Title

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Permalink

Journal

ISSN

Authors

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Publication Date

DOI

Peer reviewed

Twentieth century surge of excess adult male mortality

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Using historical data from 1,763 birth cohorts from 1800 to 1935 in 13 developed countries, we show that what is now seen as normal—a large excess of female life expectancy in adulthood—is a demographic phenomenon that emerged among people born in the late 1800s. We show that excess adult male mortality is clearly rooted in specific age groups, 50–70, and that the sex asymmetry emerged in cohorts born after 1880 when male:female mortality ratios increased by as much as 50% from a baseline of about 1.1. Heart disease is the main condition associated with increased excess male mortality for those born after 1900. We further show that smoking-attributable deaths account for about 30% of excess male mortality at ages 50–70 for cohorts born in 1900–1935. However, after accounting for smoking, substantial excess male mortality at ages 50–70 remained, particularly from cardiovascular disease. The greater male vulnerability to cardiovascular conditions emerged with the reduction in infectious mortality and changes in health-related behaviors.

excess male mortality | cohort | smoking | life expectancy | aging

Women's life expectancy now exceeds that of men worldwide (1, 2). Although the female advantage in life expectancy is relatively new in some developing countries, it has remarkably increased during the 20th century in most Western countries (3–6). Focusing on ages 40–90, we show that excess male mortality emerged in birth cohorts of the late 19th and early 20th centuries. The consistency of this change is shown for 13 countries using mortality data from 1,763 historical birth cohorts. In addition, we clarify which causes of death account for trends in sex differences in mortality to examine their concordance with potential explanations of the observed changes.

The use of cohort data differentiates our work from earlier analyses that typically relied on period data (3, 7–10). Cohort data allow us to examine the emergence of sex differentials in mortality from chronic conditions that develop slowly during adult life and to link mortality change across age within cohorts. Our results support the hypothesis that sex differences in mortality are linked to increases in male smoking, to greater adult male vulnerability to cardiovascular disease, and to changes in diet that emerged at the same time as populations reached lower mortality levels with an accompanying change in the disease structure. Besides historical lifestyle changes, we also discuss sex-linked biological factors.

Expanding sex differences in mortality emerged in national historical populations during the late 19th century and the early part of the 20th century (11, 12). Using period data for the first half of the 20th century from 50 countries, Preston (7) showed that, as the overall mortality level improved, at older ages the male mortality disadvantage increased for most causes of death. For the United States, for example, the increasing excess male mortality for the white population at ages 45–64 in 1929–1931 and in 1956–1958 was due to higher male death rates from heart disease and cancer (13).

Male excess mortality expanded during the long-term demographic/epidemiologic transition in which infectious disease mortality was replaced by chronic disease mortality among adults. The increasing male relative mortality is often described as a male epidemic (14) in association with increasing cardiovascular disease (CVD). Changes in smoking and diet and other behavioral or lifestyle factors may have affected men more than women (3, 14, 15).

The relative excess of male CVD mortality throughout the 20th century is documented for developed countries (13, 14, 16–18). Starting around 1920, period mortality data from England and the United States showed the emergence of excess male CVD mortality among people aged 35–84 (19). In England and Wales, the male:female (M:F) ratio of CVD mortality increased from about 1.5 in the 1920s to more than 3.5 among those aged 34–75 from 1950 to 1998 (14). In the United States, the M:F ratio of deaths from CVD for ages 45–64 increased by threefold from 1900 to the late 1960s (16). Moreover, CVD mortality rates from 1970 to 1986 in eight countries (Finland, New Zealand, England and Wales, Sweden, Hungary, Poland, Japan, United States) had M:F mortality ratios greater than 2 for ages 40–69 (17).

The increasing CVD rates for males have been attributed to behavioral factors, particularly diet and smoking. Lawlor et al. (14) posited the importance of increasing consumption of fat and sex differences in the resulting change in serum lipid profiles. Since 1950, a substantial portion of sex differences in adult mortality can be attributed to behavioral factors, particularly smoking (20). Smoking has had a large impact on cohort mortality trends and on sex differences in mortality and life expectancy of many countries (3, 15). Using period data for 1930 and 1963, Preston (21) showed that smoking-attributable deaths accounted for most of the excess male mortality observed at ages 40–69 in 16 developed countries. Importantly, the timing of increased smoking differs considerably by sex, with males showing an early uptake (22). Smoking spread earlier in Belgium, The Netherlands, and in the English-speaking countries, and was greater in Northern than in Southern Europe, with an inverse M:F smoking prevalence (22). For example, Portugal had high male smoking prevalence but low female prevalence, whereas Sweden and the United States showed the opposite trend with relatively low male and relatively high female smoking prevalence.

