Title: Median/modal death ages of pooled European male cohorts were near-constant for ~25/30 years.

Running head: Male death-age averages constant: ~30y

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ABSTRACT.

Background. Longevity is of considerable interest. Collation of recent data after World War II by the Human Mortality Database allowed analyses, previously unattainable, of modal death-ages for sufficient numbers of large European pooled cohorts. **Objective.** To track modes, means and medians (≥60 years old (y)) of all-cause mortality for both sexes. **Methods.** The only highest-quality, large-number Lexis data available were pooled from nine European countries: Denmark, Finland, France, Iceland, Italy, Netherlands, Norway, Sweden and Switzerland; raw-data modes (and means/medians ≥60y, plus thin-plate-splines), were analyzed, plus loess-smoothed equivalents for individual countries. **Results.** Here we show that for ~25-30 years (cohorts 1880-~1909) dramatic overall sex differences existed between pooled raw-death-age changes: male modal ages being near-constant (77.2y ± standard deviation 1.58y); females' increased. Overall, for available cohorts (1880-1904) male raw medians were exactly constant (76y); male means showed slight increase (0.0193y/year; compare female: 0.146y/year). Male deaths ≥60≤76y compared with >76y, as percentages of total, were near-equal, whereas in females the former decreased. Only after ~1910 did male modal ages rapidly increase (other averages not calculable). Individual country results showed that males in Finland, France, Switzerland were affected less than other countries. **Conclusions.** Results clarify previously knowledge concerning sex differences during this period. Despite improved environment during late adulthood, this did not translate into increased male longevity and earlier events might have sealed their fate, especially in Denmark, Italy, Netherlands, Norway, and Sweden. One hypothesis concerns long-term effects of the 1918-1919 influenza pandemic, perhaps directly relevant to the Covid-19 pandemic at present.

Keywords: Cardiovascular Disease; Demography; Longevity; Thin-plate Spline.

Declarations.

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INTRODUCTION

Longevity is of considerable interest. A lay person's view of increased longevity often means an expectation that adults have generally been living to increased ages barring early mishap (accidents and/or infectious disease), often presuming the existence of "healthy" ageing included in "life-style" mortality e.g. with cardiovascular diseases and cancer which have risk factors partly affected by life-style [1]. The question to be addresssed is whether this longevity has been increasing for males and females over the recent past for cohorts for which data is now available i.e. for those which have recently become extinct.

To answer this question the choice of descriptive parameters is critical. If cohort parameters are available then the parameter "life expectancy from birth" is in general not appropriate for this purpose as Ouellette and Bourbeau [2] have indicated: "when the latest figure of life expectancy is announced, many people may take it as the "typical" (i.e. most frequent) length of life. For example, life expectancy at birth for French females is [in 2011] around 85 years. They tend to miss the fact that the typical age at death has actually been over 90 years in the period life tables since 2006." In turn, the period parameters themselves (from which life expectancies are calculated) cannot give an accurate description of what has happened to the death densities at particular times for one particular cohort; a cohort being a group born at one particular time (in this article during one year). (Period parameters come from measured deaths at one particular time for a whole population group consisting of many successive cohorts.) The choice to study cohort data in the present study, which reflect the true life histories of given birth cohorts, is therefore based on a desire to know what actually happened to particular cohorts; for which we now have full data (i.e. from birth to extinction), with sufficient highest-quality extinct-cohort data only recently available. (Note that extrapolation towards future prediction is not analysed in the present article; note also that cohort averages are NOT available after the period

studied in the present article and in the period before, which includes direct world war deaths, could be severely distorted).

The three most common measures of location are the mean, median and mode and all can be used to represent "typical" ages of death [3]. The benefits of using these cohort average parameters (as opposed to life expectancy) have been extensively discussed by Horiuchi et al. [3] (as well as others) who also described the use of p-splines (as the optimal smoothing technique at that time; note that the graphs in Horiuchi et al [3] are plotted against calendar year, not cohort birth date).

Only the mode is independent of earlier deaths, and for another thorough analysis of both period and cohort modal parameters please see Cheung et al. [4] especially Fig. 4 in this reference which shows how cohort major modal ages (referred to in this reference as "late modal ages" and in the present article as "modal ages") have changed for cohorts from France, Italy and Sweden. Means and medians change according to numbers of earlier deaths and therefore we have arbitrarily chosen to calculate these from age 60 years old (see Kannisto on the merits or otherwise of the use of median cut-offs [5]). (Cohen and Oppenheim [6] have also analysed a type of median and show that these increased over the period studied for the ages birth to extinction for both males and females.) The age range >60 years old defines the "bulk" of deaths in the present article (roughly corresponding to Horiuchi et al.s' "heap" in [3]).

In addition to the use of raw data, thin-plate splines were used to smooth death-density distributions from excessive mortality changes in particular years. Instead of using parametric methods (e.g. gompertz, logistic, Weibull, quadratic, normal or skew-t [7]) which make some assumptions concerning underlying distributions, we follow Ouellette and Bourbeau [8] and Horiuchi et al. [3] in the use of non-parametric splines. Horiuchi et al. [3] discuss the "potential

theoretical importance of [the mode] in ageing research" and conclude that use of non-parametric fitting (p-splines) gave "noticeably different" modal trends to those from parametric fits (Gompertz, logistic, Weibull and their Makeham variants). Thin-plate splines can be regarded as more advanced than p-splines in the sense that they produce smooth surfaces infinitely differentiable with an interpretable energy function [9]. In addition they require no manual tuning, best fits were found automatically and visually they seemed to fit well right up to the oldest age. In contrast, with preliminary studies using p-splines the degrees of freedom had to be manually adjusted to achieve "best" fits (introducing questions regarding the validity of male/female comparisons if different degrees were needed for each).

It was determined to monitor death-density modes with highly-accurate cohort data from the Human Mortality Database (HMD; www.mortality.org), from all countries with Life-Table Lexis data (curated to the highest quality): Denmark, Finland, France, Iceland, Italy, Netherlands, Norway, Sweden and Switzerland, as well as with pooled data from all countries (referred to as "Europe", or with the largest two countries, Italy and France, removed: "EUM"). The subject of this paper therefore concerned all-cause old-age mortality (including life-style mortality and/or healthy aging) with cohorts from 1880 to 1919, chosen to include sufficient data both sides of the mode to allow spline fits or to avoid World-War-II direct-death increases in mortality. Raw data, or thin-plate spline interpolation, were analysed. Additionally, medians and means were calculated for cohort "bulk" deaths, arbitrarily defined as $\geq$60 y, using raw data or spline integration.

An important aspect of the work was that there were apparently no other high-quality Lexis cohort life-table data available anywhere for the period studied.

The pooled analysis aimed to use the maximum amount of the highest quality data (from the Human Mortality Database) in order to produce overall location parameters for mortality at

all ages (modes) and at ages from 60 years old (means and medians) for as many (male and female) cohorts as possible over the period studied with the maximum number of countries possible (nine countries, as in [6]). The reasoning behind this method is that the more data analysed the smoother the density distributions become, smoothing country to country variations and providing parameters which, in this case, can be used to represent "Europe" (used loosely here to refer to the pooled countries of Denmark, Finland, France, Iceland, Italy, Netherlands, Norway, Sweden and Switzerland). This is partly justified by the fact that these countries are spread throughout Europe and include western and central mediterranean countries as well as from central and northern Europe, the largest countries in the group being Italy and France: contributing approximately one third each to the numbers but, as we will see, contribute very differently to the overall results. The period was specifically chosen to avoid direct deaths from the world wars. It might be objected that the pooled data (inevitably) includes heterogenous data. However, it should be noted that the pooled results provide a useful starting point, and that further analysis, both here with individual countries and in future research, provide and will provide clarification of the pooled results. Contributions from individual countries were therefore also assessed by analysing individual country results using loess smoothing (quadratic, following ref. [6]).

The actual reasons why there might be differences in the way that male and female death density distributions changed over time cannot be concluded from this study. However, some speculation can follow from previous studies. It is well established that conditions in infancy, childhood and adulthood might affect adult mortality (sometimes divided into early, middle and late life events or factors). Adult mortality is affected by earlier life events, involving factors which affect growth and development of infants, epidemiology, nutrition, salubriousness (sewerage and housing) and schooling [4], as well as access to smoking, red meat and trends in

exercise, all of which affect later mortality [10]. During the period studied cardiovascular diseases gave the leading cause of world mortality with risk factors from obesity and/or smoking [11]. From this period it is possible that the "bulk" of adult deaths is increasingly determined by lifestyle, including "healthy" (if such a thing exists), mortality. The death distributions of all-cause mortality are therefore likely to become of great interest, especially in high-income countries, as these might start to reflect underlying biological mechanisms of aging.

It was therefore of interest to measure pooled cohort central-tendency location parameters which describe "bulk" death-ages (i.e. from 60 years of age) without being affected by younger deaths, i.e. the modes (and medians and means above a certain age), as well as to analyse the contributions from all individual countries. This was possible due to recent availability of sufficient cohorts with full data from birth to extinction.

The primary question investigated was how did the differences in male and female cohort death-density distributions change over the period of study, using the maximum amount of pooled highest quality data available (from the Human Mortality Database) and in individual countries separately. The main a priori hypothesis was that there were changes in the modal death-ages over time in either sex.

METHODS and METHODOLOGY.

Data sources.

