



Women live longer than men even during severe famines and epidemics

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Women in almost all modern populations live longer than men. Research to date provides evidence for both biological and social factors influencing this gender gap. Conditions when both men and women experience extremely high levels of mortality risk are unexplored sources of information. We investigate the survival of both sexes in seven populations under extreme conditions from famines, epidemics, and slavery. Women survived better than men: In all populations, they had lower mortality across almost all ages, and, with the exception of one slave population, they lived longer on average than men. Gender differences in infant mortality contributed the most to the gender gap in life expectancy, indicating that newborn girls were able to survive extreme mortality hazards better than newborn boys. Our results confirm the ubiquity of a female survival advantage even when mortality is extraordinarily high. The hypothesis that the survival advantage of women has fundamental biological underpinnings is supported by the fact that under very harsh conditions females survive better than males even at infant ages when behavioral and social differences may be minimal or favor males. Our findings also indicate that the female advantage differs across environments and is modulated by social factors.

famines | epidemics | mortality | survival | gender

Women are the life-expectancy champions: They can expect to live longer than men almost anywhere in the world today (1–3). This pervasive inequality has intrigued researchers for decades (4). The cumulative corpus of research supports the conclusion that the gap has biological underpinnings modulated by social and environmental conditions. Deeper understanding could benefit from biodemographic research (5). Here we present some results of such research.

Support for a biological root of the gender gap in survival stems from studies of groups in which men and women have more similar lifestyles than in the general population, such as among nonsmokers (6, 7) or within religious groups such as active Mormons (8) or cloistered monks and nuns (9). Findings indicate that, even though men and women in these groups have more similar lifestyles and men are exposed to fewer risk factors than men in the general population, a gender gap in life expectancy still persists. Excess male mortality is also found among newborns and infants (10–12), when behavioral differences are unlikely to play a crucial role and social factors may be neutral or favor male survival. An untapped source of information is the reverse situation, when both men and women experience high, perhaps extreme, levels of mortality risk. A finding that men and women have similar life expectancies under these conditions would challenge the notion that the survival advantage of women is fundamentally biologically determined in all environments. Therefore, we study here the survival of both sexes in populations enduring mortality crises.

While women have lower mortality than men in modern populations, evidence for a female survival advantage under crisis

conditions is sparse. A well-known story concerns the Donner Party, a group of settlers that lost twice as many men as women when stranded for 6 mo in the extreme winter in the Sierra Nevada mountains (13). While accounts like this are anecdotal, a variety of studies provide evidence that women appear to survive cardiovascular diseases, cancers, and disabilities longer than men (14–18). However, the generality of this notion needs to be treated with caution, since findings on sex differences in survival after myocardial infarction and stroke are mixed (19–21).

Additional support for female hardiness comes from the fact that, in most countries, the sex difference in remaining healthy life expectancy is smaller than the difference in total life expectancy. The difference becomes even smaller later in life: For example, the gender gap in life expectancy at age 65 y for France and Sweden in 2015 was 4.1 and 2.6 y, respectively, while the gap in healthy life expectancy at age 65 y was only 0.9 and 1.1 y (ec.europa.eu/eurostat/web/population-demography-migration-projections/deaths-life-expectancy-data/main-tables; ec.europa.eu/eurostat/statistics-explained/images/3/3d/Healthy_life_years%2C_2015_%28years%29_YB17.png). Thus women live more years than men and are able to do so even though they are in bad health for a substantial part of those extra years of life.

We investigate whether the ability of women to survive better under difficult circumstances extends to crises such as famines, epidemics, or slavery.

Significance

Women live longer than men in nearly all populations today. Some research focuses on the biological origins of the female advantage; other research stresses the significance of social factors. We studied male–female survival differences in populations of slaves and populations exposed to severe famines and epidemics. We find that even when mortality was very high, women lived longer on average than men. Most of the female advantage was due to differences in mortality among infants: baby girls were able to survive harsh conditions better than baby boys. These results support the view that the female survival advantage is modulated by a complex interaction of biological environmental and social factors.

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Data and Methods

High-Mortality Populations. We analyzed seven documented populations with extremely low life expectancies (20 y or less) for at least one of the sexes, due to extreme conditions such as famines, epidemics, or slavery. Even though a life expectancy of 20 y might seem unrealistically low, some populations in temporarily extreme conditions had a life expectancy below this value (22). Historical demographic data can be often problematic. However, the cases used in this study have all been previously published in a respected peer-reviewed journal. This, by itself, should ensure data quality and reliability. Nevertheless, for each case we discuss potential problems and biases that can affect the gender difference in survival.

Freed Liberian slaves (23, 24). Between 1820 and 1843, freed American slaves were encouraged to migrate back to Africa. Many undertook the risky trip and went to Liberia, where they encountered a very different disease environment than the one in which they grew up. McDaniel (24) used data collected by the American Colonization Society from 1820 to 1843 and estimated life tables for the former slaves. The data show the highest mortality ever registered in recorded human history. The arrival in Liberia was a mortality shock. About 43% died during the first year, and life expectancy at birth was 1.68 y for men and 2.23 y for women. Data come from a dataset referred to as “the roll,” which lists those who emigrated from the United States under the monitoring of the American Colonization Society and the US government between 1820 and 1843. McDaniel and Preston (23) and McDaniel (24) performed multiple data checks, matching them with other administrative sources, and concluded that the emigrant population was monitored carefully. The data were then compared with the model life tables and show the same age pattern as the so-called “North model” but at more extreme levels. That is, the data show a pattern of mortality over age that corresponds with that of other human populations. However, even though the determination of gender of the individuals (which was not available in the original data) was carefully done with special software and a name dictionary, following a rigorous procedure, the sex of 181 of the 4,472 cases (4%) remained unidentified. This might have caused some bias in the sex-specific death rates, but the percentage is small enough to conclude that any bias would be weak.

Plantations slaves in Trinidad (25, 26). At the beginning of the 19th century pro- and antislavery forces clashed about the emancipation of the slaves in the British Caribbean. The antislavery campaign obtained an annual registration of the slaves in the colony of Trinidad, the only colony controlled directly from London. Since unregistered slaves were confiscated by the Crown, owners had a strong incentive to comply with the order. The register contains the age of slaves in 1813 and in 1816 and records how many deaths and births occurred during the period. John (25) analyzed the data in the register. She concluded the data were complete for all age groups except for infants (age category 0–1 y), who were underreported, and that the data were affected by age heaping on multiples of five. Unfortunately, this could not be smoothed because the age and sex composition of the population was shaped by the slave trade, but she limited its interference by estimating life tables by 5-y age groups. She then produced period life tables for the male and female slaves in the plantations of Trinidad, which showed that life expectancy could have been as low as 15.18 y and 13.21 y (lower bound) or 19.45 y and 20.58 y (upper bound) for males and females, respectively. Most of the variation derived from uncertainty about the level of infant mortality, and this uncertainty could be a source of bias in the computation of the gender survival gap. However, the author notes that most of this uncertainty can be ruled out by conditioning the survival curves to having survived up to age 1 y and that the conditional survival functions for the upper and lower bounds of the life table are virtually identical (25). The conditional survival curves, obtained by dividing each value of the survival curves by the survival value at age 1 y (as reported in ref. 26), are very similar in their upper and lower bounds. Most importantly, they show the same age and gender pattern as the unconditional survival curves.