Significance

Female life expectancy now exceeds that of males in all countries. Although this gender difference has become accepted as normal, it is a relatively recent demographic phenomenon that emerged with the reduction of infections and the increase in the share of adult mortality attributed to cancer and cardiovascular disease. Heart disease is the main condition associated with increased excess male mortality, making the strongest contributions in birth cohorts of 1900–1935. Smoking behavior accounts for about 30% of male excess mortality at ages 50–70 for those born in 1900–1935. The remaining excess male mortality may be explained by underlying traits of vulnerability to cardiovascular disease that emerged with the reduction of infections and changes in diet and other lifestyle factors.
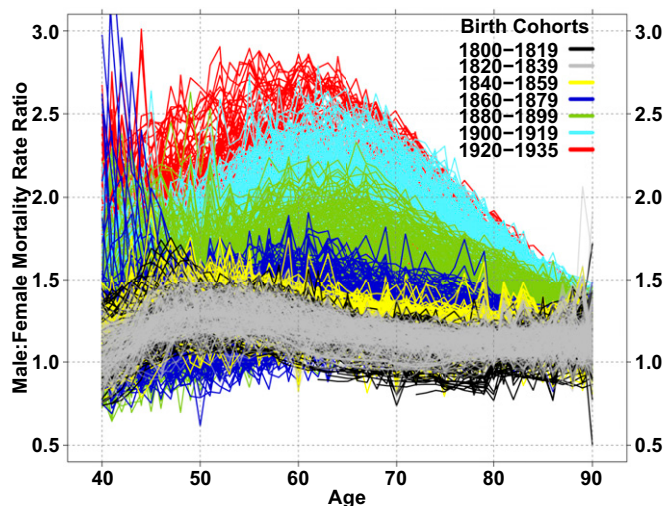
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Fig. 1. M:F mortality ratios for single-year birth cohorts for 13 countries: 1800–1935. Source: Human Mortality Database (42).

National differences in smoking patterns should result in different patterns of mortality change by sex and cohort.

Here, we examine trajectories of sex differences in mortality for ages 40–90 in 13 developed countries for birth cohorts from 1800 to 1935. Cohort data are used because they allow us to examine the emergence of sex differentials through the life cycle of cohorts as mortality increasingly results from chronic conditions that develop slowly during adult life. We study historical cohorts across the period when the smoking epidemic and heart disease became the major contributors to adult mortality and to demonstrate the consistency of sex differentials across countries. The analysis focuses on adult mortality after age 40, when chronic diseases become increasingly important in mortality change over time. Specifically, we examine the relation of mortality trends to cause-specific mortality differentials by sex, particularly for smoking-attributable deaths, heart disease, stroke, cancers, and influenza and pneumonia. Our focus is to clarify cohort trends in mortality in a period when mortality increasingly became dominated by cancer and vascular conditions.

Results

Mortality rates of both men and women decreased throughout the 19th and 20th centuries (*SI Appendix*, Fig. 1). After 1880, female mortality decreased 70% faster than male mortality as the average annual change in age-standardized cohort mortality rates at ages 40–90 from 1880 to 1917 were −0.0029 for females vs. −0.0017 for males (*P* value = 0.0000 for significant sex difference). This sex-specific difference in adult mortality improvement is the source of the increasing female life expectancy in adulthood relative to that of males. As a result, the M:F cohort life expectancy ratio at age 40 progressively declined for those born around 1880, from a M:F ratio of 0.93 for those born in 1880 to 0.85 for those born at the turn of the 20th century.

Fig. 1 shows age-specific M:F cohort mortality rate ratios (referred to hereafter as M:F mortality ratio), in which cohorts born after 1880 have increasingly higher relative male mortality in 1,612 cohorts. For instance, the black and gray lines representing cohorts born before 1840 show a M:F mortality ratio close to 1.0. The yellow and blue lines corresponding to those born in 1840–1879 show progressively higher M:F mortality ratios.