High quality dx data was used from Cohort Life Tables from the Human Mortality Database (HMD; www.mortality.org, RRID:SCR_002370, electronic access from 2015 to 2018, last download 09-11-2018; last checked 12-08-2019), to take advantage of the decisions made by experienced demographers (methods for extracting data, and dx calculations, were double-

checked via email communication with HMD). Surprisingly, only few countries submit life-table cohort data, with many submitting period or other data for the study period (including e.g. USA, China). (Further high quality, large number data is apparently not available from other web sites.). Data from all available countries: Denmark, Finland, France, Iceland, Italy, Netherlands, Norway, Sweden and Switzerland, were pooled for analysis as well as analysed separately (source references are found in the Online-Resource-OR5).

For each country, all HMD cohort life-table data for one sex (Age interval×Year interval: 1x1) were downloaded into a "country" (Excel 2007; Microsoft, Washington, USA) file; plus Births data. In each country file, HMD columns were removed except: "Year" (which means year of birth in HMD cohort sections; here changed to "Cohort"), "age" and "dx"; and an additional column added: "Year of death" (="Cohort"+"age"; 110+ as 110). Separate Excel files were created for each cohort (hereon meaning deaths of one sex born in one year), and filtered cohort data from each country file transposed into each cohort file; plus births. In a cohort file, for each country an additional row “Actual (numbers of) deaths” (dx’) was calculated by [dx values×births/100000 (the radix)] (This was checked against the summed "actual deaths" for each country cohort and found to be accurate; for another use of the radix see [4]). dx' values from each country were summed to give dx' values for a European pooled cohort. By duplicating these files and removing data for Italy and France the pooled data for "EUM" were created. (All cohort files are combined in "dx_primed_creation.xlsx" Online-Resources-OR1,OR2.) The pooled dx' data, plus dx' data for individual countries, were transferred to the columns in "dx_primed_collation" Online-Resources-OR3,OR4.

Data from each cohort is for distinct individuals. Any (rare) missingness procedures were operated by HMD (www.mortality.org).

Period of study.

Cohorts born from 1880-1919 (n=40). Cohorts were chosen with enough data so that modes of spline fits would not be affected by edge effects: the early-age and old-age borders were the ages with numbers of deaths 3/4 of numbers dying at the mode. (see below, and vertical lines in Fig 1). These borders were independent of the modal age; earlier cohorts were prohibited due to direct excess male deaths around World War II and later cohorts through lack of data. Means and medians could only be calculated for cohorts 1880 to 1904 because of lack of old age data for later cohorts.

As any results from the pooled data were likely to have different contributions from each country, all countries were similarly analysed individually. Additionally, the entire study was also carried out with data pooled from seven countries (without Italy and France = "EUM").

Interpolation.

Interpolation was regarded as an important way to avoid abrupt changes found in the raw data (with the assumption that people die throughout the year, not just on one particular date !) and generally spline curve smoothing was noticeable even though pooled cohorts had rather a large numbers of deaths recorded.

Coding for raw data and spline analyses.

A main coding file (Word 2007, Microsoft; Online-Resource-OR5_Coding_INDIVIDUAL_countries) carried R coding to access the data in the Online-Resources-OR3,OR4. Analyses used the R statistical platform version 4.0 (https://cran.r-project.org). A broad plan of the coding follows. For R function stability data zeros were changed to 1×10^{-2} and 110+ y data was kept in a separate vector. For one cohort, a thin-plate spline was fitted to the actual numbers of deaths (dx', Online-Resources-OR3,OR4) from age 40 to the oldest age (ages centered to +0.5y), using the R function *Tps* [fields] with weights 1/dx' and lambda automatically found by generalized cross-validation. The function *predict* [fields] and 0.01 y

grids were used to determine age limits with 3/4 numbers of modal deaths (two vertical lines in Fig 1). Cohorts without enough data to allow computation of these limits were not analyzed (see above). Raw modes were identified. Interpolated modal ages were found as follows. A preliminary thin-plate spline mode was found by finding the age with spline maximum (with a tie, the midway point was taken). A fine grid of interpolated age points was created around this age to find the final interpolated thin-plate spline modal age.

Raw medians and means were calculated for each cohort from age ≥60 y to 110+ (as 110) y. For each cohort the function *integrate* [stats], using the same age range, estimated an interpolated median by integrating successive 0.001 ages until half of the integral for the whole age range was reached, or an interpolated mean by multiplying the integrals by age values and dividing by number of values. Integrals were checked graphically with raw data. Percentages of raw numbers of death fractions (over births) were also plotted.

Non-graphical coding in two parts (for females and males) of the coding file is identical with only one part-string difference i.e. "fem" or "MALE", the female parameters computed first. Each time the main coding file was run (for a country or pooled countries) it appended 20 columns to the "parameter_results.xlsx" Online-Resource-OR6: column modeage2dp = interpolated mode; moderawage = raw mode; rawMedian = raw median; rawMean = raw mean; IntMedian = interpolated median; IntMean = interpolated mean; bulkperctotal = bulk deaths (as % of total deaths); perc76overTotal = % of fraction >76 y; perc60to76 = deaths (%) of fraction ≥60<76 y; perceld0 = deaths (%) of fraction ≥95 y; with prefixes for males (M) or females (F) and country: Denmark ("De" or "DEN"), Finland ("Fi", "FIN"), France ("Fr", "FRA"), Iceland ("Ic", "ICE"), Italy ("It", "ITA"), Netherlands ("Ne", "NET"), Norway ("No", "NOR"), Sweden ("Swe", "SWE") and Switzerland ("Swi", "SWI").

Graphical analyses.

Graphs of numbers of deaths against birth age for each cohort were drawn using *ggplot* [ggplot2] (see Figs 1:6). For individual country graphical visualisation of parameter results, loess smoothing was used with standard defaults (quadratic, span = 0.75). For all analyses residuals plots were generated. Final graphs were produced using graphics software (Irfanview, www.irfanview.com; Designworks version 3.5, Greenstreet Software, Huntingdon, UK).

Statistics.

Statistics coding, found in the "STATISTICS_CODING and RESULTS" Online-Resource-OR7, when run in R read data from Online-Resource-OR6, analysed statistics and produced further graphs. The gradient of linear models was used for description only (not for comparison). Initially comparative linear regressions were performed but in many cases the assumptions for linear regression were not met (mostly with heteroscedasticity) and only results from independence tests based on Kendall rank correlations are shown in the Tables. Kendall rank tests are standard tests used for non-parametric comparisons which might have tied data. For all male:female comparisons Kendall independence tests were carried out using Kendall correlation coefficients. The latter followed conversion from Kendall's tau to Pearson's r, as in [12], which is probably conservative. Male/female comparison statistical results are printed into Online-Resource-OR7, including effect sizes for linear regression and Kendall independence tests (using $r = Z/\sqrt{N}$). The alpha level for significance was set at 0.05 and all comparisons were two-tailed. Online-Resources are available for this paper and all analyses can be re-run using the coding and data files provided on (Mac; Apple Inc., Cupertino, California, USA or Windows; Microsoft, Redmond, Washington, USA) computers (further details in Online-Resource-OR5).

RESULTS.

From large sample-size cohorts, created by pooling life-table cohort data from nine European countries (separately for males and females), raw data or thin-plate spline fits allowed changes in pooled mortality density averages to be followed. Typical mortality density curves (using relative numbers of deaths estimated from dx data; dx') for males and females (cohorts with birth date in 1881 common era year; CE) are shown in Fig 1. Fig 1a shows the bump at ~65 y from direct male World-War-II deaths, which deterred use of cohorts before 1880 CE. (Narrow confidence intervals not shown (as these would be hardly visible) but can be generated by running Online-Resource-OR5.)

FIGURE 1

Fig 2 shows cohort modes from (a) raw data, or (b) thin-plate splines, against cohort birth date. Over the first ~30 years, dramatic differences between modal changes for males and females over time were evident (more clearly seen with thin-plate spline interpolation; Fig 2b): average modal ages for male bulks were near-constant whereas females showed a large and significantly different increase (see Table 1). (These results are almost identical if the two largest countries are removed from these pooled averages: see "EUM" graphs in Online-Resources-FigS1:FigS4.) Fig 3 shows why the overall male results were near-constant, with large dips in the death-density modal ages found for all countries apart from France, and over the first 30 years the modal ages decreased slightly for Sweden, Italy, Norway, the Netherlands and Denmark, whereas modal ages increased for France, Iceland, Switzerland and Finland. It should be noted that the thin-plate spline equivalents, while having overall similar results, show slightly different timings in the changes observed (compare Fig 3a with 3b). The countries with most submitted data showed

almost balancing and opposite trends, with a slight male increase over the first 30 years in spline modal age for France (0.048 y/year) but a decrease for Italy (-0.069 y/year). Female modal ages in general increased over this period, although in the first 10 years there were decreases for Finland and Norway (for Fig 3b the order of lines on the right borders is given in the Figure legend; the left border order is, from the top: Females (green dot-dashed lines): No, Ne, Fr, Swi, Ic, Swe, It, De, Fi; Males (red solid): No, Swe, Ne, De, It; Males (dots): Ic; Males (black longdash): Swi, Fr, Fi.)

FIGURE 2

FIGURE 3

Fig 4 and Table 2 show how pooled raw cohort means and medians ($\geq$60 y) changed over time: male medians constant at 76 y, whereas means increased slightly; female averages increased dramatically over this period. (Integrated median/mean graphs are similar, see Online-Resource-FigS5; and results are very similar if Italy and France data are removed: see Online-Resources-FigS1:FigS4) Fig 5 shows why the male averages were constant or near-constant over this period: in Sweden, Italy, Norway, the Netherlands and Denmark the average death-ages were either near-constant or showed an increase followed by a protracted decrease. In France, Switzerland and Finland there were either very slight increases for male medians or constant increases for the means (probably indicating that in these countries the oldest old were influencing changes to the male means). All female results show increases during this period (apart from in Norway during the first 5 years).

