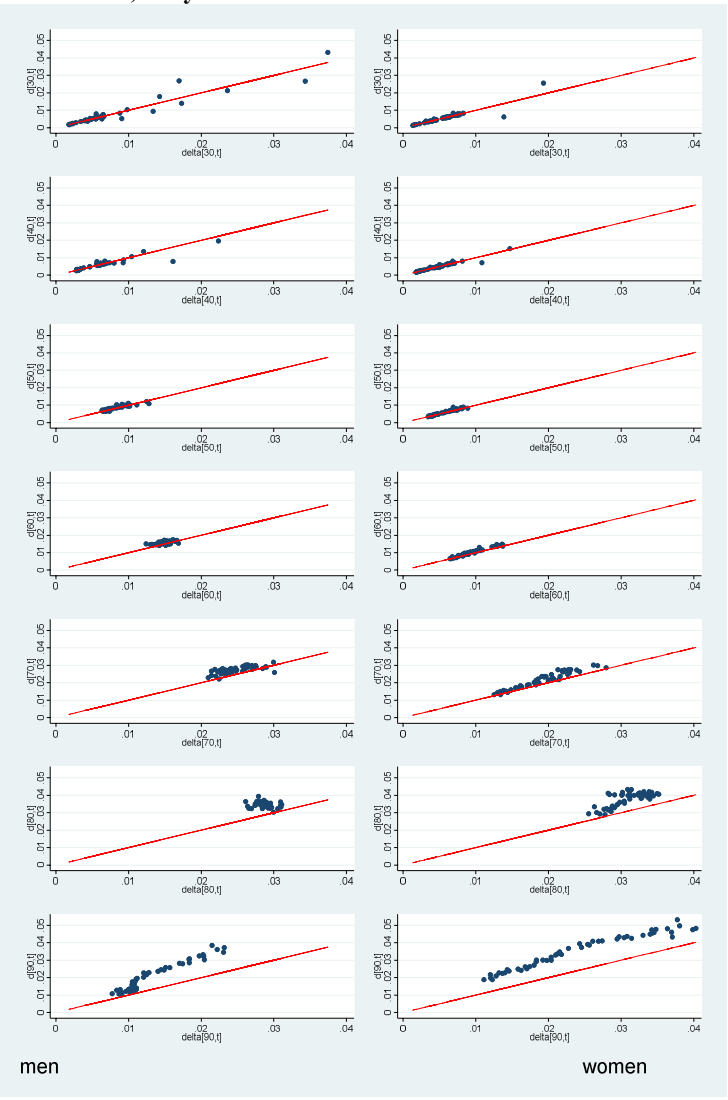


Figure A-1: (Continued) Scattergrams for the relationship between cohort probabilities δ[*x*,*t*] (horizontal axis) and period probabilities *d*[*x*,*t*] (vertical axis). Solid red lines are lines with slope 1, passing through the origin. Ages 30(10)90; cohorts 1850–1923; years 1880–2013. Men and women, the Netherlands