For birth cohorts of 1880–1899 (green lines), the M:F mortality ratio exceeds 1.5 at ages 50–70, whereas for those born after 1900 it exceeds twofold (light blue and red lines). After age 80, the M:F mortality ratios decline to levels of earlier adult ages to a mean

value of 1.18 [95% confidence interval (CI), [1.17,1.19]] across all cohorts by age 90. Thus, the major increase in the sex mortality difference occurred in late middle age and early old age. For instance, the mean M:F mortality ratio at age 50 continually increased for those born after 1880, from 1.39 (95% CI, [1.36,1.42]) in 1880–1899 to 1.68 (95% CI, [1.65,1.70]) in 1900–1919 and 1.90 (95% CI, [1.86,1.93]) in 1920–1935. A similar pattern occurred for ages 60 and 70, although the mean M:F mortality ratios are higher, greater than 2 for births after 1900.

This pattern is fairly similar across all countries (Fig. 2), although some countries showed earlier emergence of excess male mortality (*SI Appendix*, Fig. 2). Country-specific trends show average M:F mortality ratios at ages 50–70 exceeding 1.5 starting with cohorts born in 1880–1899. Men in Australia, France, and Switzerland experienced early onset of excess mortality at age 50 that reached 45–49% for birth cohorts of 1860–1879. Later onset countries, Denmark, Netherlands, Norway, and Sweden, lagged in this trend, with males consistently reaching 50% excess mortality among those born in the 1900s. The trend toward an increasing male excess mortality may have ended in several countries among the more recent cohorts born from 1920 to 1935 (Fig. 2, red lines), e.g., England and Wales, Norway, and the United States. For other countries, the increase in the M:F mortality ratio has persisted, e.g., France, Italy, and Spain.

Fig. 3 summarizes M:F mortality ratios for all countries from Fig. 1 as the unweighted mean and its 95% CI by age and cohort. This figure confirms a continuous increase across birth cohorts in the M:F mortality ratio between ages 50 and 70 starting around 1880. It also shows that excess male mortality diminishes after age 90. We assess the impact of smoking-attributable deaths and cause-specific mortality on this trend in M:F mortality ratios using two approaches. First, we quantify excess male mortality due to smoking and specific causes of death on each birth cohort by eliminating specific causes of mortality (cause elimination). Second, we estimate how much of the change in M:F mortality ratios between cohorts is due to changes in cause-specific mortality between males and females (cause decomposition).
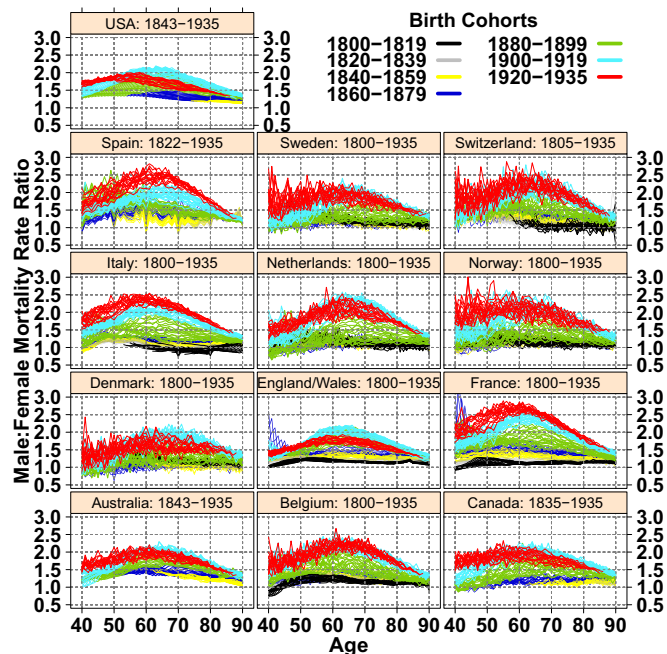

Fig. 2. M:F cohort mortality rate ratios for single-year birth cohorts including "incomplete" cohorts by country: 1800–1935. Note: Incomplete cohorts correspond to those that did not have the complete history of mortality rates for every age between 40 and 90. Source: Human Mortality Database (42).
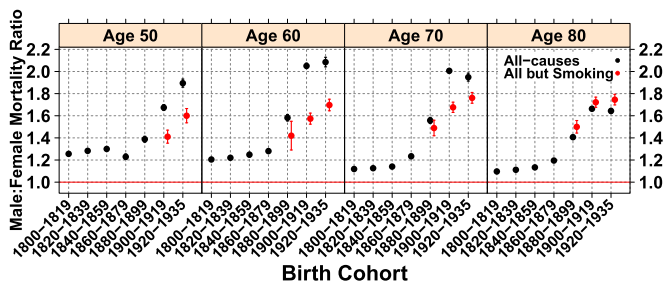
Fig. 3. Unweighted mean M:F cohort mortality rate ratios and their 95% CI by age and birth cohort for 13 countries, 1800–1935. Note: Values in black color correspond to all-cause mortality ratios from Fig. 1. Values in red color are estimated after excluding smoking-attributable mortality from all-cause mortality rates by age, cohort, and sex. There are no cause-specific mortality ratios at age 90 because data on deaths from WHO are top-coded at age 85+.