FIGURE 4

FIGURE 5

Kendall independence tests showed significant differences between sexes for pooled data for all averages (see Figs 2, 4) showing that these had moved differently over time (Tables 1,2; effect sizes given; note the Kendall rank tests simply show whether there were significant changes over time for each parameter, whereas the Kendall independence tests show the differences between males and females). (Loess-smoothed curves can be generated, i.e. equivalent to Figs 3 and 5, for all pooled countries ("Europe") or for pooled countries minus Italy and France ("EUM") by running Online-Resource-OR5 in R; the loess standard errors are very small for both males and females, presumably because of the very large datasets, in these graphs.)

Further statistics including effect sizes and confidence intervals for Kendall independence tests are given in Online-Resource-OR7.

As the median age for pooled male data was 76 y for the entire period of study, it was of some interest to discover how percentage fractions of deaths varied around this value. Fig 6 compares numbers of deaths (as percentages of total deaths) for the age fractions: bulk (as % of total deaths); from ages $\geq$60<76 y; >76 y and $\geq$95 y. For males the fractions $\geq$60<76 y and >76 y had near-equal percentages for the 25 years available. The actual percentages of these two fractions with males were near-constant for 15 years and then both increased, whereas with females the former decreased and the latter increased steadily.

FIGURE 6

If Online-Resource-OR7 is run in R then results and graphs can be obtained and, in addition, results are also printed into this file. All residuals plots were generated but slight curvatures in the data do not affect non-parametric statistical comparisons.

Table 1. Male, female MODAL and gradient death-age parameters from raw data or thin-plate splines, from data pooled from nine European countries.

Population fraction, Cohorts	Method[1]	MODES global mean ± S.D. (y)	MODES range (y)	Gradient[2] (y/y), Kendall p	Male vs. female independence test p value, effect size
MALES, 1880 to 1919 (n=40)	Interpolated	78.3 ± 1.51	76.0 to 81.6	0.0801, p=0.0140	p<0.001, r=1.17
	Raw data	78.2 ± 2.28	73.5 to 82.5	0.108, p<0.001	p<0.001, r=0.465
FEMALES, 1880 to 1919 (n=40)	Interpolated	84.3 ± 2.62	80.5 to 89.0	0.221, p<0.001	-
	Raw data	84.3 ± 2.83	78.5 to 89.5	0.219, p<0.001	-
MALES, 1880 to 1909 (n=30)	Interpolated	77.6 ± 0.578	76.0 to 78.5	-0.0251, p=0.0353	p<0.001, r=1.41
	Raw data	77.2 ± 1.58	73.5 to 80.5	-0.0214, p=0.558	p<0.001, p=0.684
FEMALES, 1880 to 1909 (n=30)	Interpolated	83.1 ± 1.90	80.5 to 87.1	0.208, p<0.001	-
	Raw data	83.1 ± 2.19	78.5 to 87.5	0.203, p<0.001	-

Note. [1]Thin-plate spline interpolation or raw data. [2]Line of best fit (not used for comparisons). Kendall p = Kendall rank correlation p value; -: value given in male row; S.D. = standard deviation; Column "global Mean"

= mean over all cohorts. All values given to three significant figures (or range: one decimal place).

Table 2. Male, female (≥ 60 years old, y) median or mean death-age parameters from raw data or thin-plate splines, from data pooled from nine European countries.

Population fraction, Cohorts	Method[1]	Global mean ± S.D. (y)	range (y)	Gradient[2] (y/y), Kendall p	Male vs. female independence test p value, effect size
MALE MEDIANS	Integrated	76.6 ± 0.177	76.2 to 76.9	0.00703, 0.216	p<0.001, 2.00
	Raw data	76.0 ± 0.00	76 to 76	0.00	not calculable[3]
FEMALE MEDIANS	Integrated	80.5 ± 1.20	78.6 to 82.6	0.161, p<0.001	-
	Raw data	79.9 ± 1.32	78 to 82	0.175, p<0.001	-
MALE MEANS	Integrated	76.7 ± 0.190	76.3 to 77.0	0.0184, p<0.001	p<0.001, 1.72
	Raw data	76.0 ± 0.200	75.6 to 76.3	0.0193, p<0.001	p<0.001, 1.74
FEMALE MEANS	Integrated	80.0 ± 1.08	78.4 to 81.9	0.146, p<0.001	
	Raw data	79.4 ± 1.09	77.7 to 81.3	0.148, p<0.001	-

Note. [1]Thin-plate spline integration or raw data. [2]Line of best fit (not used for comparisons); [3]not calculable: zero variance. Kendall p = Kendall rank correlation p value; -: value given in male row; S.D. = standard deviation; Column "global Mean" = mean over all cohorts. All values given to three significant figures (or

range: integer or one decimal place).

DISCUSSION.

Pooled analyses.

It can be seen immediately from the pooled raw results (Fig. 2a) that there is a large (statistically significant; Table 1) difference in modal death-age changes between males and females across the first thirty years of study, from 1880 to ~1909, with a fairly constant increase for females and near-constant modal death-ages for male pooled data. The pooled raw-data median and mean death-ages (≥60 years old (y), Fig 4) show similar differences in an even more striking fashion, with male median death-ages exactly constant (at 67 y) over the 25 years for which data was available. It can be conjectured that these pooled results, and the pooled European cohort dx' graphs from which they are obtained, represent the sum total of highest-quality human data knowledge concerning the mortality density of these cohort bulks, as it is possible that similar highest-quality data simply do not exist for these cohort birth dates.

Divergence between male and female modal death-ages during this period has been previously noted by many studies e.g. see [4] (and Fig 7 in this reference; with quadratic fits) in which French and Swedish data are shown for this period (note the x-axis in this Figure shows calendar year, not cohort birth date, and the study concerns mostly period comparisons). Beltrán-Sánchez et al. [13] stated that "a relatively new demographic phenomenon .... emerged among people born in the late 19th century", referring to the divergence.

In the present study the differences between males and females are emphasized using all the highest-quality data available, and precision regarding the timing of the changes in given by thin-plate spline fits. The thin-plate spline fits show similar results overall to those from raw data but show reduced variability around trend lines and reduced loess standard errors (which are very narrow in any case; loess lines and standard errors can be generated by running Online-Resource-

OR5). Even so, thin-plate splines are not necessary to show the main effects which can be seen quite clearly using raw data, and only the raw data graphs are shown in Fig 4 for means and medians (with the thin-plate spline equivalents in Online-Resource-OR5).

It might be argued that the smoothing of the modes provided by the thin-plate splines is too great but this is not the case. The male raw modal death ages (Fig 2a) give iterated downward slopes which correspond to peaks resulting from a particular years with excessive mortality which have affected several cohorts at once. That these have been correctly smoothed can be seen by running the main coding (Online-Resource-OR5: the graphs will run like a motion picture film) in which such peaks will be seen to move from right to left while the thin-plate spline mode is more stable.

It might also be tempting to compare the results obtained with those of life expectancy at birth, but the latter is inappropriate to answer the question raised if cohort parameters are available, and this has not been done (see Introduction; similar arguments apply to life expectancy from age 60 y). As Cheung et al. [4] have stated: "the advantages of using the late modal age at death .....[have been] regularly underlined in the past [14–16]". The study of "rectangularization" [17] or compression of mortality [4] could also be studied, although this phenomenon appears to be waning [6, 18]. This study has used some of the best techniques and only the highest quality data available to calculate cohort death-density modes using raw data or thin-plate splines. Other parameters could be used e.g. mortality rates, but we think dx' gives clarity.

It is recognised that pooled results contain heterogenous contributions from different countries (and perhaps cities), although the larger the datasets the smoother the density distributions (even without splines, see Fig 1). In the future as more data is collected it might still be useful to pool all highest quality data for analyses involving, for example, parametric

interpretations, because not only have the data points aggregated a maximal amount of random-noise averaging, but also reflect averaged systematic differences among the countries analysed and are therefore most likely to portray underlying biology.

Individual country analyses.

The individual country results in the present study show that female mean death-ages (Fig 5b) increased over the period studied in all countries, but for males only in Switzerland, France and Finland and Iceland (Icelandic results are regarded as anomalous as discussed below.) In Fig 3 in Horiuchi et al. [3] similar results can be seen for French males and females (note that in this reference these are plotted against calendar year, not cohort birth date); and other studies have previously presented modal death-ages [14–16, 19, 20]. In the present study, male mean death-ages in Norway, Sweden, the Netherlands, Denmark and Italy were either near-constant or decreased over the first 30 years, and all gave an overall decrease. However, it must be remembered that means are highly sensitive to changes in the death-ages of the oldest old and should therefore be treated with caution in their use as location parameters for "bulk" deaths.

It is clear from Fig 5a that the individual country male median death-ages (except perhaps in France) did not change in accordance with female medians, which all showed rapid increases in median death-age (with Finland and Norway delayed by around 10 and 5 years, respectively). For males in Switzerland, France, Finland and Iceland median death-age increases were observed over this 25 year period, but less than for females. In the five countries of Norway, Sweden, Netherlands, Denmark and Italy males showed decreasing or near-constant median death-ages over this period (with a hump with Finland medians resulting in an overall decrease).

(The graphs for thin-plate spline median and mean death-ages, which will be necessary for precision work regarding timing, are shown in Online-Resource-FigS5, but look fairly similar to the raw data graphs.)