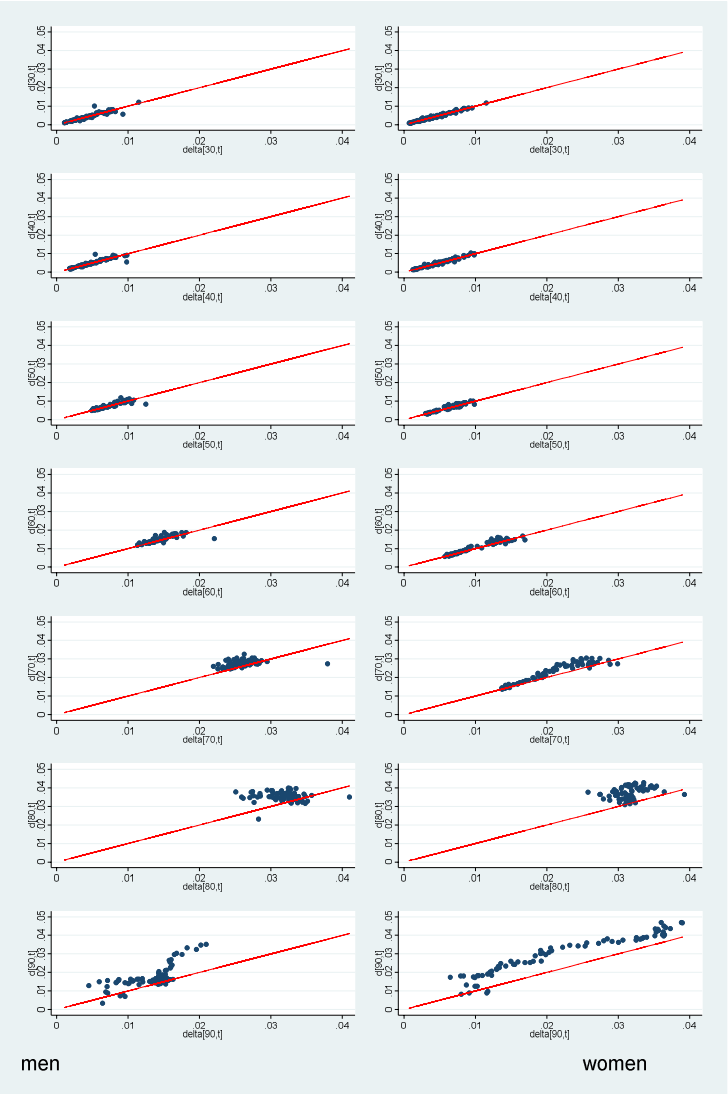


Figure A-1: (Continued) Scattergrams for the relationship between cohort probabilities δ[*x*,*t*] (horizontal axis) and period probabilities *d*[*x*,*t*] (vertical axis). Solid red line are lines with slope 1, passing through the origin. Ages 30(10)90; cohorts 1751–1923; years 1781–2013. Men and women, Sweden

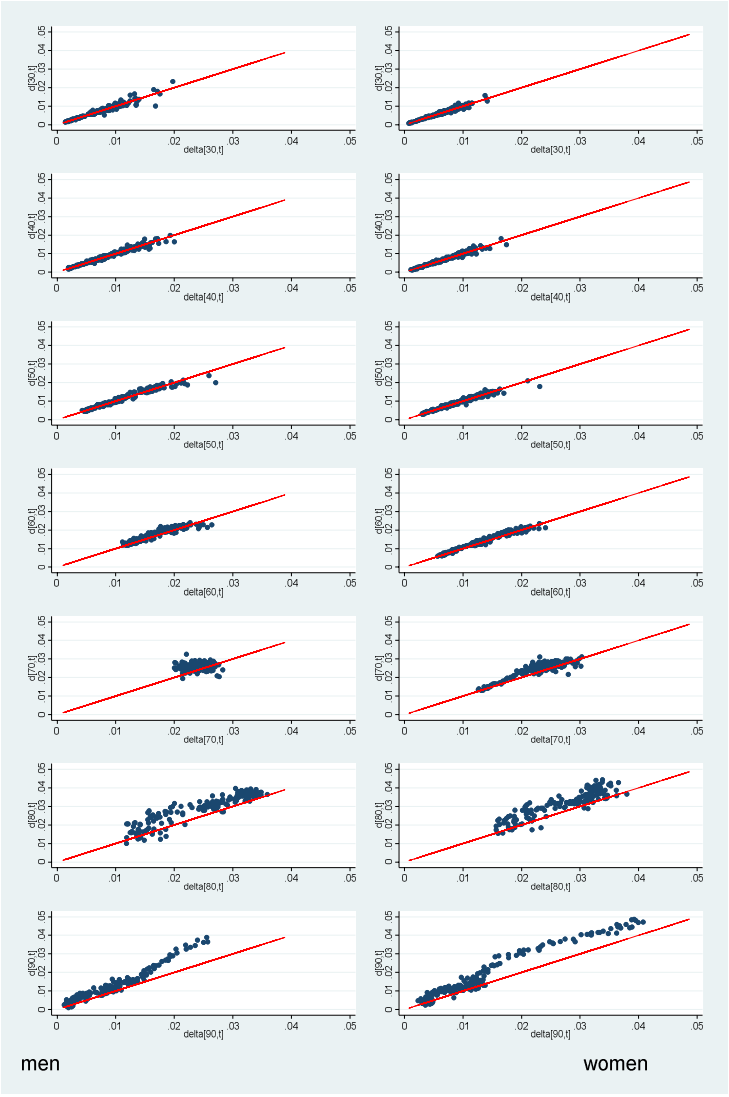


Figure A-1: (Continued) Scattergrams for the relationship between cohort probabilities δ[*x*,*t*] (horizontal axis) and period probabilities *d*[*x*,*t*] (vertical axis). Solid red lines are lines with slope 1, passing through the origin. Ages 30(10)90; cohorts 1876–1923; years 1906–2013. Men and women, Switzerland