Effect of Smoking. The effect of eliminating smoking-attributable deaths on the M:F mortality ratios by age and cohort are shown in Fig. 3. The M:F mortality ratio consistently declines at ages 50–80 across cohorts when excluding smoking-attributable deaths, with larger declines in cohorts born in the 1900s. The contribution of smoking-attributable deaths to excess male mortality by age and cohort (Table 1) shows the larger contribution of post-1900 birth cohorts. Smoking-attributable deaths account for about 30% of excess male mortality at ages 50–70 for those born in 1920–1935. When smoking-attributable deaths are excluded, excess mortality at ages 50, 60, and 70 for 1900–1919 is about one-third less than when smoking deaths were included (Table 1). This pattern is also observed for those born between 1880 and 1899 but with smaller smoking contribution (about 20%). However, even after excluding smoking-attributable deaths between ages 50 and 70, the mean M:F mortality ratio remains above 1.5 among the most recently born cohorts (Fig. 3).

Fig. 3 indicates that the largest increase in all-cause M:F mortality ratios occurred between cohorts born in 1880–1899 and 1900–1919. To better understand the effect of smoking on this trend, we quantify how much of the increase in all-cause M:F mortality ratio is due to smoking-attributable deaths (*SI Appendix,* Table 1). Between cohorts 1880–1899 and 1900–1919, smoking accounted for about one-fourth of the increase in all-cause M:F mortality ratios at ages <60, and over 35% at ages 65+. This is due to large increases in male smoking-attributable deaths among those born at the turn of the 20th century. On the contrary, the slight reduction in all-cause M:F mortality ratios between cohorts 1900–1919 and 1920–1935 is due to increases in female smoking-attributable mortality, particularly at ages 50–70.

Further decompositions by country show similar results with a distinct pattern based on smoking uptake. Larger contributions of smoking between cohorts 1880–1899 and 1900–1919 in countries with earlier smoking uptake (e.g., Belgium and The Netherlands) and stronger smoking contributions between 1900–1919 and 1920–1935 in countries lagging behind in smoking uptake, e.g., France and Spain (*SI Appendix,* Table 2). Between cohorts 1880–1899 and 1900–1919, smoking accounted for over 40% of the change in all-cause M:F ratios at ages 60+ in Australia, Belgium, and The Netherlands. Smoking reached similar contributions in Canada, England and Wales, Italy, and the United States at ages 70–80. Between cohorts 1900–1919 and 1920–1935, male smoking-attributable mortality increased in countries lagging behind the smoking uptake (France, Italy, and Spain) accounting for over 30% of the upward trend in all-cause M:F ratios at ages 50–60. Additionally, between these same cohorts, females in Australia, Canada, Denmark, England and Wales, The Netherlands, Norway, and United States experienced

an increase in smoking-attributable mortality that contributed to reductions in all-cause M:F mortality ratios.

Effect of Specific Causes of Death. Estimated smoking-attributable deaths are a combination of deaths from many causes including heart disease, stroke, and cancer and cannot be separated into the contribution of individual causes. The effect of eliminating all mortality from heart disease, cancer, stroke, and influenza/pneumonia on M:F mortality ratios by age and cohort are shown in Fig. 4 (rows 1, 2, 3, and 4, respectively). The M:F mortality ratio for each of the individual causes increases over time. Importantly, heart disease is the main condition associated with increased excess male mortality with the strongest increases in birth cohorts of the 20th century (Fig. 4, row 1). The M:F mortality ratio for heart disease increases across cohorts up through age 80 and shows excess male mortality among recent birth cohorts (born after 1900). This ratio is far higher than the ratio of all causes at ages 50 and 60, with males having about threefold more heart disease mortality than females. If heart disease is excluded, the M:F mortality ratio would be somewhat lower than that observed for all causes. The changes in the M:F mortality ratios for stroke (row 3) and for influenza/pneumonia (row 4) approximate changes in the M:F mortality ratio for all causes. On the other hand, differences in the M:F mortality ratio for cancer mortality (row 2) are relatively small at ages 50 and 60, whereas the older ages have a M:F cancer mortality ratio that is closer to all-cause mortality.