With individual-country modal death-age results (Fig 3), males from the same five countries (Norway, Sweden, Netherlands, Denmark and Italy) showed decreases over the first 25 years followed by increases (giving overall decreases over the first 30 years). For males in Switzerland, France, Finland and Iceland overall increases were observed over the first 30 years, although Switzerland and Finland show dips in the modal death-ages at around 1900 and Iceland modes started decreasing from 1900. Technically, conclusions regarding modes are independent of other distributional characteristics (whereas the medians depend on the lower cut-off boundary chosen: here $\geq$60 y).

The modal results from thin-plate spline fits not only show reduced standard errors around the loess lines, but also the timings of some modal changes are noticeably different between raw data and thin-plate spline fits (compare Fig 3a and 3b; for example, the peak for male Iceland modes is at ~1898 for raw data, but ~1902 for thin-plate splines, and the male lines for Netherlands and Denmark differ). It is expected that p-splines would also give different timings but not necessarily exactly as for thin-plate splines, and the latter should be regarded as optimal. The robustness of the thin-plate spline fits can be seen visually by looking closely at fits to the oldest old up to 110 y in the cohort graphs in Fig 1 and in those seen if the main coding is run from Online-Resource-OR5: the fits appear to be good in every case, despite the low amounts of data at the oldest ages.

Overall, individual country results provide clarification as to why large differences between males and females exist in the pooled results, allowing two groups of countries to be defined: Group A, with interpolated modal decrease over 30 years: Norway, Sweden, Netherlands, Denmark and Italy; Group B with an increase: Switzerland, France, Finland and Iceland (anomalous). It is clear from the methods and presentation used that in Group A countries the male average bulk death-ages were either near-constant or decreased over the period of cohort

births from 1880 to ~1909, in contrast to the variable but overall increases in Group B. Of importance, this shows that the two countries contributing the largest populations, France and Italy, had opposing changes in male average death-ages (and the remaining seven countries, referred to as "EUM", gave overall constant pooled male averages, see Online-Resources-FigS1:FigS4). This is surprising given, for example, that Italy and France were both affected greatly be the two world wars and that they both (or at least France in part) have Mediterranean diets and climate.

Study limitations.

(1) As Crimmins et al. [21] have indicated, conclusions might well only apply to the countries studied. For many large countries e.g. the USA or China, life-table Lexis data were not available for the period studied, and caution is necessary to draw conclusions from other data types e.g. period data. We could not add data similar to that analyzed because, as far as we know, none exists. (2) The nine countries contributed ~30% of the non-Russian/non-Ottoman European population [22]; pooled conclusions do not necessarily apply to Europe as a whole, and further data curation is awaited. (3) Although conclusions regarding modes are independent of population fraction, medians and means are affected by the fraction analysed (here $\geq$60 y) and medians and means would change if another fraction were considered.

Health, stress and mortality.

It is important to realise that we do not know the reasons as to why the male average death-ages decreased in Group A countries over this period. In a recently published and authoritative article by Crimmins et al. [21] concerning health, morbidity and mortality they state that "our strongest conclusion is that male/female differences in health are highly dependent on historical time and geographic location." This is certainly true for the countries analysed; there do not seem to be immediately obvious collective differences between the two country groups (although one might

be found !). We sincerely hope that the cohorts studied showed a quirk in male mortality parameters due to historical events which will not be repeated, but this cannot be guaranteed.

It is possible that male mortality in Group A countries was predominantly affected by early or middle-age factors and not late-age factors, or that there were serious male-biased factors which off-set positive late-age factors, for two reasons: (1) during the period in which these males were actually dying (at least after World War II), there has been a fairly constant increase in gross domestic product per person, health care, diet and general living standards in all the countries studied, which suggests that mortality in these males was either not affected by these events or there were factors which seriously offset any improvements; (2) the female average death-ages increasesd during the same period, and presumably females were exposed to many of the same factors. As we find it unlikely that late-age factors do not have effects on male mortality, we concentrate on male-biased off-sets, which might themselves have originated in early, middle or late-age. Even so, the results appear to provide weak evidence that early or middle-age factors, rather than late-age factors, are critical for male mortality.

Current thinking is, as mentioned in the Introduction, that as mortality due to infectious diseases decreased, cardiovascular disease, cancers and other chronic diseases became more important factors [21]. Mortality, however, has multivarious causes, and factors affecting infancy, adolescence and young adulthood in terms of growth and development in an epidemiological or nutritional context plus salubriousness, might well have more influence than is at first apparent. According to Cohen and Oppenheim [6] mortality depends on environmental, behavioural, sanitary, nutritional, and medical conditions as well as public health, social, political and economic organisation, food supplies, education and human values.

Beltrán-Sánchez et al. [13] analysed cohorts with birth dates between 1800 to 1935 in 13 countries and found that only after 1880 did male mortality rates increase relative to that of women.

Possibilities for the divergence in male/female mortality rates from 1880 onwards are thought to have arisen from males having:

(A) a greater vulnerability of males to cardiovascular diseases and a higher frequency of lethal conditions such as heart disease, stroke, and diabetes [21]. Perhaps most importantly for 50-70 year olds from the cohorts studied, dramatic sex differences were found in UK cardiovascular disease deaths which began ~1925, with male numbers increasing to ~1975, but female numbers decreased (see Fig 1 in [23]), and similar trends were found in Europe [24]. Quite possibly this increase in male-cardiovascular and other life-style disease frequencies contributed to the male longevity stagnation and after this period there was a measured fall in e.g. cardiovascular diseases, which might explain why the male modal death ages subsequently increased.

The genetic basis as to why men might be more susceptible to chronic diseases such as cardiovascular diseases is well known, with strong sexual dimorphism in aging and disadvantage in survival among men likely resulting from a male-specific mitochondrial mutation load, which might also affect (B) and (C) below [21].

(B) different behavioural responses to environment, including increased uptake of smoking [21] (relative to women), and with increased wealth an increase in access to red meat, alcohol and less overall exercise, which all increased the risk of cardiovascular diseases. It is thought that men are "more likely to engage in risky and dangerous behavior and women more likely to engage in health-seeking behavior" [21, 25]. Animal product consumption has been suggested to have significantly contributed to cardiovascular and/or cancerous diseases, with possibly greater negative effects in men than women [26]. Possibly males did not (or were unable to) take

advantage of beneficial environmental changes. We can speculate that as prosperity increased, women thrived with better life-style choices, whereas males might have relatively consumed more red meat and alcohol, smoked, and become less active, all risk factors for an earlier death [27].

(C) greater susceptibility to the long term effects of the 1918 influenza pandemic (colloquially known as the "Spanish Flu"). This hypothesis cannot be discounted as a major determinant yet because of the huge numbers of people infected (it caused acute illness in 25–30 percent of the world's population [28]) plus the fact that the sex differences in infection rates were enormous with an age-standardized death-rate difference of 174 per 100 000 [29]. According to Azambuja [30] white men born from 1880 to 1900 (20–40 years old) were preferentially killed because of an unusual immune response and survivors of infection might also have exhibited the same response giving a "primed" later susceptibility to coronary heart disease mortality [30]. (Note that if this was the prime determinant then, from the present study, susceptibility to this phenomenon might have declined slightly with age from adolescence to age ~40 y.)

While (C) is an interesting theory, and one which could be further investigated by comparing the timing of age-distributional infection rates in individual countries with the graphs in Figs 3b and Online-Resource-FigS5, it should be noted that recently some evidence has been gathered against this idea [31]. In this reference a small study compared >1000 military males who had contracted 1918 influenza with controls and found no differences in long-term survival [31], but as the authors mention this is probably not applicable to whole populations including non-military-personnel. As the world is currently in the midst of a Covid-19 pandemic, lessons learned from this period might be directly relevant to those living today.

Although explanations concerning cardiovascular diseases seem persuasive, in any case for the generation studied many had fought in two wars and the deferred effects of war

(psychological/injury) might have exacerbated behavioral or other differences. It may be that the cohorts studied show peculiar features with atypical mortality patterns which might reflect, for instance, harmful effects from the two world wars. Such effects can be seen among cohorts which directly participated in a war, or in people who during adolescence suffered from undernourishment due to a war. However, it must be remembered that war-related hypotheses need to account for differences (or lack of them) between countries at war or neutral countries: in particular note that Sweden and Switzerland were neutral throughout both world wars but are in opposite groups, and France and Italy were at war in both world wars but are also in opposite groups, as far as male mortality changes are concerned. Denmark, The Netherlands and Norway were neutral throughout World War I, and are in Group A, with decreases in average death-ages during this period, which might count against a war-related hypothesis (unless war had positive effects on later mortality).

Excess male migration is also a factor which might be considered (with predominantly healthy males migrating), although this is not thought to have affected collectively the groups of countries studied.

Any theory which purports to explain the presented results will need to explain why the scourge which apparently affected males in Group A countries affected males in Finland, France or Switzerland to a lesser degree. It is therefore interesting to note that with some cardiovascular risk markers at some times and in some countries, women have have had higher or similar cardiovascular risk to that of males [21]. As Crimmins et al. [21] have indicated, hypertension levels (a risk factor for cardiovascular diseases) are usually greater for males than females in most countries, but there are several countries where the prevalence of hypertension is higher for women.