The Ukrainian famine in 1933 (27). In the twentieth century, the Ukraine experienced particularly turbulent demographic trends that mirror a history of major crises. Among these, the great famine in 1933 that followed the collectivization of agriculture is documented by Meslé and Vallin (27), who painstakingly reconstructed several data series. They estimated that period life expectancy during the crisis dropped to 7.3 y for men and 10.9 y for women from 41.58 and 45.93 y for men and women, respectively (average of the 5 y before). The authors used census data and vital registrations available before and after the crisis (in 1926 and 1939), between which they applied a series of methods such as interpolation, forward and backward projections, assumptions for fertility and migration during the period, and correction coefficients for the underreported deaths during the crisis. The basic idea was to compare the actual 1939 population reported in the census

with a hypothetical population that would have existed without the crisis, to obtain the effect of the crisis corrected for fertility loss and migration flows produced by the famine. These could be estimated only by applying some assumptions. If the assumption of constant total fertility rate at the level of 1931 is not likely to affect the estimates by gender, the assumption related to migration could introduce a bias in the estimated gender patterns of survival, e.g., if, a sex was attributed with a different migration rate than the actual one. However, the authors based their work upon a solid base of historical and statistical references that represents the most reliable source of available knowledge about that period.

The Swedish famine in 1772–1773 (28). This is described as the last major famine that caused starvation across most of Sweden. Abnormal weather conditions in the summer of 1771, followed by widespread crop failures, caused a sudden and sharp increase in food prices. Consequently, mortality due to starvation increased. When the difficult crop conditions continued throughout 1772, mortality increased even further in 1773. Approximately 50% of the excess mortality was due to dysentery, a disease related to the malnutrition (28). Since the famine affected most of the Swedish population, we used male and female life tables from the Human Mortality Database (www.mortality.org) for Sweden in 1773, when life expectancy plummeted to 17.15 y for males and 18.79 y for females. The Human Mortality Database is the best source of historical and current death rates for national populations. The very high quality of its data is ensured by the database being limited to populations for which death registration and census data are virtually complete, since this type of information is necessary for the uniform method used to reconstruct the data series. Among the countries included in the dataset, Sweden has the longest time series. The vital registration system in this country was established in the 17th century and was serviceably accurate by the mid-18th century.

The Icelandic epidemics in 1846 and 1882 (29). Because the population of Iceland was small, measles was not endemic and was devastating when epidemics struck. In 1846 and 1882 Iceland experienced its two major measles epidemics of the 19th century. The disease spread rapidly through most of southern Iceland, the most populated area of the country. Even though official registration of deaths by measles started only in 1904, the two epidemics were documented in parish registries and reports from physicians. Both epidemics spread from Danish boats landing in the late spring of the respective year. Severe weather and unsanitary wet conditions facilitated the spread of the disease by causing many complications such as diarrhea and chronic bronchitis (29). We used life tables from the Human Mortality Database that show a sudden drop in life expectancy of both sexes in 1846 (from 35.35 y to 17.86 y for males and from 40.81 y to 18.82 y for females) and 1882 (from 37.62 y to 16.76 y for males and from 43.99 y to 18.83 y for females).

The Irish famine in 1845–1849 (30). By 1845 potatoes were the staple food for the majority of the Irish. When the mold *Phytophthora infestans* infected the plants and caused nearly total crop failures over three consecutive years, the Irish population starved. The population shrank due to extremely high mortality, emigration, and fewer births. Life tables for the famine years were constructed by combining various data sources (30). Life expectancy dropped from about 38 y for both sexes in the prefamine years to 18.7 y for men and 22.4 y for women. The history of the Irish population was shaped by extensive migration during both nonfamine and famine years. The two major destinations were North America and Britain. Controlling for migration, therefore, became a crucial aspect of the reconstruction of the toll of the famine, to reduce the impact of a potentially severe bias. Fortunately, the authors had available several analyses of the migration flows, mostly based on ship passenger lists and on British censuses (which recorded the Irish-born population resident in Britain), which allowed them to estimate quite precisely the age and sex profile of the migrants (for example, the male:female ratio was about 60:40 for emigrants to North America and 55:45 for emigrants to Britain).

Mortality Comparison. We compared mortality between the sexes and among the populations using different mortality measures, including the probability of survival from birth to age x (henceforth “survival”), the probability of dying between age x and $x + 1$ (henceforth “mortality”), life expectancy (at birth), and the age at which 5% of a synthetic same-sex cohort would still be alive (henceforth “extreme age”). We took these measures from the life tables, when available, and otherwise applied standard demographic methods to compute the life tables (31). For comparing mortality between the sexes, we further computed male:female mortality ratios and differences for each population and decomposed the sex differences in life expectancy by age (32).

Table 1. Absolute and relative differences in male and female life expectancy for seven high-mortality populations during (and, when available, before and after) extreme mortality conditions

Population	Life expectancy						Female–male difference in life expectancy					
	Male			Female			Absolute, y			Relative		
	Pre	Crisis	Post	Pre	Crisis	Post	Pre	Crisis	Post	Pre	Crisis	Post
Liberia 1820–1843	—	1.68	22.87*	—	2.23	24.62*	—	0.55	1.25*	—	0.33	0.05*
Trinidad 1813–1816	—	15.18–19.45 [†]	—	—	13.21–20.58 [†]	—	—	–1.27–1.13 [†]	—	—	–0.08–0.06 [†]	—
Ukraine 1933	41.58	7.30	45.12	45.93	10.85	50.49	4.35	3.55	5.37	0.1	0.49	0.12
Sweden 1773	32.31	17.15	37.61	35.19	18.79	39.85	2.88	1.64	2.24	0.09	0.09	0.06
Iceland 1846	35.35	17.86	33.13	40.81	18.82	38.31	5.46	0.96	5.18	0.15	0.05	0.16
Iceland 1882	37.62	16.76	37.82	43.99	18.83	43.74	6.37	2.07	5.92	0.17	0.12	0.16
Ireland 1845–1849	38.3	18.7	—	38.3	22.4	—	0	3.70	—	0	0.20	—

*Life expectancy at age 1 y.

[†]Values refer to lower and upper bound.

Results

Overview of Gender Difference in Mortality in Normal Conditions.

Data for pre- and postcrisis times were available only for the Ukraine in 1933, Sweden in 1773, Iceland in 1846, and Iceland in 1882. In these cases, during normal years, women had higher life expectancy than men (Table 1). The female–male difference for each population was remarkably stable between the precrisis and the postcrisis years (Table 1), a sign that the ep-

idemics or the famines affected the gender difference in survival only temporarily.

For the other populations analyzed in this study only partial information about mortality in normal conditions was available. For the freed Liberian slaves, a life table for those who survived the critical first year of arrival shows that women had 1.25 y longer life expectancy at age 1 y (the table necessarily excludes the first year of life) than men: 24.62 y vs. 22.87 y. The estimated