Fig. 5 further resolves cause of death contributions responsible for increases in all-cause M:F mortality ratios across cohorts. Between the cohorts of 1880–1899 and 1900–1919, heart disease and stroke accounted for over 40% of the increase in all-cause M:F mortality ratio at ages 55–80 (*SI Appendix,* Table 3). This is due to females experiencing a consistently faster mortality decline than males across all adult ages (55–80). Although male mortality from stroke and influenza/pneumonia declined between these cohorts, cancer consistently increased and heart disease declined only at older ages (70–80). Between cohorts 1900–1919 and 1920–1935, the M:F ratio increased at ages 50–60 with a slight decline at older ages. This is the result of offsetting trends in sex-cause–specific mortality. Heart disease and stroke contributed to raising the M:F ratio at ages 50–60 due to faster mortality declines among females, whereas changes in cancer mortality contributed to reducing it because males benefited more from the mortality decline. The

Table 1. Contribution of smoking-attributable deaths to excess male mortality by age and cohort

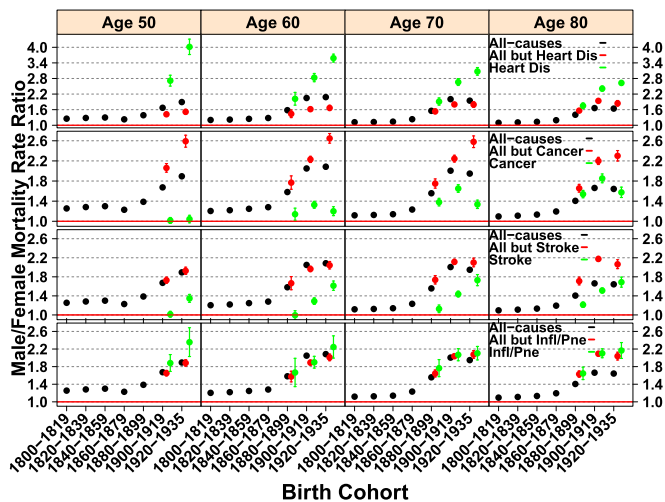| Age	Mean M:F ratios		Excess male mortality Due to smoking, %
	All causes	All but smoking	
Cohort 1880–1899			
55	1.52	1.42	19.79
60	1.58	1.49	15.91
65	1.60	1.50	17.16
70	1.56	1.41	25.85
Cohort 1900–1919			
50	1.67	1.41	39.06
55	1.93	1.57	38.38
60	2.05	1.68	35.62
65	2.10	1.72	34.37
70	2.01	1.67	33.86
Cohort 1920–1935			
50	1.90	1.60	32.90
55	2.04	1.70	32.79
60	2.09	1.76	29.79
65	2.05	1.75	29.20
70	1.95	1.69	27.77

Fig. 4. Unweighted mean M:F cohort mortality rate ratios and their 95% CI for all-cause and cause-specific mortality by age and birth cohort for 13 countries, 1800–1935. Inf/Pne, influenza/pneumonia. Black values come from Fig. 3. Mortality ratios in red are estimated after excluding cause-specific mortality from all-cause mortality rates by age, cohort, and sex. There are no cause-specific mortality ratios at age 90 because data on deaths from WHO are top-coded at age 85+.

reduction in the M:F ratio at ages 70–80 is associated with larger mortality declines among males from causes other than heart, cancer, stroke, and influenza/pneumonia.

This pattern of cause-specific contributions to changes in all-cause M:F mortality ratio is fairly similar across countries (*SI Appendix*, Figs. 3 and 4). Between cohorts 1880–1899 and 1900–1919, heart disease contributed the most to increasing all-cause M:F mortality ratios in all countries except Australia and France. In Australia, stroke had the largest contribution, whereas cancer did so for France at ages <70. Between cohorts 1900–1919 and 1920–1935, heart disease and stroke remained the leading contributors in France, Italy, and Spain. In countries lagging behind in excess male mortality (Denmark, The Netherlands, Norway, and Sweden), increases in all-cause M:F mortality ratios at ages <60 are also due to heart disease and stroke, whereas reductions in the ratio at older ages resulted from declines in cancer mortality. A large fraction of these deaths are likely due to smoking because it accounted for a large fraction of the increases in all-cause M:F mortality ratios.