The large sex differences in mortality shown during the first 30 years of the present study is unlikely to affect cohorts from ~1909 onwards, unless hypotheses concerning the 1918 influenza pandemic are correct and similar long-term effects are found for Covid-19, or war. According to Crimmins et al. [21] "sex differences in disease prevalence and mortality rates may recede" as risks for cardiovascular disease mortality reduce and as men and women behave more similarly. Data from the National Health and Nutrition Examination Survey (NHANES) showed that by 2010 there were no sex differences in mean age-specific cardiovascular risk markers at ages >50 years [32], and in the USA and Europe there has been increasing similarity in smoking habits between men and women [33].

The use of optimal parameters will be critical in the analysis of the timing of past events. Of some importance could be the fact that the timing, of e.g. changes in modal death-ages, sometimes depends on whether thin-plate splines have been used or not; the thin-plate splines theoretically providing a more accurate representation. In the future, as cohort parameter data catch up with data collected health dimensions (which have only been collected since the 1980s with national-level surveys on multiple dimensions of morbidity for large samples of both sexes [1, 21]) it will be the cohort parameters with optimal smoothing functions (e.g. thin-plate splines) which will provide the correct insights into the timing of past events.

The results from Iceland are anomalous (and note that the population is so small that Iceland data contribute only negligible changes to pooled data) but are of considerable interest because of a difference in timing in the downturn in male mortality parameters of approximately 20 years (see Figs 3, 5). It follows that, if it is assumed that similar deleterious factors operated in Iceland to those in other countries, it might be able to resolve these factors by simple investigation of relative timings, e.g. of the 1918 influenza pandemic, in different countries.

Although all-cause mortality studies by themselves cannot offer concrete conclusions to explain the timings of the mortality decreases in the cohorts studied, we hope that the way in which the data has been presented (which we think is optimal) will encourage further research into these cohorts - especially relative to the timings of the many possible factors associated with mortality.

Conclusions

In summary, there are many possible causes to the sex differences in the changes in mortality for cohorts born from 1880 to ~1909. Suspicion lies with changes connected with cardiovascular and other "life-style" diseases. It is a sobering thought that at least a part of a generation of men might in general not have taken (or been able to take) advantage of environmental changes for the opportunity for longer life, and might have suffered the long-term effects of the 1918 influenza pandemic and the deferred effects of war. The result showing that the bulks of males in the countries of Norway, Sweden, Netherlands, Denmark and Italy actually lived less long at the end of a 30 year period is an important presentation, and the exact timings of the downward trends as displayed by thin-plate splines are likely to be useful for important future analyses, perhaps directly relevant to the possible long-term effects of Covid-19.

References

Data source references (required by the Human Mortality Database rules) are found at the end of Online-Resource-OR5.

1. Seeman TE, Singer BH, Rowe JW, Horwitz RI, McEwen BS. Price of adaptation–allostatic load and its health consequences. MacArthur studies of successful aging. Archives of Internal Medicine. 1997;157:2259-2268.

2. Ouellette N, Bourbeau R. Changes in the age-at-death distribution in four low mortality countries: A nonparametric approach. Demographic Research. 2011;25:595-628.
3. Horiuchi S, Ouellette N, Cheung SLK, Robine J-M. Modal age at death: lifespan indicator in the era of longevity extension. Vienna Yearbook of Population Research. 2013;11:37-69.
4. Cheung SLK, Robine JM, Caselli G. The use of cohort and period data to explore changes in adult longevity in low mortality countries. Genus. 2008;64:101-129.
5. Kannisto V. Mode and Dispersion of the Length of Life. Population. 2001;13:159-171.
6. Cohen JE, Oppenheim J. Is a limit to the median length of human life imminent? Genus. 2012;68:11-40.
7. Clark JSC, Kaczmarczyk M, Mongiało Z, Ignaczak P, Czajkowski AA, Klęsk P, Ciechanowicz A. Skew-t Fits to Mortality Data—Can a Gaussian-Related Distribution Replace the Gompertz–Makeham as the Basis for Mortality Studies? The Journals of Gerontology: Series A. 2013;68:903-913.
8. Nadine O, Robert B. Changes in the age-at-death distribution in four low mortality countries: A nonparametric approach. Demographic Research. 2011;25:595-628.
9. Nychka D, Furrer R, Paige J, Sain S. Fields: Tools for spatial data. 2017
10. Robine J-M. Life Course, Environmental Change, and Life Span. Population and Development Review. 2003;29:229-238.
11. Beltrán-Sánchez H, Soneji S, Crimmins EM. Past, Present, and Future of Healthy Life Expectancy. Cold Spring Harbor Perspectives in Medicine. 2015;5:a025957.
12. Walker D. JMASM9: Converting Kendall's Tau For Correlational Or Meta-Analytic Analyses. Journal of Modern Applied Statistical Methods. 2003;2:525-530.
13. Beltrán-Sánchez H, Finch CE, Crimmins EM. Twentieth century surge of excess adult male mortality. Proceedings of the National Academy of Sciences. 2015;112:8993-8998.

14. Elderton WP. Graduation and Analysis of a Sickness Table. Biometrika. 1903;2:260-272.
15. Greenwood M, Irwin JO. The biostatistics of senility. Human Biology. 1939;11:1-23.
16. Gumbel EJ. La Duree Extreme de la Vie Humaine. The Mathematical Gazette. 1938;22:422.
17. Fries JF. Aging, natural death, and the compression of morbidity. The New England Journal of Medicine. 1980;303:130-135.
18. Wilmoth JR, Horiuchi S. Rectangularization revisited: Variability of age at death within human populations. Demography. 1999;36:475-495.
19. editor. A bio-actuarial approach to forecasting rates of mortality. Proceedings of the Centerary Assembly of the Institute of Actuaries. 2; 1950; 1950.
20. Le Bras H. Lois de mortalité et age limite. Population (French Edition). 1976;31:655-692.
21. Crimmins EM, Shim H, Zhang YS, Kim JK. Differences between Men and Women in Mortality and the Health Dimensions of the Morbidity Process. Clinical Chemistry. 2019;65:135-145.
22. Wood J. The Nuttall Encyclopaedia. London and New York: Frederick Warne.; 1920
23. Nikiforov SV, Mamaev VB. The development of sex differences in cardiovascular disease mortality: a historical perspective. American Journal of Public Health. 1998;88:1348-1353.
24. Glei DA, Horiuchi S. The Narrowing Sex Differential in Life Expectancy in High-Income Populations: Effects of Differences in the Age Pattern of Mortality. Population Studies. 2007;61:141-159.
25. Rogers RG, Everett BG, Saint Onge JM, Krueger PM. Social, Behavioral, and Biological Factors, and Sex Differences in Mortality. Demography. 2010;47:555-578.

26. Montesanto A, De Rango F, Pirazzini C, Guidarelli G, Domma F, Franceschi C, Passarino G. Demographic, genetic and phenotypic characteristics of centenarians in Italy: Focus on gender differences. Mechanisms of Ageing and Development. 2017;165:68-74.

27. Zheng Y, Li Y, Satija A, Pan A, Sotos-Prieto M, Rimm E, Willett WC, Hu FB. Association of changes in red meat consumption with total and cause specific mortality among US women and men: two prospective cohort studies. BMJ. 2019;365:l2110.

28. Taubenberger JK. The Origin and Virulence of the 1918 “Spanish” Influenza Virus. Proceedings of the American Philosophical Society. 2006;150:86-112.

29. Noymer A, Garenne M. The 1918 Influenza Epidemic’s Effects on Sex Differentials in Mortality in the United States. Population and development review. 2000;26:565-581.

30. Azambuja MIR. Spanish Flu and Early 20th-Century Expansion of a Coronary Heart Disease–Prone Subpopulation. Texas Heart Institute Journal. 2004;31:14-21.

31. Wilson N, Boyd M, Nisa S, Clement C, Baker MG. Did exposure to a severe outbreak of pandemic influenza in 1918 impact on long-term survival? Epidemiology & Infection. 2016;144:3166-3169.

32. Kim JK, Ailshire JA, Crimmins EM. Twenty-year trends in cardiovascular risk among men and women in the United States. Aging Clinical and Experimental Research. 2019;31:135-143.

33. Janssen F. Similarities and Differences Between Sexes and Countries in the Mortality Imprint of the Smoking Epidemic in 34 Low-Mortality Countries, 1950–2014. Nicotine & Tobacco Research. 2019;22:1210-1220.

Figure legends.