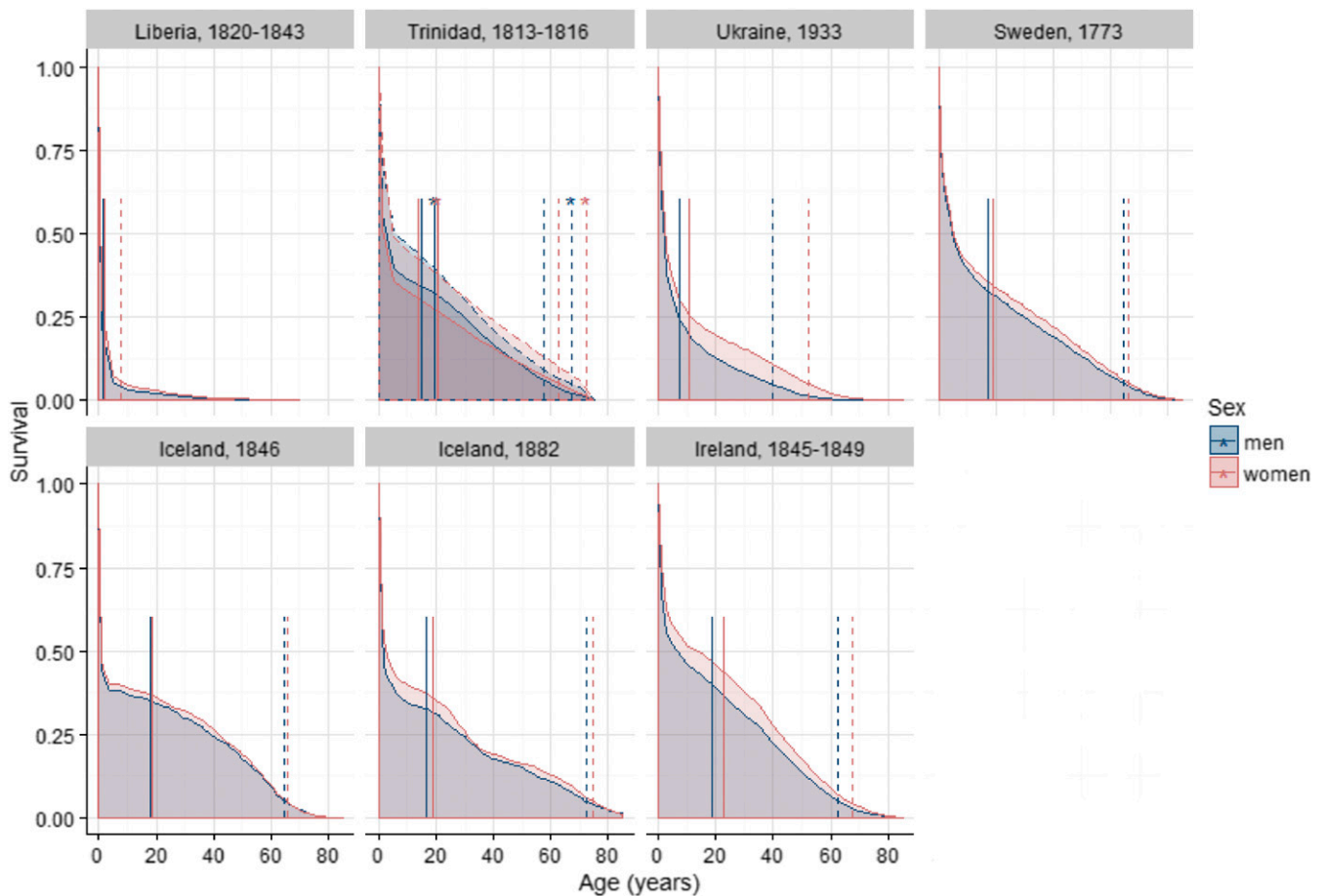


Fig. 1. Survival curves (shaded areas), life expectancies (solid vertical lines), and ages at which only 5% of a synthetic same-sex cohort would still be alive (dashed vertical lines) for seven high-mortality populations. For Trinidad, dashed survival curves and vertical lines with asterisks represent estimated upper bounds. Source: authors' calculations based on published data from ref. 25 for Liberia, from ref. 26 for Trinidad, from ref. 28 for Ukraine, from ref. 31 for Ireland, and from the Human Mortality Database (www.mortality.org) for Sweden and Iceland.

life expectancy in Ireland before the famine was 38.3 y for both men and women. No information pertaining to a condition of freedom was available for the slave population in Trinidad.

Gender Difference in Mortality During the Crisis. Life expectancy was higher for women than for men for all populations, with the partial exception of the Trinidad slaves for whom, according to the lower-bound life table, males lived slightly longer than females (Fig. 1 and Table 1). In the lower-bound life table, female slaves suffered from higher mortality than male slaves from birth until age 25 y, while in the upper-bound life table women had lower mortality than men at birth, experienced higher mortality until age 15 y, and afterward experienced lower mortality again. The general survival advantage of women is also reflected in the fact that the extreme age was higher for females than for males for all populations (Fig. 1).

For five populations—the two Icelandic populations and the Swedish, Irish, and Ukrainian populations—life expectancy estimates were available before and after the crisis, thus permitting evaluation of the absolute and relative impact of the crises for men and women (Table 2). The absolute reduction in life expectancy was higher for males than for females in Ireland; for the other four populations the absolute reduction was greater for females (who had higher life expectancies than males in these populations both before and during the crisis). The relative decrease in life expectancy was higher for males in the Ukraine and Ireland, higher for females in Iceland, and roughly the same in Sweden.

The sex difference in life expectancy varied among the populations (Table 1). Women among the freed slaves migrating

Table 2. Male and female decrease in life expectancy for five high-mortality populations during extreme mortality conditions

Population	Male		Female	
	Absolute, y	Relative	Absolute, y	Relative
Ukraine, 1933	34.28	0.82	35.08	0.76
Ireland, 1845–1849	19.60	0.51	15.90	0.41
Iceland, 1846	17.49	0.49	21.99	0.54
Iceland, 1882	20.86	0.55	25.16	0.57
Sweden, 1773	15.16	0.47	16.40	0.46

back to Liberia had the smallest absolute advantage over men (0.55 y). In Trinidad, as mentioned above, males lived longer than females (1.27 y) in the lower-bound scenario but not in the upper-bound scenario, in which the advantage was in favor of women (1.13 y). In relative terms, the largest advantages of women over men during crisis were in the Ukraine (a difference of almost 50%), Liberia (33%), and Ireland (20%). Among the other populations, the relative advantage was 12% or smaller.

A decomposition of the difference in life expectancy by age shows that the biggest contribution to these differentials comes from strikingly large mortality differences between male and female infants (Fig. 2). After age 1 y, mortality differences between the sexes contributed less and less to the total gap in life expectancy. Table 3 reports the share of the contribution of the 0–1-y age group and all other ages together with the total sex