Discussion

This analysis shows that excess adult male mortality is clearly rooted in specific age groups, 50–70, and that further sex asymmetry emerged in cohorts born after 1880 when M:F mortality ratios increased by as much as 50% from baselines of about 1.1. Birth cohorts born after 1900 in 13 countries experienced twofold higher male mortality than females at ages 50–70. However, by age 90, the excess male mortality among the cohorts returned toward the historical baseline of about 1.1. These findings are consistent with prior analysis of period data, which also showed excess adult male mortality throughout the 20th century (3, 7, 10, 13, 15, 19, 21).

These findings are previously unidentified in showing that excess male cohort mortality at ages 40–90 arose because of slower improvement of male mortality than female. Among those born after 1880, women had 70% faster decline than men in age-standardized mortality rates between ages 40 and 90. Underlying this change is a dramatic age patterning of excess male mortality in which the M:F mortality rate ratio exceeded 2 at ages 50 through 70 for those born after 1900.

Explanations for Smoking. About 30% of the excess male mortality at ages 50–70 after 1880 is attributable to smoking. As expected, national sex differences in smoking patterns are linked with different patterns of mortality change. For instance, smoking accounted for over 40% of the change in M:F ratios between cohorts 1880–1899 and 1900–1919 at ages 60+ in Australia, Belgium, and The Netherlands, which is consistent with early smoking uptake among males in these countries (22). On the other hand, the trend toward an increasing male excess mortality has ended among those born in 1900–1935 in countries that experienced an early male smoking uptake, e.g., England and Wales and the United States. However, there still remains a large fraction of unexplained excess male mortality among recently born cohorts, with M:F mortality ratios greater than 1.5 when smoking-attributable deaths are excluded. In addition to sex differences in smoking, several other behaviors can contribute to the M:F mortality difference. We know that diets and energy expenditure changed across these cohorts, possibly more for males than for females.

Explanations for Heart Disease. Additional analyses using data on food supply from animal fats from Food and Agriculture Organization Corporate Statistical Database indicate associations of trends in per-capita animal fat intake and heart disease M:F mortality ratios in these countries (*SI Appendix*, Figs. 5 and 6). Countries with early onset of excess male mortality (e.g., France and Italy; *SI Appendix*, Fig. 5) shared a similar pattern of increase in fat intake across all cohorts and ages, whereas in countries with a late onset of excess male mortality (e.g., Denmark, Norway, and Sweden; *SI Appendix*, Fig. 6) this pattern was only observed for birth cohorts after 1900. Also notable is the tight cohort-age pattern between animal fat intake and heart disease mortality in France and Italy, the two countries that continue to show an upward trajectory in all-cause M:F ratios for ages 50–80 for those born in 1900–1935 (*SI Appendix*, Fig. 2). Evidence from England and Wales suggests that the initial increase in the sex ratio of CVD in part resulted from increasing consumption of fat and sex differences in the resulting change in lipid profiles (14). A metaanalysis including 171 studies in the United States showed higher total fat consumption by males aged 18–65 from 1950 to 1985, including more energy derived from saturated fatty acids (23). Denmark experienced a progressive increase fat intake after 1900 with increasing saturated fats after 1920 (24), whereas in Norway fat intake increased from about 1880 to 1950, and then declined (25). We suggest that men not only had more changes in diet and energy expenditures than women but that men also had underlying greater biological vulnerability to these changes.

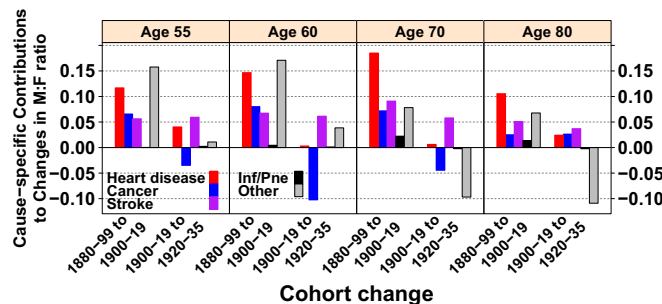**Fig. 5.** Contributions of cause-specific mortality rates to changes in the M:F mortality rate ratio by age and birth cohort for 13 countries: 1880–1899 to 1900–1919 and 1900–1919 to 1920–1935. Note: Inf/Pne, influenza/pneumonia. Positive values indicate that mortality changes in a cause of death between men and women contributed to raising the ratio, whereas negative values contributed to reducing it.