Fig. 1 Death (dx') distributions, 1881 CE. Numbers of deaths (estimated from dx) from male (a, x) and female (b, +) European pooled cohorts from 1881 common era year (CE) versus age of death (years old; y). Curves show thin-plate spline fits (narrow confidence intervals not shown but can be generated by running Online-Resource-OR5). Vertical lines show ages with interpolated numbers of deaths 3/4 of numbers dying at the mode. The second-world-war direct-death mortality bump is seen in the male, but not female, cohort. Source of raw data: Human Mortality Database (2019)

Fig. 2 Modal death ages: pooled European data. Female (+; solid line: 30 year trend) and male (x; dashed line) cohort modal death ages (years old; y) from (a) raw mortality (dx') data; or (b) interpolated thin-plate splines; versus cohort birth date (common era year; CE) or date cohort defined as extinct (at 110 y). Interpolated pooled modal-death ages show a near-constant increase with female, but not male, cohorts. Source of raw data: Human Mortality Database (2019)

Fig. 3 Modal death ages: Individual countries. Female and male cohort modal death ages (years old; y; loess-smoothed) from (a) raw mortality (dx') data or (b) interpolated thin-plate splines; from individual countries, versus cohort birth date (common era year; CE) or date cohort defined as extinct (at 110 y). Order (codes from text) in 3b from top, at right border: Females (green dot-dashed lines): It, Fr, Swi, Swe, No, Ic, Ne, Fi, De; Males (red solid): Swe, It, No, Ne, De; Males (dots): Ic; Males (black longdash): Fr, Swi, Fi. Standard errors (grey). Female modes

increased, but in five (red solid) countries, male modes gave negative net overall trend in modal death age over first 30 years. Source of raw data: Human Mortality Database (2019)

Fig. 4 Cohort (a) median, and (b) mean, death ages. Female (+; dashed line) and male (x; solid line) cohort (a) median or (b) mean death ages (years old; y) from raw mortality (dx') data, versus cohort birth date (common era year; CE) or date cohort defined as extinct (at 110 y). Raw male median-death ages are exactly constant over this period whereas females' increase. Source of raw data: Human Mortality Database (2019)

Fig. 5 Cohort (a) median, and (b) mean, death ages: Individual countries. Female and male cohort loess-smoothed (a) median or (b) mean death ages (years old; y) from individual-country raw mortality (dx') data, versus cohort birth date (common era year; CE) or date cohort defined as extinct. Order (codes in text) from top at right border: 5a: Females (green dot-dashed lines): Ic, No, Swe, Swi, Fr, Ne, De, It, Fi; Males (red solid): No, Swe, Ne, De, It; Males (black longdash): Swi, Fr, Fi. 5b: Females: Fr and No swap places; Males (red solid): Swe, No, remainder as for 5a. Standard errors (grey). For both averages, female death-ages increased but in five (red solid) countries male death-ages decreased or were near-constant over the first 30 years. Source of raw data: Human Mortality Database (2019)

Fig. 6 Percentage deaths at different age fractions. Numbers of cohort deaths (as percentages of total deaths) from raw data for (a) males and (b) females: (1) $\geq$95 years old (y); green solid triangles; (2) $\geq$60 to 76 y; blue solid diamonds; (3) >76 y; black empty diamonds; (4) >60 y; red empty triangles; versus cohort birth date (common era year; CE) or date cohort defined as extinct (at 110 y). Male fraction (2) percentage deaths are constant for ~15 years followed by an increase; female percentages decrease. Source of raw data: Human Mortality Database (2019)

Table legends.

Table 1. Male, female MODAL and gradient death-age parameters from raw data or thin-plate splines, from data pooled from nine European countries.

Table 2. Male, female (≥ 60 years old, y) median or mean death-age parameters from raw data or thin-plate splines, from data pooled from nine European countries.

Online Resources:

OR1_Female_dx_primed_creation.xlsx

Pooling calculations for European Female data from nine countries.

OR2_Male_dx_primed_ creation.xlsx

Pooling calculations for European Male data from nine countries.

OR3_Female_dx_primed_collation.xlsx

Collated dx' female data from EUROPE, EUM (= with Italy and France removed) and individual countries.

OR4_MALE_dx_primed_collation.xlsx

Collated dx' male data from EUROPE, EUM (= with Italy and France removed) and individual countries.

OR5_Coding_INDIVIDUAL_countries.docx

Main coding file. If all header rows are removed (apart from one) from OR3 and OR4 and these saved as .csv, in R OR5 will read OR3 and OR4 and produce results for chosen country, or EUROPE or EUM, which will be written to OR6.

OR6_parameter_results.xlsx

Results from running OR5.

OR7_STATISTICS_CODING and RESULTS.docx

Statistics coding file. If all header rows are removed (apart from one) from OR6 and this saved as .csv, in R OR7 will read OR6 and generate statistics and graphs. Results have been printed into OR7.

Online Resource Figure legends.

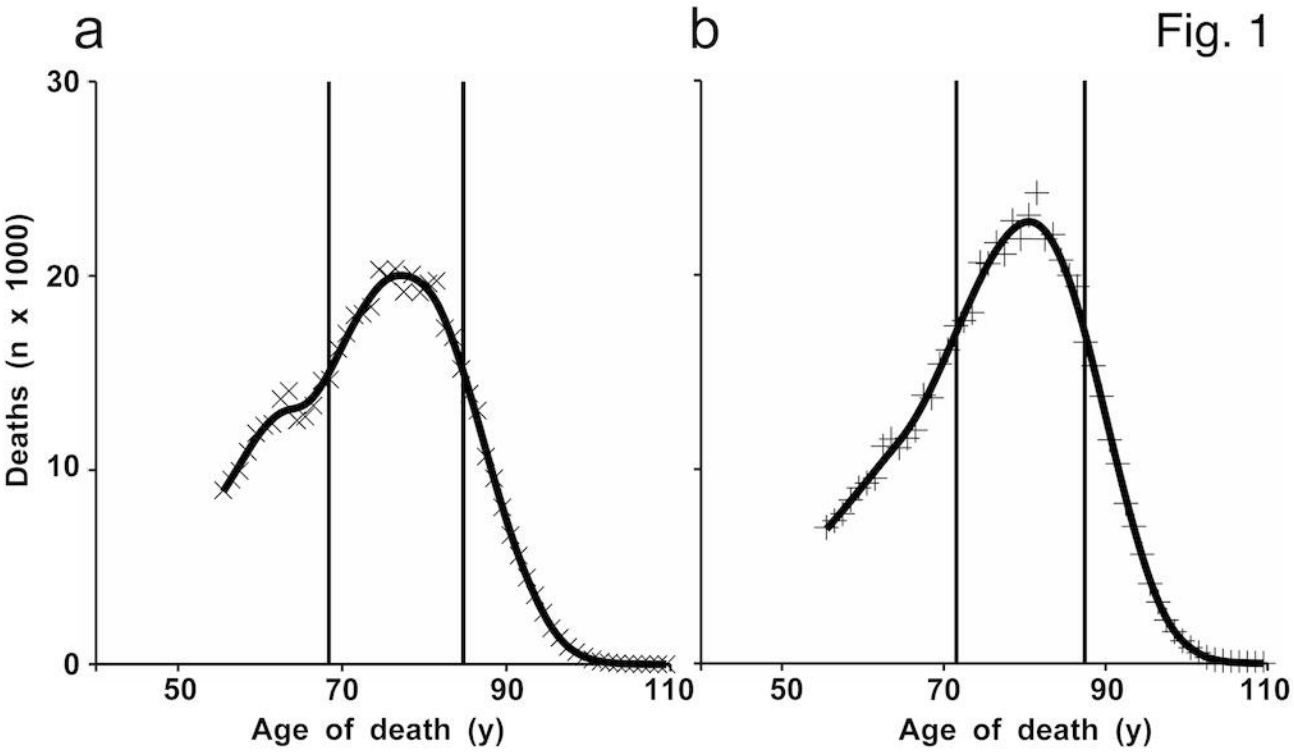
Online-Resource-FigS1 Seven countries. Death (dx') distributions, 1881. Data pooled from all countries except France and Italy ("EUM"). Numbers of deaths (estimated from dx) from male (a, x) and female (b, +) pooled cohorts from 1881 common era year (CE) versus age of death (years old; y). Curves show thin-plate spline fits (narrow confidence intervals not shown but can be generated by running Online-Resource-OR5). Vertical lines show ages with interpolated numbers of deaths 3/4 of numbers dying at the mode. In contrast to the full study, the second-world-war direct-death mortality bump can hardly be seen at all with the male data. Source of raw data: Human Mortality Database (2019)

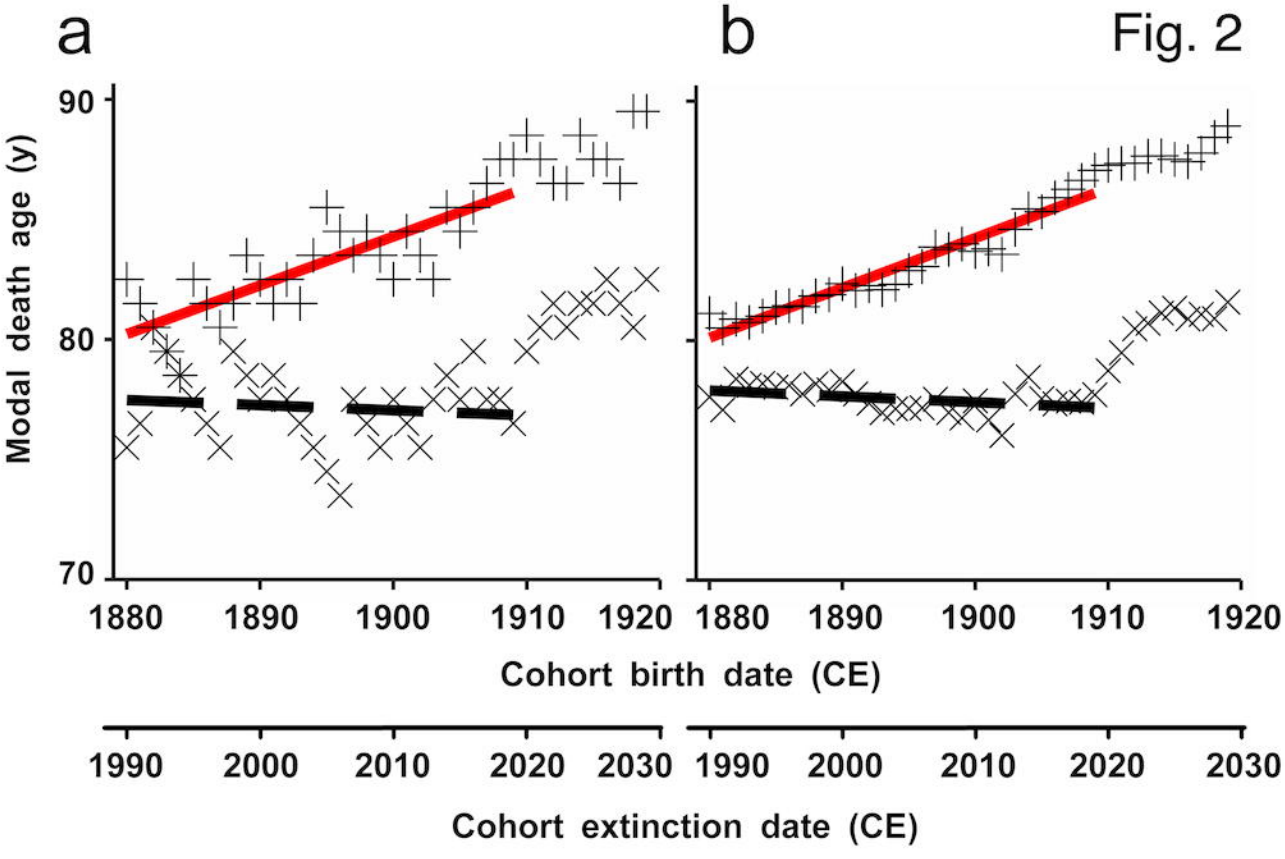
Online-Resource-FigS2 Modal death ages: seven countries ("EUM"). Female (+; solid line: 30 year trend) and male (x; dashed line) cohort modal death ages (years old; y) from (a) raw mortality (dx') data; or (b) interpolated thin-plate splines; versus cohort birth date (common era year; CE) or date cohort defined as extinct (at 110 y). Interpolated pooled modal-death ages show a near-constant increase with female, but not male, cohorts. Source of raw data: Human Mortality Database (2019)