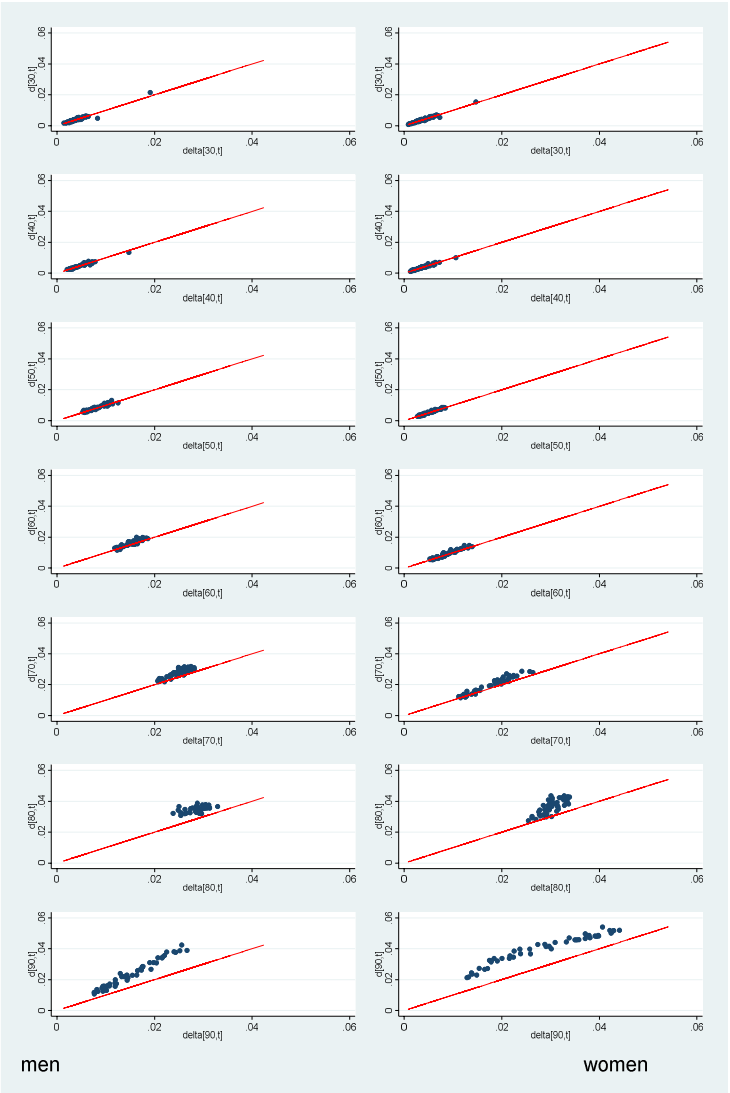
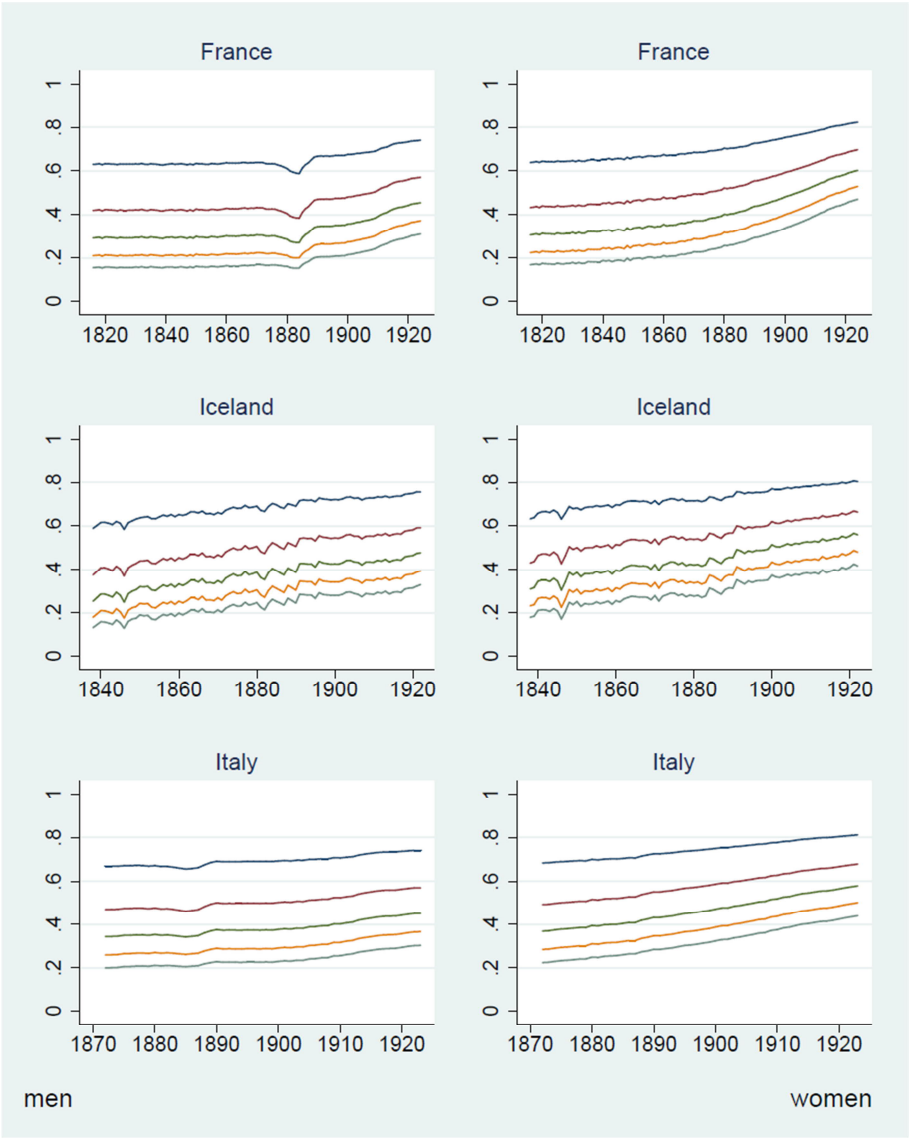