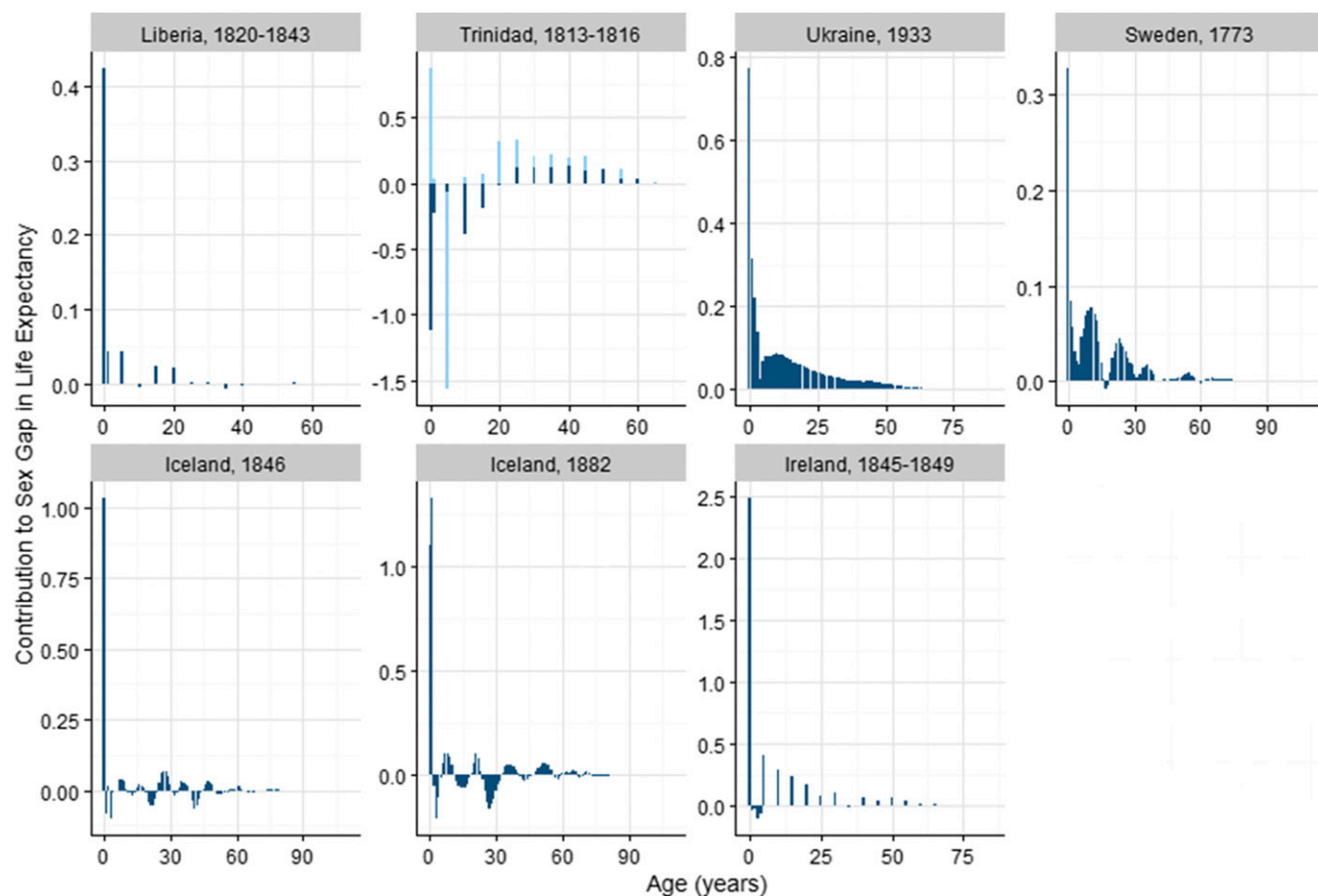


Fig. 2. Age decomposition of the differences in life expectancies between males and females for the eight high-mortality populations. Light blue bars for Trinidad represent the decomposition of the upper-bound life expectancy values. See Table S1.

Table 3. Age-specific share, in percentage points, of the total male–female difference in life expectancy for eight high-mortality populations during extreme mortality conditions

Population	Age 0–1 y, %	Age >1 y, %
Liberia, 1820–1843	77	23
Trinidad, 1813–1816	88–77*	12–23*
Ukraine, 1933	22	78
Sweden, 1773	20	80
Iceland, 1846	108 [†]	–8 [†]
Iceland, 1882	53	47
Ireland, 1845–1849	67	33

*Values refer to lower and upper bound.

[†]The values of 108% and –8% are explained by the fact that the contribution of the 0–1 age group to the sex gap in life expectancy was 1.033 y in favor of women, while the overall difference was 0.96 y because other ages contributed with negative values (in favor of men).

difference in life expectancy. In Liberia, Trinidad, Iceland, and Ireland infants contributed more than half of the total difference (with values in some cases reaching 70–80% of the difference). In Sweden and the Ukraine, the shares of the 0–1-y age group were around 20%, which still can be considered a substantial contribution for a single age group compared with all other ages.

Women lived longer than men in almost all populations, and higher proportions of women than men survived from birth to each age (Fig. 1). The female survival curves were higher than the male ones at all ages, with the exception of the Trinidad slaves. Here survival was higher for men than for women until age 50 y in the lower-bound scenario or until age 25 y in the upper-bound scenario, as a consequence of the higher female mortality in childhood and at young adult ages.

The absolute and relative differences in male vs. female mortality at each age provide further details about sex differences in mortality (Fig. 3). Within the study populations, the absolute and relative differences mostly follow a similar age trajectory, so that both absolute and relative differences are largest at similar ages. An exception is the Ukraine, where the absolute difference increases sharply from age 40 y, whereas the relative difference increases rapidly from birth to age 20 y and then decreases from age 60 y.

The ages at which the relative difference is highest vary. Almost all populations show a relative female survival advantage across all ages, with the exception of Liberia and Trinidad. Here males had a survival advantage at adult ages for Liberia (between age 35 y and age 49 y), and at infant and juvenile ages for Trinidad (until age 15 y or 25 y, for the upper- and lower-bound estimates, respectively).

Discussion

The conditions experienced by the people in the analyzed populations were horrific. Even though the crises reduced the female survival advantage in life expectancy, women still survived better than men. In all populations men had equal or higher mortality than women across almost all ages. A substantial part of the overall female advantage in life expectancy was due to survival differences among infants. Further support for the hypothesis of an overall ability of women to withstand high-mortality crises better than men comes from a different mortality measure: For all populations, the extreme age (the age to which 5% of the population survived) was higher for females than for males.

A female survival advantage has also been documented in more recent and less extreme famines. During the Dutch Hunger Winter (33), the famines of Madras and Bombay (34), five south Asian famines, the Bengal famine, and the famine in the Matlab region (35), the overall effect of the crisis was greater for men than for women, even in regions where women usually had higher mortality than men. The Matlab famine did not signifi-

cantly affect neonatal mortality, which increased only slightly (35). However, data on infants, especially during crises, must be considered with caution: Infants' deaths could be underreported when mortality increases (during a crisis) because more children die at very young ages, which increases the probability of the death not being reported (36).

In all populations under study, with the partial exception of the Trinidad slaves (in the case of the lower-bound scenario), females lived longer than males. These results indicate an important distinction: In populations that are exposed to harsh famines and epidemics the female survival advantage holds at all ages, whereas in slave populations in which stressors are or have been under some human control, males can have higher life expectancy and lower mortality than females, at least across some ages.

The slaves of Trinidad differ from the other populations in that their age-structure and mortality are heavily influenced by the decisions of the slave owners. Among the Trinidad slaves, young adult men had lower mortality than young women, perhaps because a premium was placed on their survival. Several studies show that male slaves employed in the plantations during the 19th century had a higher monetary value than female slaves (for both creole and African-born slaves) in the United States, Cuba, the British West Indies, and Brazil; only occasionally did female prices exceed those of males, namely, in urban areas, where women were valued for domestic work (37–39). The higher male mortality after age 15 y or age 25 y, depending on whether the upper- or lower-bound scenario is considered, could reflect their harder working conditions. A series of frequent revolts between 1638 and 1838 in the British Caribbean testifies to very tough working conditions (40–42). For example, as late as 1823 the planters of Barbados refused a proposal to give the slaves 1 d off per week, and those of Trinidad and British Guiana rejected a document by the British governor which proposed, among other things, a day off to permit religious instruction and the abolition of the whip; the planters argued that the whip was necessary to maintain discipline, and time for religious duties would merely encourage idleness among the slaves (43). Moreover, to keep sugar mills and boilers operating 24 h a day, slaves could work shifts up to 30 h long (25) while having minimal and inadequate nutrition, affected by periodic severe dietary deprivation and occasional near starvation, as showed by physical anthropological evidence (44). The skeletal and dental analyses suggest an average life expectancy at birth of 29 y for a population of slaves in a sugar plantation of Barbados between 1660 and 1820 (44). However, the authors point out, this estimate was severely biased by the highly inaccurate skeletal estimate of infant survival (95% infant survival against slightly more than 50% obtained from more accurate historical records for the same population) (44), implying that the real life expectancy value was much lower. Finally, the low life expectancy of the slave population of Trinidad could also be the consequence of Trinidad's being one of the three Caribbean colonies with the most rapidly expanding export sector in the 19th century. The demand for newly arrived slaves was larger in these colonies than in other colonies. Slaves just arrived in the Americas had lower life expectancy than those born there or who had been there already for some time, because of the adaptation period (called "seasoning") that lasted about 1 y after the arrival (45).