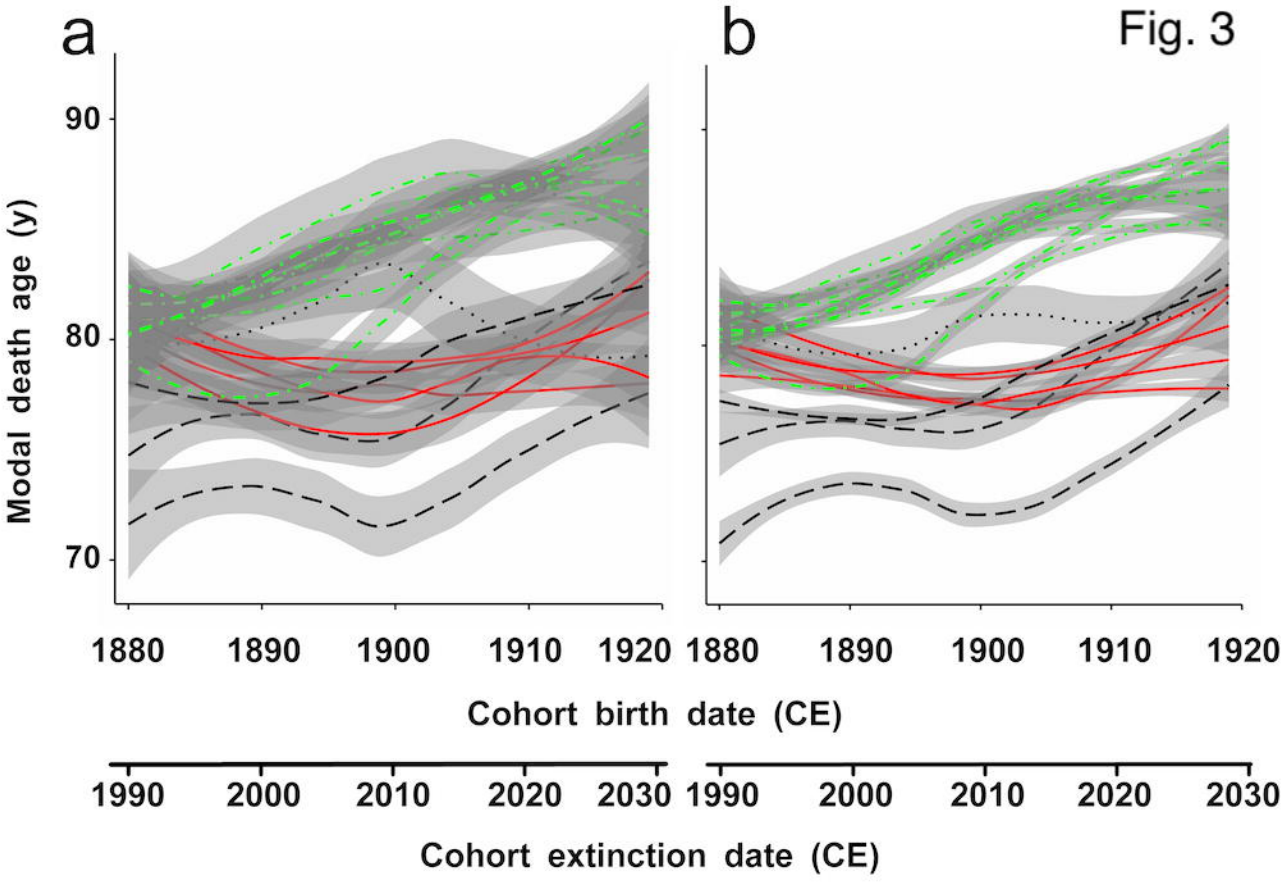
Online-Resource-FigS3 Seven countries ("EUM"). Cohort medians (a) and means (b). Female (+; dashed line) and male (x; solid line) cohort (a) median or (b) mean death ages (years old; y) from raw mortality (dx') data, versus cohort birth date (common era year; CE) or date cohort defined as extinct (at 110 y). Raw male median-death ages show a slight decline over this period whereas females' increase; raw male mean-death ages are almost constant. Source of raw data: Human Mortality Database (2019)

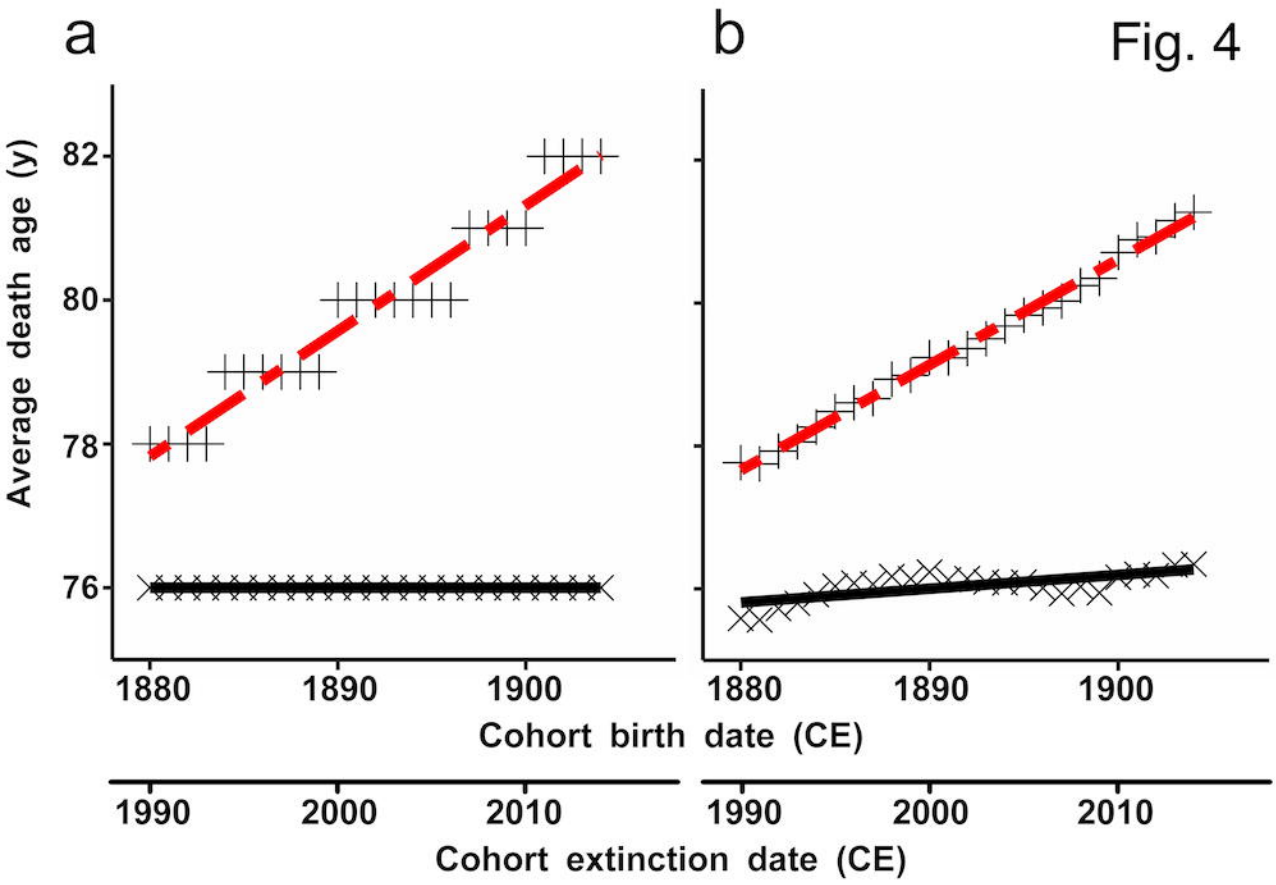
Online-Resource-FigS4 Seven countries. Percentage deaths at different age fractions. Numbers of cohort deaths (as percentages of total deaths) from raw data for (a) males and (b) females: (1) ≥95 years old (y); green solid triangles; (2) ≥60 to 76 y; blue solid diamonds; (3) >76 y; black empty diamonds; (4) >60 y; red empty triangles; versus cohort birth date (common era year; CE) or date cohort defined as extinct (at 110 y). Male fraction (2) percentage deaths increase slightly; female percentages decrease. Source of raw data: Human Mortality Database (2019)