Our results also indicate that heart disease is the main condition associated with increased excess male mortality. Heart disease and stroke accounted for over 40% of the increase in M:F mortality ratios between cohorts 1880–1899 and 1900–1919. Males may have an intrinsically greater vulnerability to CVD that emerged with the reduction of infections; for instance, because the distribution of fat among men and women differs and their differing patterns of adiposity could make men more vulnerable to the increasing weight that resulted from changes in diet and activity (26). Changing blood lipids could be another factor (14). High levels of total cholesterol and low levels of high density lipoprotein cholesterol (HDL) are risk factors for CVD with the ratio of the two the most predictive of CVD (27). HDL is generally lower among men (28, 29). Differences in HDL are one mechanism through which blood estrogens provide premenopausal women with relative protection from CVD (30–32). Because HDL is lower among those with past and current exposure to infections (33, 34), the increasing male vulnerability might also arise from greater increases in total cholesterol relative to HDL with increased fat consumption and weight gain.

Current observations of M:F differences indicates that biologically seated sex differences in arterial function could also be involved in changing M:F mortality ratios of heart disease. The presence of estrogens in females up until menopause is thought to be cardioprotective (31, 32). In contemporary populations, men show greater risk of atherosclerotic plaque rupture by 1.9-fold (35). Relevant to blood pressure regulation, arterial endothelial responses deteriorate 15 y earlier in men than women (36). Although these endothelial responses coincide with the peak age of excess male mortality, their timing diverges from the sharp postmenopausal decline of estrogens. Moreover, animal models further show greater intrinsic male cell vulnerability in experimental conditions. Vascular endothelial cells from male rats are less resistant to vascular barrier challenge (37, 38), whereas male-derived cardiomyocytes are more vulnerable to ischemia (39). Thus, greater male vulnerability in arterial function could produce excess male mortality from heart disease as this condition became more prevalent with the reduction of infections during the demographic transition. Infection-driven inflammation was gradually replaced by sterile inflammatory processes associated with smoking, and with lifestyle changes in diet and physical activity that promoted vascular risk factors.

Could differing gene–environment interactions by sex contribute to the excess CVD male mortality? Some Y-chromosome haplotypes with CVD associations (40) also alter the expression of immune-related genes in macrophages (41). Thus, there could be trade-offs during declining mortality from infections between Y-chromosome genetic variants that conferred resistance to infections but contributed to the increasing male excess of heart disease.

Summary and Conclusions

These findings show that the current excess of female life expectancy in adulthood is a relatively new demographic phenomenon that emerged among people born in the late 19th century. As mortality improved among these cohorts, cardiovascular disease became a more important cause of death concurrently with changes in behaviors that affect the development of chronic disease. We suggest greater adult male vulnerability to cardiovascular disease may also involve sex-specific linked biological factors that emerged during the reduction of mortality from infections.

Materials and Methods

Data. Single-year cohort mortality rates from the Human Mortality Database (HMD) (42) are used to examine mortality from age 40 through 90 when mortality from chronic conditions is concentrated. Ages below 40 were excluded to minimize effects of wars, accidents, and violence; the upper age was chosen at 90, after which data from historical cohorts are less reliable. Mortality was examined for all countries with available cohort mortality

before 1900 and where the populations are large enough for stable annual mortality rates by single years of age (e.g., we eliminated Finland and Iceland because of excessive fluctuations in mortality rates). Fig. 2 shows the countries and birth cohorts considered. For most European countries, the data begin with the cohort born in 1800 (Sweden, France, Denmark, Norway, The Netherlands, Italy, England and Wales, Belgium); Switzerland (1805) and Spain (1822) have data beginning somewhat later. "New World" data are not available until later: Canada (1835), Australia (1843), and the United States (1843). Thus, we have data on both "complete cohorts," i.e., cohorts who had a complete history of mortality rates for every age between 40 and 90, and "incomplete cohorts," i.e., cohorts lacking a complete history of mortality rates (*SI Appendix*, Fig. 7A). For example, for the United States, a complete mortality history from age 40 to age 90 is only available for those born in 1883 or later, rendering incomplete data for prior cohorts of 1843–1883. Cause-specific death counts from the World Health Organization (WHO) Mortality Database were used to create death rates by cause and birth cohort (43). Death rates by cause are only available from 1950 onward for people aged 50–84, which limits the number of birth cohorts (*SI Appendix*, Fig. 7B). Total number of deaths and exposure to risk from 1950 to 2005 for age groups 50–54 to 80–84 for each country was obtained from the HMD. Because HMD does not include data by cause of death, we used cause-specific mortality from the WHO by aggregating cause-specific deaths for age groups 50–54 to 80–84 for every 5 consecutive years between 1950 and 2005. We focus on five causes of death: smoking-attributable deaths (estimated from lung cancer and all other causes), CVD, stroke, cancers, and influenza and pneumonia (International Classification of Diseases codes; *SI Appendix*, Table 4).