A similar explanation is not available for Liberia, where males had lower mortality than females between ages 35 y and 49 y. Several explanations can be hypothesized. The impossibility of determining the gender for 4% of the records, as mentioned above, might have caused some bias in the sex-specific death rates. A second explanation could be related to the need to establish a stable colony in an initially very hostile environment, which might have favored the individuals considered more important for this purpose, namely, adult men in the most productive

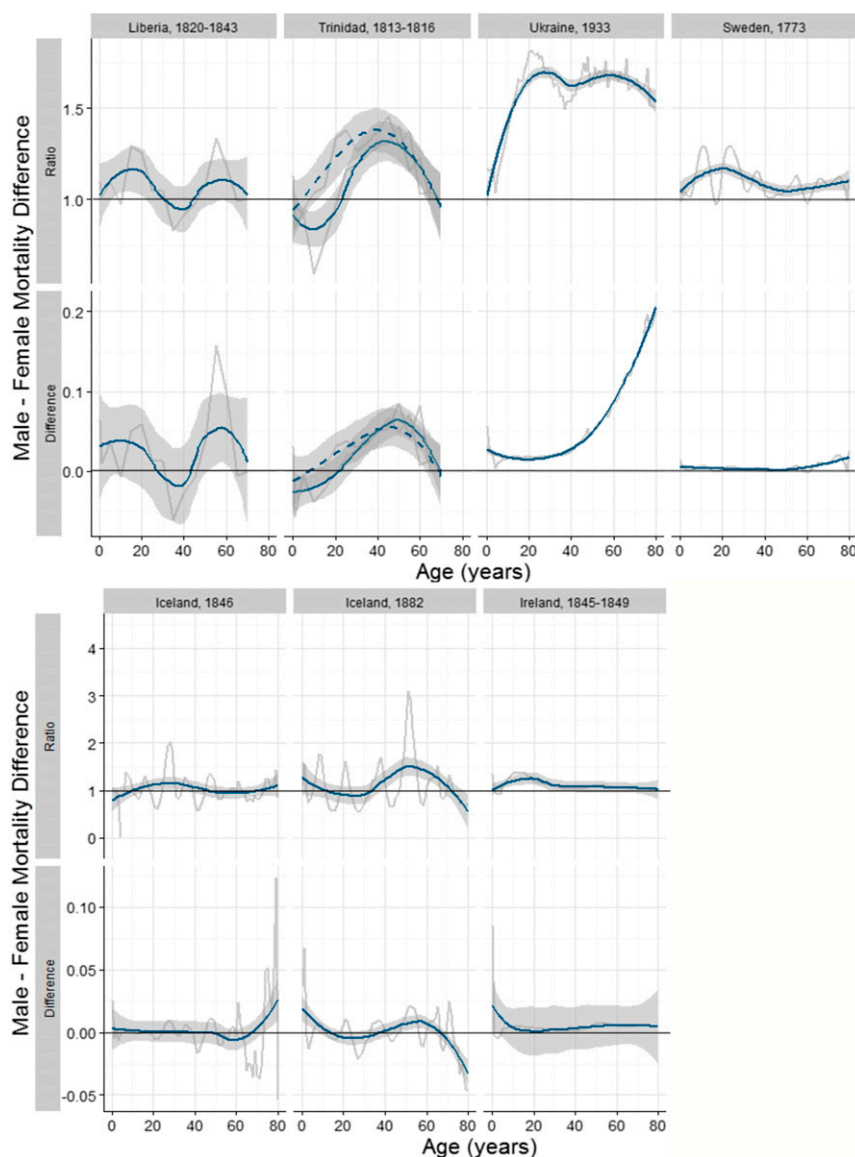


Fig. 3. Male:female mortality ratios and differences over age for seven high-mortality populations. Gray lines represent the unsmoothed data, blue lines represent the smoothed data [obtained with the R function `stat_smooth(100)`], and the gray shaded areas represent the SEs of the smoothing. The dashed lines for Trinidad represent the smoothed upper-bound values. Source: authors' calculations based on published data from ref. 25 for Liberia, from ref. 26 for Trinidad, from ref. 28 for Ukraine, from ref. 31 for Ireland, and from the Human Mortality Database (www.mortality.org) for Sweden and Iceland.

ages. Overall males in Liberia still had a lower life expectancy than females because adult ages contributed little to life expectancy, as shown by the age decomposition analysis.

A specific distribution of causes of death could partly explain these patterns, but this appears not to have been the case among the Liberian former slaves. This population is the only one for which we have a detailed list of causes of death. The registration shows that the distribution of causes of death is similar for males and females, except for deaths due to childbirth and gynecological diseases, which obviously affected only women, and deaths due to accident and violence, which were higher among men. This suggests another important point of discussion: that in times of crises, the well-known propensity of men more than women to die from accidents or violence could partly be the cause for their higher mortality. However, this is hard to assess with certainty because of the lack of data on causes of death in all the other cases (and for crisis populations in general). Secondly, if men die more from these causes, women still die from

childbirth and gynecological diseases that affect women exclusively, even in a context of reduced fertility such as that characterized by critical mortality conditions.

At the onset of famines and epidemics, the age structures of the populations at risk were shaped by previous mortality, fertility, and migration patterns. The risk of death then suddenly rose to extreme levels for everyone. Under these conditions, the youngest ages contributed the most to sex differences in life expectancy. The importance of infant and early childhood mortality was particularly striking in the two Icelandic epidemics. This was expected, as measles is mostly a childhood disease. Moreover, Iceland is the only case in which mortality increased relatively more for women than for men. This is consistent with the pattern of sex difference in measles survival: Excess female measles mortality is found worldwide (46), even with equal vaccination rates for both sexes, lower incidence of measles for girls than for boys, and higher and longer-persisting antibody titers on vaccination among girls than among boys (47, 48). It was

hypothesized that this difference could be due to different treatments by sex or to higher intensity of exposure for females, but studies from rural Senegal and Guinea Bissau showed no sign of sex bias in health maintenance, nutritional status, or breastfeeding patterns (49–52). Moreover, for the age group 5–14 y (in which most measles cases are contracted in school), conditions were similar for boys and girls, and among those infected at home boys and girls had the same mean intensity of exposure (49). Further investigations have led to the discovery that the administration of the high-titer measles vaccine at age 4–5 mo and the resulting shift of diphtheria, pertussis, and tetanus (DTP) and inactivated polio vaccine (IPV) administration after measles vaccination were associated with an increased female:male mortality ratio compared with the administration of the medium-titer measles vaccine at 9 mo, which usually comes after the DTP and IPV vaccines. These findings highlight a differential effect of vaccines depending on sex and sequence of vaccinations (53, 54). The relative increase of mortality in the two Icelandic epidemics was indeed bigger for women than for men, especially at young ages; this is the only case where this happens among the seven populations considered in this study. However, women still showed a marked overall survival advantage, and the survival advantage of girls contributed the most to the entire gender difference in life expectancy.