Figure A-2: Moments of orders one (uppermost curve) to five (lowermost curve) of cohort AADD. Men and women by year of birth

Note 1: different time scales.
Note 2: age *x* rescaled as *x*/100.

Figure A-2: (Continued) Moments of orders one (uppermost curve) to five (lowermost curve) of cohort AADD. Men and women by year of birth



Note 1: different time scales.
Note 2: age *x* rescaled as *x*/100.

Figure A-2: (Continued) Moments of orders one (uppermost curve) to five (lowermost curve) of cohort AADD. Men and women by year of birth

Note 1: different time scales.
Note 2: age *x* rescaled as *x*/100.

Figure A-3: LE (scale left, in years) and standard deviation (scale right, in years) by birth cohort and by calendar year. Men and women. Thick green line: cohort LE. Thick orange line: cohort standard deviation. Thin blue line: period LE. Thin brown line: period standard deviation

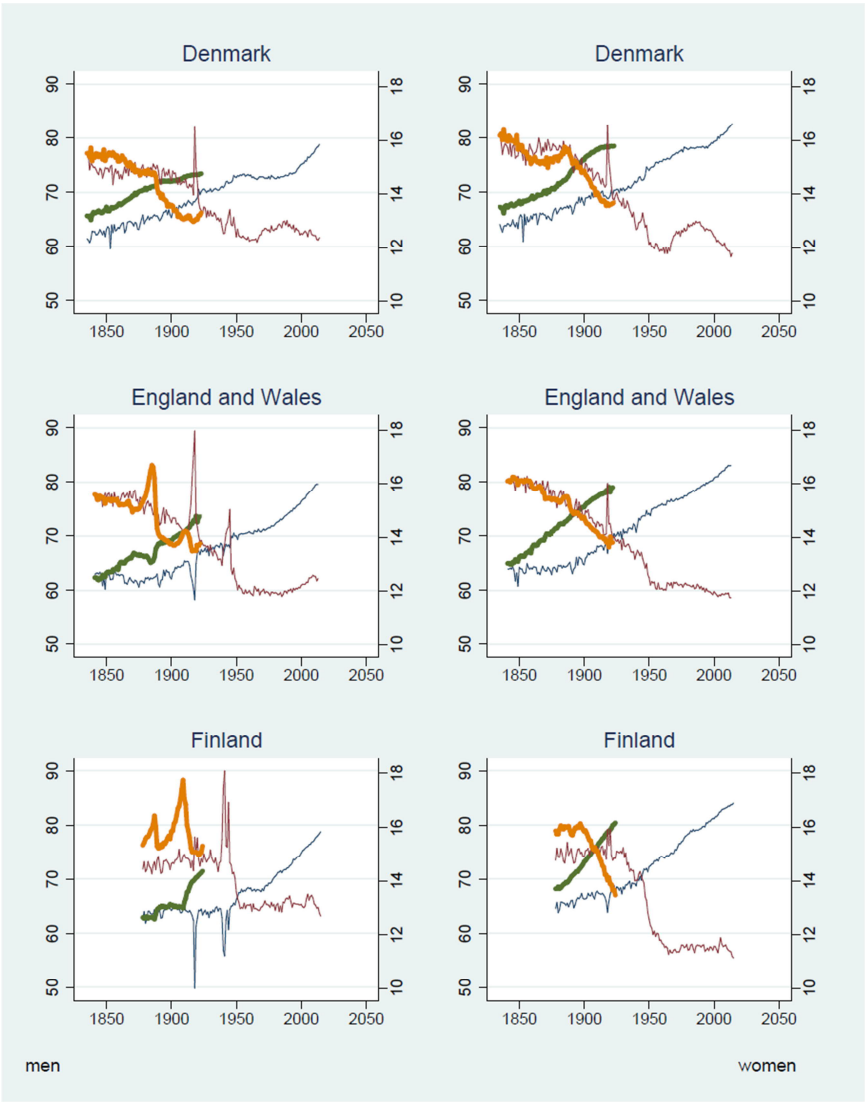


Figure A-3: (Continued) LE (scale left, in years) and standard deviation (scale right, in years) by birth cohort and by calendar year. Men and women. Thick green line: cohort LE. Thick orange line: cohort standard deviation. Thin blue line: period LE. Thin brown line: period standard deviation

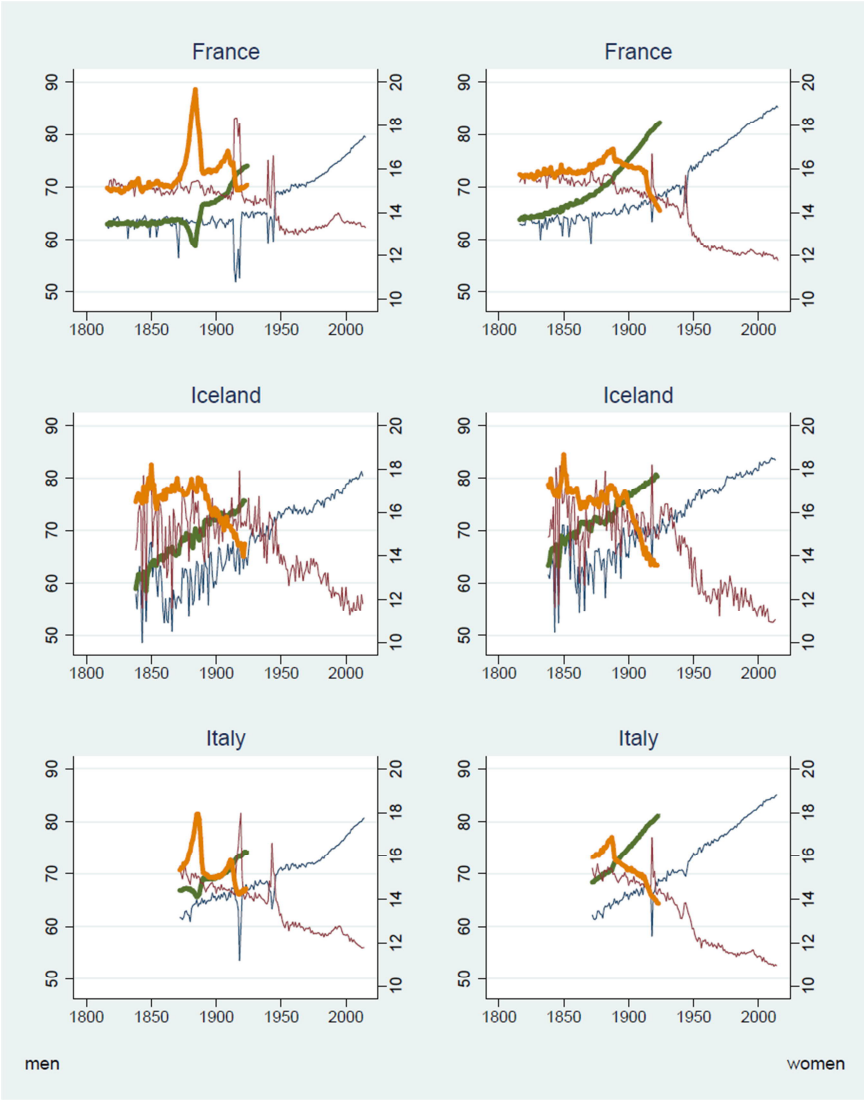


Figure A-3: (Continued) LE (scale left, in years) and standard deviation (scale right, in years) by birth cohort and by calendar year. Men and women. Thick green line: cohort LE. Thick orange line: cohort standard deviation. Thin blue line: period LE. Thin brown line: period standard deviation