Age-Standardized Cohort Mortality Rates. Age-standardized mortality rates were estimated by cohort–country–sex for ages 40–90 using the World population from the World Health Organization as the standard. We computed unweighted means of age-standardized mortality rates across countries by sex and cohort.

Male/Female Cohort Mortality Ratios. Let $M_{x,j}^k$ represent cohort mortality rates at age x, cohort j, sex k {k = male,female}. Male/female cohort mortality rate ratios are estimated as $MFR_{x,j} = M_{x,j}^m / M_{x,j}^f$.

Cause-Specific Mortality. Smoking-attributable deaths were estimated using an approach developed by Preston et al. (15). Briefly, we used the coefficients derived in Preston et al. (15), combined with lung cancer mortality rates from WHO to estimate smoking-attributable deaths by country–year–sex and age group. The coefficients are applied to lung cancer mortality rates from WHO and total number of all-cause deaths from HMD. For the other causes of death (cancer, heart disease, stroke, and influenza/pneumonia), we apply the distribution of deaths by cause from WHO to the death counts and rates from HMD to calculate cause-specific death counts and rates for each country–year–sex–age group. Cohort-cause–specific mortality rates were constructed as the diagonals as shown in *SI Appendix*, Fig. 7B. Cause-specific M:F mortality ratios were estimated by age and cohort. The computation of smoking-attributable M:F mortality ratios is restricted to cases in which females have at least 10 deaths per 1,000 people. This process reduces instability in the ratios. For example, female smoking-attributable deaths are zero for most countries included in the analysis for those born in the late 1800s and early 1900s (15), which leads to undefined ratios. Additionally, we estimate changes in age-specific all-cause M:F mortality ratios due to cause-specific mortality by subtracting cause-specific deaths from all-cause mortality by age, sex, and cohort. There are thus five cause-specific M:F mortality rate ratios: all but smoking, all but heart disease, all but stroke, all but cancer, and all but influenza/pneumonia.

Decomposition of Changes in All-Cause M:F Mortality Ratio Due to Changes in Cause-Specific Mortality Rates. Let $M_{x,j,i}^k$ correspond to mortality rates at age x, cohort j, cause of death i, and sex k {k = male,female}. For n mutually exclusive and exhaustive causes of death, $M_{x,j}^k = \sum_{i=1}^n M_{x,j,i}^k$. We estimated changes in $MFR_{x,j}$ over cohorts by taking derivatives of the all-cause mortality ratio (13):

$$\frac{\partial MFR_{x,j}}{\partial j} = \sum_{i=1}^n MFR_{x,j} \left[\frac{\partial M_{x,j,i}^m / \partial j}{M_{x,j}^m} - \frac{\partial M_{x,j,i}^f / \partial j}{M_{x,j}^f} \right],$$

$$\Delta MFR_x = \sum_{i=1}^n \overline{MFR}_x \left[\frac{\Delta M_{x,i}^m}{\overline{M}_x^m} - \frac{\Delta M_{x,i}^f}{\overline{M}_x^f} \right], \tag{1}$$

where ΔMFR_x is absolute change in MFR_x between cohorts j_1 and j_2; $\overline{MFR}_x$ is average MFR_x between cohorts j_1 and j_2; $\Delta M_{x,i}^k$ is absolute change in death rates at age x from cause i sex k between cohorts j_1 and j_2; and $\overline{M}_x^k$ is average

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mortality rate for age x, sex k between cohorts j_1 and j_2. Eq. **1** shows that changes in all-cause M:F mortality ratio equal the weighted sum of the ratio with weights corresponding to the difference in relative changes between males and females in cause-specific mortality rates.

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