Infant age affects life expectancy the most when infant mortality is high, even in noncrisis years. However, it is striking that during epidemics and famines as harsh as those analyzed here newborn girls still survived better than newborn boys. Even in Liberia, the population with the lowest life expectancy, newborn girls were harder than newborn boys. That females survived more than males even at the infant ages, when behavioral differences are minimal, lends support to a biological underpinning of the female survival advantage. However, even if behavioral differences among infants are small, parents can have different attitudes toward children, depending on their sex. Studies show that in preindustrial Europe, the addition of a child increased the parents' mortality risk when resources were scarce and had to be shared. If the child was a boy, the mortality increase was the same for both mother and father, but if the child was a girl, the father's risk did not increase because he was not willing to share resources with an additional girl, while the mother's risk increased even more (55). Other studies found that the number of sons or daughters born or raised to adulthood had no effect on paternal longevity but did affect maternal postreproductive longevity (56) and that, irrespective of access to resources, having many sons reduced the survival of mothers but not of fathers (57).

Except for the slave population of Trinidad, in all the other cases starvation dysentery and diarrhea are likely to have been major causes of death. These causes are strongly associated with nutritional status, and therefore, the allocation of food might have played a key role in shaping the survival patterns. Evidence suggests that sudden changes in availability of food may not influence infant mortality during crises characterized by nutritionally related diseases: When the mother breastfeeds, the infant is protected because breast milk appears to be sufficient until the mother is nearly starved (58). For other age groups, studies on practices of resource allocation from various areas of preindustrial Europe show a penalty for women, especially at young ages. From 1775 to 1850 the preexisting female excess mortality between the ages of 1 and 14 y increased sharply, mostly due to discrimination in the resource allocation within the household (55, 59).

Widespread social practices could be disrupted by famines or epidemics. Famines were often accompanied by prostitution, child abandonment or infanticide, aberrant food practices, and massive migration flows (60). Some of these factors could act in favor of women; others could be detrimental to them. While increased prostitution rates and migration could partly contrib-

ute to the higher survival chances of women (through prostitution women are able to get extra resources; migration reduces the pressure on the scarce resources, thus offering some relief from hunger to those who stay, composed mostly of women, children, and the elderly), child abandonment and infanticide, at least in some cases, could favor boys at the expenses of girls (60). Various stories from different crises testify to maternal resilience and tell of mothers taking extreme actions that led to the survival of both mother and infant (61–63).

A growing body of research on sex differences in mortality and immunoresponse among humans and other mammals supports the fundamentally biological foundations of sex differences in human mortality. Biological factors include hormonal and chromosomal genetic differences. Sex hormones seem to play a key role (64, 65): estrogens have antiinflammatory, vasoprotective effects (66–68), whereas testosterone seems to increase the mortality risk for certain diseases (69, 70), although the evidence on this point is mixed (71, 72). Moreover, while estrogens enhance immune defenses, testosterone and progesterone may have immunosuppressive effects (73–75). The presence of two X-chromosomes may pose a further advantage with respect to specific X-linked diseases (e.g., hemophilia A) due to an amelioration of harmful gene mutations through nonmutated alleles on the other X-chromosome. The possibility of having two different alleles on the two X-chromosomes further contributes to the physiological diversity that can be advantageous when encountering new immune challenges (76–79).

Females live longer than males in humans and in the large majority of monkeys and apes for which data are available, in both captive and wild populations (80). Mammalian females generally outlive males in species in which males compete with each other for opportunities to mate (81, 82). This occurs in polygynous species and is commonly accompanied by sexual dimorphism in body size, which helps males compete for females. The sexual dimorphism in human body size indicates that our evolutionary history contained a long period of polygynous reproduction (83). Furthermore, the ratio of testes to body size is larger in polygynously mating species than in monogamous species (84). The relative testes size of humans in comparison with other species is further evidence that humans mated polygynously during their evolutionary history (84). Therefore, from an evolutionary perspective, the observed sex differences in human mortality are not exceptional; instead, humans fall well within the range of sex differences observed in other mammal species (81, 82, 85). Furthermore, among vertebrates males are more likely to be infected with parasites and to carry a greater intensity of infection than female conspecifics (86). It has been argued that this is due to an immunosuppressive effect of testosterone (86), but evidence is mixed (87). An alternative explanation comes from one experimental study that points toward a role of testosterone in altering social behavior so as to increase exposure to infection rather than the hormone acting as an immunosuppressant (88). It has further been argued that increases in Darwinian fitness accompanying a higher investment in the immune system in females, but not in males, may be sufficient to explain the observed sex differences in immune response (87, 89). Female mammals not only seem to be better at dealing with infection but also survive better than male mammals under harsh environmental conditions—an observation confirmed by a large comparative study on 26 ungulate populations (90), among others.

Research has also provided evidence for an apparent female advantage in immune protection among humans: The incidence of many bacterial, viral, parasitic, and fungal infectious diseases (e.g., leptospirosis, chistosomiasis, brucellosis, rabies, leishmaniasis, pulmonary tuberculosis, hepatitis A, meningococcal and pneumococcal infections, and seasonal influenza) is substantially higher in men than in premenopausal women. This suggests that progesterone and testosterone have mainly immunosuppressive

effects, whereas estrogens enhance immune defenses (73–75) and act as antioxidant (91). Moreover, autoimmune diseases are more prevalent in women than in men, as is a stronger immune response to vaccinations (74, 92). These findings led researchers to conclude that low male immunocompetence contributes to sex differences in mortality (93), but the mechanisms through which sex hormones affect immune responses in humans have not been fully elucidated.

Additionally, behavioral factors have been identified as important determinants of the male–female survival difference in contemporary populations (94, 95). The high preponderance of risk-taking behaviors among men contributes substantially to the sex gap in life expectancy. Men consume tobacco, alcohol, and psychoactive substances in greater quantities, drive less safely, and eat less salubriously than women do; this results in elevated

risks of cardiovascular diseases, lung cancer, liver cirrhosis, and accident fatalities (96, 97). In high-income countries cigarette smoking has been identified as the largest factor contributing to the mortality differential (98, 99). However, although behaviors are important factors, they cannot fully explain the sex difference in survival, as suggested by the fact that some female advantage is found among nonsmokers (6, 7), devout Mormons (8), and Catholic nuns vs. monks (9).

In almost all human populations women live longer than men. In this study we found that the female survival advantage extends to seven documented populations experiencing high-mortality crises. Our results add another piece to the puzzle of gender differences in survival. They suggest that the female advantage stems from fundamental biological roots and is influenced by socially and environmentally determined risks, opportunities, and resources.