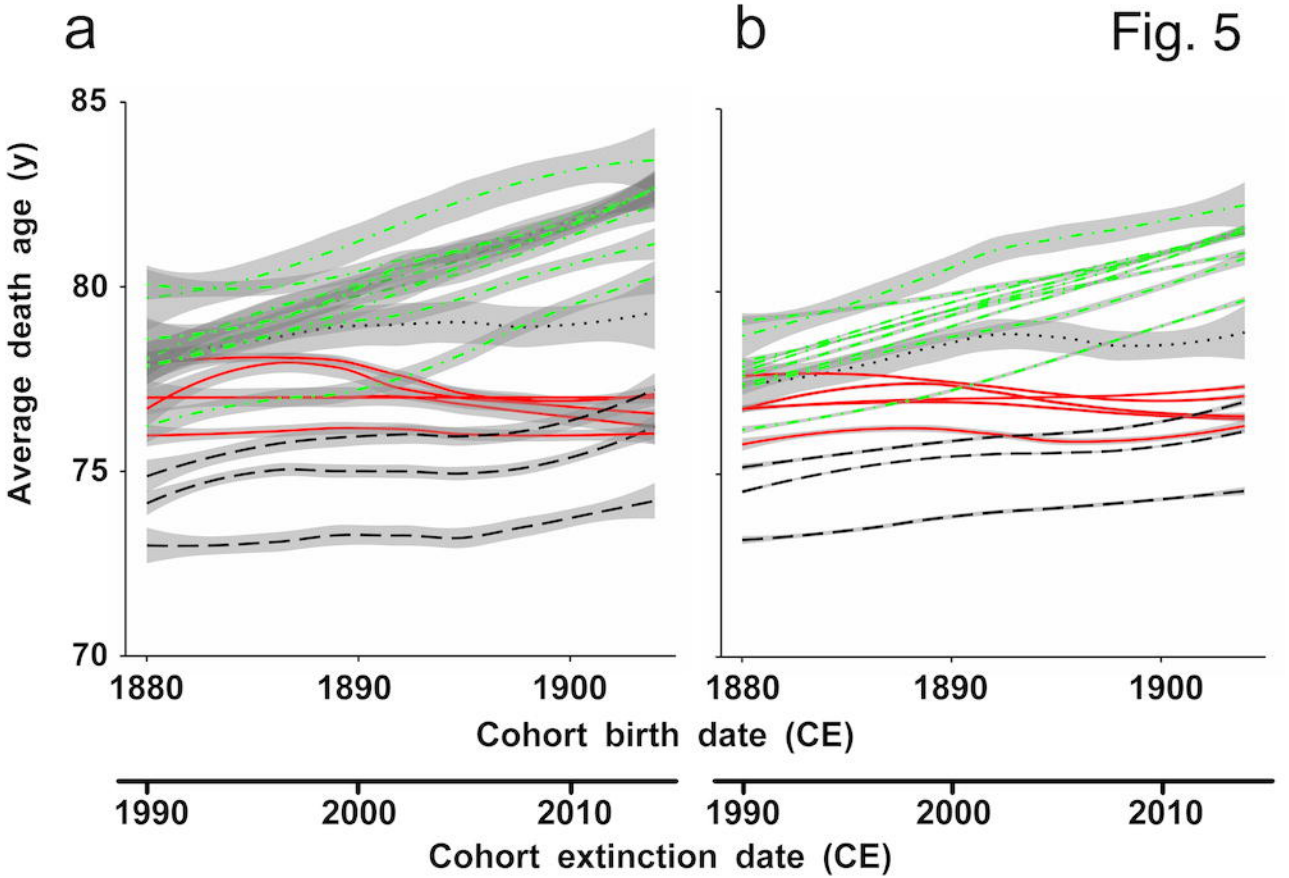
Online-Resource-FigS5 Cohort (a) median, and (b) mean, interpolated death ages: Individual countries. Female and male cohort loess-smoothed (a) median or (b) mean death ages (years old; y) from individual-country integrated thin-plate spline fits to mortality (dx') data, versus cohort birth date (common era year; CE) or date cohort defined as extinct. Order as in Fig 5. Standard errors (grey). For both averages, female death-ages increased but in five (red solid) countries male death-ages decreased or were near-constant over the first 30 years. Source of raw data: Human Mortality Database (2019)

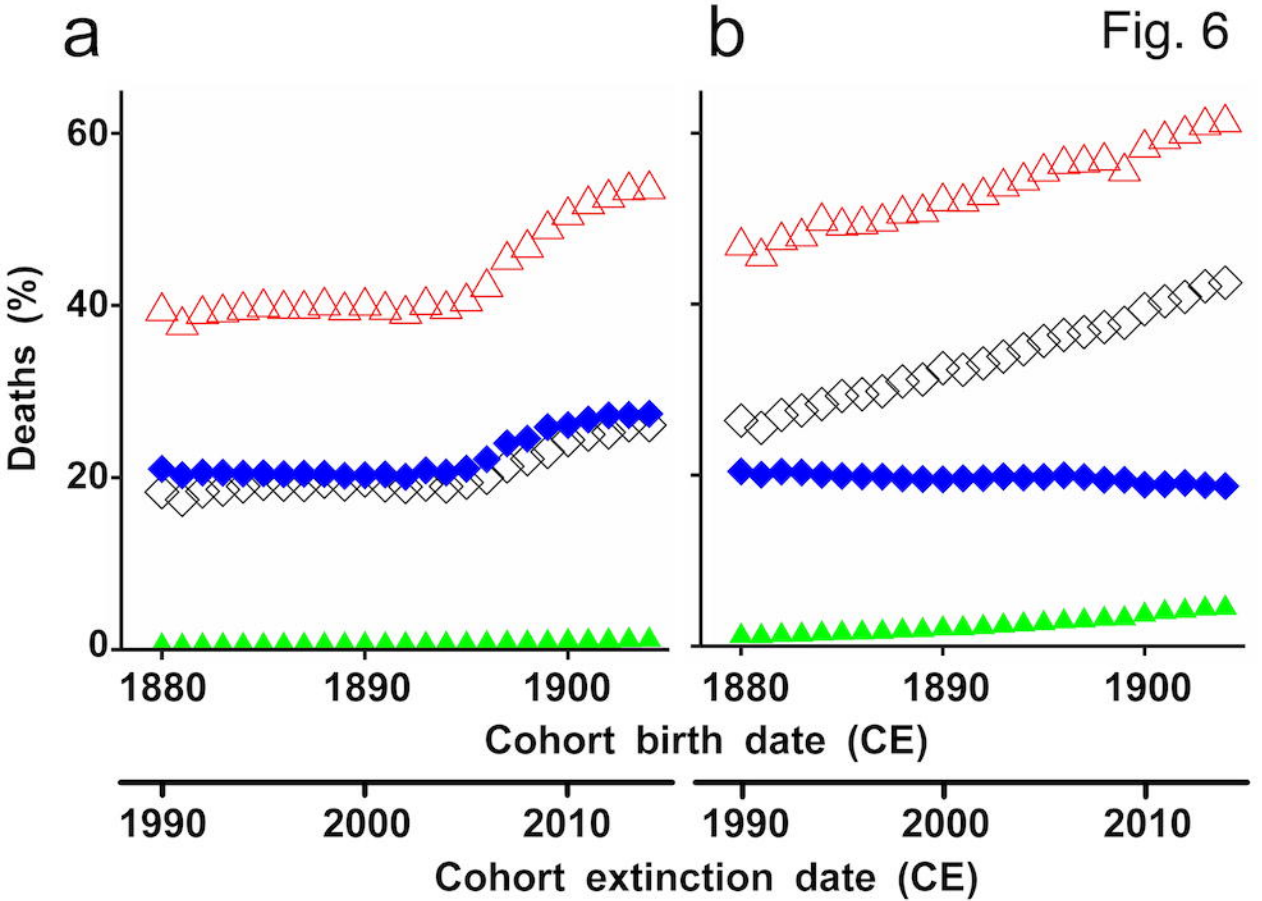

a
b
Fig. 1
Deaths (n x 1000)
30
20
10
0
50
70
90
110
Age of death (y)
50
70
90
110
Age of death (y)


a
b
Fig. 2
Modal death age (y)
90
80
70
1880
1890
1900
1910
1920
Cohort birth date (CE)
1990
2000
2010
2020
2030
Cohort extinction date (CE)


a
b
Fig. 3
Modal death age (y)
90
80
70
1880
1890
1900
1910
1920
Cohort birth date (CE)
1990
2000
2010
2020
2030
Cohort extinction date (CE)


a
b
Fig. 4
Average death age (y)
82
80
78
76
1880
1890
1900
Cohort birth date (CE)
1990
2000
2010
Cohort extinction date (CE)


a
b
Fig. 5
Average death age (y)
85
80
75
70
1880
1890
1900
Cohort birth date (CE)
1990
2000
2010
Cohort extinction date (CE)


a
b
Fig. 6
Deaths (%)
60
40
20
0
1880
1890
1900
Cohort birth date (CE)
1990
2000
2010
Cohort extinction date (CE)