- Austad SN (2006) Why women live longer than men: Sex differences in longevity. *Gend Med* 3:79–92.
- Barford A, Dorling D, Davey Smith G, Shaw M (2006) Life expectancy: Women now on top everywhere. *BMJ* 332:808.
- Glei DA, Horiuchi S (2007) The narrowing sex differential in life expectancy in high-income populations: Effects of differences in the age pattern of mortality. *Popul Stud (Camb)* 61:141–159.
- Kalben BB (2000) Why men die younger: Causes of mortality differences by sex. *N Am Actuar J* 4:83–111.
- Wachter KW (2008) Biodemography comes of age. *Demogr Res* 19:1501–1512.
- Bronnum-Hansen H, Juel K (2001) Abstinence from smoking extends life and compresses morbidity: A population based study of health expectancy among smokers and never smokers in Denmark. *Tob Control* 10:273–278.
- Rogers RG, Powell-Griner E (1991) Life expectancies of cigarette smokers and non-smokers in the United States. *Soc Sci Med* 32:1151–1159.
- Lindahl-Jacobsen R, et al. (2013) The male-female health-survival paradox and sex differences in cohort life expectancy in Utah, Denmark, and Sweden 1850–1910. *Ann Epidemiol* 23:161–166.
- Luy M (2003) Causes of male excess mortality: Insights from cloistered populations. *Popul Dev Rev* 29:647–676.
- Boco AG (2015) Assessing sex differentials in under-five mortality in sub-Saharan Africa: A cross-national comparative analysis. *Can Stud Popul* 41:49–87.
- Drevenstedt GL, Crimmins EM, Vasunilashorn S, Finch CE (2008) The rise and fall of excess male infant mortality. *Proc Natl Acad Sci USA* 105:5016–5021.
- Ulizzi L, Zonta LA (2002) Sex differential patterns in perinatal deaths in Italy. *Hum Biol* 74:879–888.
- Grayson DK (1993) Differential mortality and the Donner Party disaster. *Evol Anthropol Issues News Rev* 2:151–159.
- Ferlay J, et al. (2013) Cancer incidence and mortality patterns in Europe: Estimates for 40 countries in 2012. *Eur J Cancer* 49:1374–1403.
- Isaksson R-M, et al. (2011) Better long-term survival in young and middle-aged women than in men after a first myocardial infarction between 1985 and 2006. An analysis of 8630 patients in the northern Sweden MONICA study. *BMC Cardiovasc Disord* 11:1–8.
- Jagger C, et al.; EHLEIS team (2008) Inequalities in healthy life years in the 25 countries of the European Union in 2005: A cross-national meta-regression analysis. *Lancet* 372:2124–2131.
- Nusselder WJ, Looman CW, Van Oyen H, Robine JM, Jagger C (2010) Gender differences in health of EU10 and EU15 populations: The double burden of EU10 men. *Eur J Ageing* 7:219–227.
- Van Oyen H, et al. (2010) Gender gaps in life expectancy and expected years with activity limitations at age 50 in the European Union: Associations with macro-level structural indicators. *Eur J Ageing* 7:229–237.
- Andersen KK, Andersen ZJ, Olsen TS (2011) Predictors of early and late case-fatality in a nationwide Danish study of 26,818 patients with first-ever ischemic stroke. *Stroke* 42:2806–2812.
- Koek HL, et al. (2006) Short- and long-term prognosis after acute myocardial infarction in men versus women. *Am J Cardiol* 98:993–999.
- Nielsen S, et al. (2014) Sex-specific trends in 4-year survival in 37 276 men and women with acute myocardial infarction before the age of 55 years in Sweden, 1987–2006: A register-based cohort study. *BMJ Open* 4:e004598.
- Preston SH, McDaniel A, Grushka C (1993) New model life tables for high-mortality populations. *Hist Methods* 26:149–159.
- McDaniel A, Preston SH (1994) Patterns of mortality by age and cause of death among nineteenth-century immigrants to Liberia. *Popul Stud (Camb)* 48:99–115.
- McDaniel A (1992) Extreme mortality in nineteenth-century Africa: The case of Liberian immigrants. *Demography* 29:581–594.
- John AM (1988) Plantation slave mortality in Trinidad. *Popul Stud (Camb)* 42:161–182.
- John AM (1988) *The Plantation Slaves of Trinidad, 1783–1816: A Mathematical and Demographic Enquiry* (Cambridge Univ Press, New York).
- Meslé F, Vallin J (2012) *Mortality and Causes of Death in 20th-Century Ukraine* (Springer Science & Business Media, New York).
- Dribe M, Olsson M, Svensson P (2015) Famines in the Nordic countries, AD 536–1875. *Lund Papers in Economic History. General Issues* (138) (Lund University, Lund, Sweden).
- Cliff AD, Haggett P, Graham R (1983) Reconstruction of diffusion at local scales: The 1846, 1882 and 1904 measles epidemics in northwest Iceland. *J Hist Geogr* 9:347–368.
- Boyle PP, O Gráda C (1986) Fertility trends, excess mortality, and the Great Irish Famine. *Demography* 23:543–562.
- Preston S, Heuveline P, Guillot M (2001) *Demography: Measuring and Modeling Population Processes* (Blackwell Publishers, Oxford).
- Andreev EM, Shkolnikov VM, Begun AZ (2002) Algorithm for decomposition of differences between aggregate demographic measures and its application to life expectancies, healthy life expectancies, parity-progression ratios and total fertility rates. *Demogr Res* 7:499–522.
- Dols MJ, Van Arcken DJ (1946) Food supply and nutrition in the Netherlands during and immediately after World War II. *Milbank Mem Fund Q* 24:319–358.
- McAlpin MB (2014) *Subject to Famine: Food Crisis and Economic Change in Western India, 1860–1920* (Princeton Univ Press, Princeton).
- Dyson T (1991) On the demography of south Asian famines. Part II. *Popul Stud (Camb)* 45:279–297.
- Menken J, Campbell C (1992) Age-patterns of famine-related mortality increase: Implications for long-term population growth. *Health Transit Rev* 2:91–101.
- Lennon C (2016) Slave escape, prices, and the fugitive slave act of 1850. *J Law Econ* 59:669–695.
- Olmstead AL, Rhode PW (2008) *Creating Abundance: Biological Innovation and American Agricultural Development* (Cambridge Univ Press, New York).
- Fraginals MM, Klein HS, Engerman SL (1983) The level and structure of slave prices on Cuban plantations in the mid-nineteenth century: Some comparative perspectives. *Am Hist Rev* 88:1201–1218.
- Beckles H (1988) Caribbean anti-slavery the self-liberation ethos of enslaved Blacks. *J Caribb Hist* 22:1–19.
- Hart R (1980) *Slaves Who Abolished Slavery: Blacks in Bondage* (Institute of Social and Economic Research, Kingston, Jamaica), Vol 1.
- Hart R (1985) *Slaves Who Abolished Slavery: Blacks in Rebellion* (Institute of Social and Economic Research, Kingston, Jamaica), Vol 2.
- Williams E (1944) *Capitalism and Slavery* (Univ of North Carolina Press, Chapel Hill, NC).
- Handler JS, Corruccini RS (1983) Plantation slave life in Barbados: A physical anthropological analysis. *J Interdiscip Hist* 14:65–90.
- Eltis D, Lewis FD, Richardson D (2005) Slave prices, the African slave trade, and productivity in the Caribbean, 1674–1807. *Econ Hist Rev* 58:673–700.
- Garenne M (1994) Sex differences in measles mortality: A world review. *Int J Epidemiol* 23:632–642.
- Garenne M (2015) Demographic evidence of sex differences in vulnerability to infectious diseases. *J Infect Dis* 211:331–332.
- Muenchhoff M, Goulder PJ (2014) Sex differences in pediatric infectious diseases. *J Infect Dis* 209(Suppl 3):S120–S126.
- Garenne M, Aaby P (1990) Pattern of exposure and measles mortality in Senegal. *J Infect Dis* 161:1088–1094.
- Svedberg P (1990) Undernutrition in Sub-Saharan Africa: Is there a gender bias? *J Dev Stud* 26:469–486.
- Aaby P, et al. (1993) Long-term survival after Edmonston-Zagreb measles vaccination in Guinea-Bissau: Increased female mortality rate. *J Pediatr* 122:904–908.
- Aaby P, et al. (1994) Sex-specific differences in mortality after high-titre measles immunization in rural Senegal. *Bull World Health Organ* 72:761–770.
- Aaby P, et al. (2007) Increased female-male mortality ratio associated with inactivated polio and diphtheria-tetanus-pertussis vaccines: Observations from vaccination trials in Guinea-Bissau. *Pediatr Infect Dis J* 26:247–252.
- Aaby P, et al. (2010) Sex differences in the effect of vaccines on the risk of hospitalization due to measles in Guinea-bissau. *Pediatr Infect Dis J* 29:324–328.
- Klasen S (1998) Marriage, bargaining, and intrahousehold resource allocation: Excess female mortality among adults during early German development, 1740–1860. *J Econ Hist* 58:432–467.
- Helle S, Lummaa V, Jokela J (2002) Sons reduced maternal longevity in preindustrial humans. *Science* 296:1085.

57. Helle S, Lummaa V (2013) A trade-off between having many sons and shorter maternal post-reproductive survival in pre-industrial Finland. *Biol Lett* 9:20130034.
58. McCann MF, Liskin LS, Piotrow PT, Rinehart W, Fox G (1981) Breast-feeding, fertility, and family planning. *Popul Rep J* 5:525–575.
59. McNay K, Humphries J, Klasen S (2005) Excess female mortality in nineteenth-century England and Wales. *Soc Sci Hist* 29:649–681.
60. Gráda C (2009) *Famine: A Short History* (Princeton Univ Press, Princeton).
61. Edgerton-Tarpley K (2004) Family and gender in famine: Cultural responses to disaster in North China, 1876–1879. *J Womens Hist* 16:119–147.
62. Greenough PR (1982) *Prosperity and Misery in Modern Bengal: The Famine of 1943–1944* (Oxford Univ Press, Oxford).
63. Hodges S (2005) ‘Looting’ the lock hospital in colonial Madras during the famine years of the 1870s. *Soc Hist Med* 18:379–398.
64. Vaccarino V, et al. (2010) Ischaemic heart disease in women: Are there sex differences in pathophysiology and risk factors? Position paper from the Working Group on Coronary Pathophysiology and Microcirculation of the European Society of Cardiology. *Cardiovasc Res* 90:9–17.
65. Waldron I (1983) Sex differences in human mortality: The role of genetic factors. *Soc Sci Med* 17:321–333.
66. Xing D, Nozell S, Chen Y-F, Hage F, Oparil S (2009) Estrogen and mechanisms of vascular protection. *Arterioscler Thromb Vasc Biol* 29:289–295.
67. Babiker FA, et al. (2002) Estrogenic hormone action in the heart: Regulatory network and function. *Cardiovasc Res* 53:709–719.
68. Wise PM, Dubal DB, Rau SW, Brown CM, Suzuki S (2005) Are estrogens protective or risk factors in brain injury and neurodegeneration? Reevaluation after the women’s health initiative. *Endocr Rev* 26:308–312.
69. Haring R, et al. (2010) Low serum testosterone levels are associated with increased risk of mortality in a population-based cohort of men aged 20–79. *Eur Heart J* 31:1494–1501.
70. Holmegard HN, Nordestgaard BG, Jensen GB, Tybjaerg-Hansen A, Benn M (2016) Sex hormones and ischemic stroke: A prospective cohort study and meta-analyses. *J Clin Endocrinol Metab* 101:69–78.
71. Ruige JB, Mahmoud AM, De Bacquer D, Kaufman JM (2011) Endogenous testosterone and cardiovascular disease in healthy men: A meta-analysis. *Heart* 97:870–875.
72. Schooling CM (2016) Could androgens be relevant to partly explain why men have lower life expectancy than women? *J Epidemiol Community Health* 70:324–328.
73. Bouman A, Heineman MJ, Faas MM (2005) Sex hormones and the immune response in humans. *Hum Reprod Update* 11:411–423.
74. Gieffing-Kröll C, Berger P, Lepperdinger G, Grubeck-Loebenstein B (2015) How sex and age affect immune responses, susceptibility to infections, and response to vaccination. *Aging Cell* 14:309–321.
75. Pennell LM, Galligan CL, Fish EN (2012) Sex affects immunity. *J Autoimmun* 38:J282–J291.
76. Morris JA, Harrison LM (2009) Hypothesis: Increased male mortality caused by infection is due to a decrease in heterozygous loci as a result of a single X chromosome. *Med Hypotheses* 72:322–324.
77. Spolarics Z (2007) The X-files of inflammation: Cellular mosaicism of X-linked polymorphic genes and the female advantage in the host response to injury and infection. *Shock* 27:597–604.
78. Christensen K, et al. (2000) X-linked genetic factors regulate hematopoietic stem-cell kinetics in females. *Blood* 95:2449–2451.
79. Christensen K, Ørstavik KH, Vaupel JW (2001) The X chromosome and the female survival advantage: An example of the intersection between genetics, epidemiology and demography. *Ann N Y Acad Sci* 954:175–183.
80. Austad SN, Fischer KE (2016) Sex differences in lifespan. *Cell Metab* 23:1022–1033.
81. Clutton-Brock TH, Isvaran K (2007) Sex differences in ageing in natural populations of vertebrates. *Proc Biol Sci* 274:3097–3104.
82. Promislow DE (1992) Costs of sexual selection in natural populations of mammals. *Proc R Soc Lond B Biol Sci* 247:203–210.
83. Mitani JC, Gros-Louis J, Richards AF (1996) Sexual dimorphism, the operational sex ratio, and the intensity of male competition in polygynous primates. *Am Nat* 147:966–980.
84. Harcourt AH, Harvey PH, Larson SG, Short RV (1981) Testis weight, body weight and breeding system in primates. *Nature* 293:55–57.
85. Colchero F, et al. (2016) The emergence of longevous populations. *Proc Natl Acad Sci USA* 113:7681–7690.
86. Zuk M, McKean KA (1996) Sex differences in parasite infections: Patterns and processes. *Int J Parasitol* 26:1009–1023.
87. Nunn CL, Lindenfors P, Pursall ER, Rolff J (2009) On sexual dimorphism in immune function. *Philos Trans R Soc Lond B Biol Sci* 364:61–69.
88. Great DA, Perkins SE, Hudson PJ (2009) Does elevated testosterone result in increased exposure and transmission of parasites? *Ecol Lett* 12:528–537.
89. Roth O, Schar sack JP, Keller I, Reusch TB (2011) Bateman’s principle and immunity in a sex-role reversed pipefish. *J Evol Biol* 24:1410–1420.
90. Toïgo C, Gaillard JM (2003) Causes of sex-biased adult survival in ungulates: Sexual size dimorphism, mating tactic or environment harshness? *Oikos* 101:376–384.
91. Moosmann B, Behl C (1999) The antioxidant neuroprotective effects of estrogens and phenolic compounds are independent from their estrogenic properties. *Proc Natl Acad Sci USA* 96:8867–8872.
92. Whitacre CC (2001) Sex differences in autoimmune disease. *Nat Immunol* 2:777–780.
93. Owens IPF (2002) Ecology and evolution. Sex differences in mortality rate. *Science* 297:2008–2009.
94. Lemaire J (2002) Why do females live longer than males? *N Am Actuar J* 6:21–37.
95. Thorslund M, Wastesson JW, Agahi N, Lagergren M, Parker MG (2013) The rise and fall of women’s advantage: A comparison of national trends in life expectancy at age 65 years. *Eur J Ageing* 10:271–277.
96. Waldron I (1985) What do we know about causes of sex differences in mortality? A review of the literature. *Popul Bull UN* 59–76.
97. Wardle J, et al. (2004) Gender differences in food choice: The contribution of health beliefs and dieting. *Ann Behav Med* 27:107–116.
98. Preston SH, Wang H (2006) Sex mortality differences in the United States: The role of cohort smoking patterns. *Demography* 43:631–646.
99. Beltrán-Sánchez H, Finch CE, Crimmins EM (2015) Twentieth century surge of excess adult male mortality. *Proc Natl Acad Sci USA* 112:8993–8998.
100. R Core Team (2013) R: A language and environment for statistical computing (R Foundation for Statistical Computing, Vienna), Version 3.3.3.