Divergent Trends of Lifespan Variation During Mortality Crises

Abstract

Lifespan variation has been attracting increasingly greater attention as a measure of population health and mortality. Several studies have analysed periods of steady mortality decline, highlighting a strong inverse relationship between lifespan variation and life expectancy. Recent research has found that this association weakens, and even reverses, when mortality does not improve equally over age. However, to date no study has comprehensively explored the behaviour of lifespan variation when mortality increases significantly. Analysing three epidemics and two famines in Europe from the eighteenth to the twentieth centuries, we find that, during these events, relative lifespan variation increases, while absolute variation declines, and that subsequently both quickly revert to pre-crisis levels. Using decomposition techniques, we show that mortality at older ages leads to a temporary increase in absolute – but not relative – variation. Moreover, female lifespan variation is less affected by the crises than that of males, because of the higher impact of infant and child mortality on male lifespan variation. By underlining different trends of lifespan variation by sex and indicator, we offer new insight into the consequences of mortality crises. Contrary to what is often asserted, we also show that the choice of lifespan variation indicator is not always inconsequential.

Introduction

Life expectancy is often used as a summary measure of population health and mortality, as it expresses the average remaining years of life in a given year, if the individuals in a population experienced the current conditions throughout their lives (Preston et al. 2001). However, it can camouflage other important characteristics, such as variation in length of life, also known as lifespan variation. Lifespan variation, which can describe uncertainty in the timing of death at the individual level and underlies heterogeneity in population health at the aggregate level, has decreased as life expectancy has increased (Aburto et al. 2020; Colchero et al. 2016; Kannisto 2001; Smits and Monden 2009; Vaupel et al. 2011). Yet, these two measures can follow divergent trends, so that an increase in life expectancy is not necessarily followed by a decline in lifespan variation (Aburto and van Raalte 2018; Brønnum-Hansen 2017; Sasson 2016; Wilmoth and Horiuchi 1999).

Studies on lifespan variation have mostly focused on populations with continued mortality improvements or else they have analysed socioeconomic differences (Edwards and Tuljapurkar 2005; Lariscy et al. 2016; Permanyer et al. 2018;van Raalte et al. 2011). More recently, studies have considered periods when life expectancy stagnates or decreases (Aburto and Beltrán-Sánchez 2019; García and Aburto 2019; Permanyer and Scholl 2019; Sasson 2016). To the best of our knowledge, lifespan variation has rarely been studied when mortality increases sharply. With the exception of Colchero et al. (2016), who find that females experience lower lifespan variation than males during mortality crises, there is a knowledge gap about trends in lifespan variation during mortality shocks.

By focusing on populations that experience mortality crises, such as famines and epidemics, we contribute to filling this gap in two ways. First of all, by analysing lifespan variation, we uncover the effect of massive mortality shocks on heterogeneity, which would be overlooked when using life expectancy (Zarulli et al. 2018). Secondly, by comparing the patterns of a diverse set of measures of lifespan variation, we shed light on their behaviour during periods of substantial mortality increase and show that the choice of indicator is not always inconsequential.

Background

A strong negative relationship between life expectancy and lifespan variation has been highlighted by numerous studies, across time, countries and social groups (Sasson 2016; van Raalte et al. 2011; Vaupel et al. 2011; Wilmoth and Horiuchi 1999). However, recent studies question this relationship. For example, since the 1980s all Danes have enjoyed longer lives on average, but lifespan variation increased for the least educated (Brønnum-Hansen 2017), a trend found also in Finland, Spain and the USA (Permanyer et al. 2018; Sasson 2016; van Raalte et al. 2014). In fact, the correlation between the two measures depends on the distribution of mortality changes across the ages (Aburto et al. 2020; Aburto and van Raalte 2018; García and Aburto 2019) and even when there is a correlation, life expectancy does not completely predict lifespan variation (van Raalte et al. 2011).

Mortality crises have also been examined. What constitutes a mortality crisis has been debated, from Goubert's initial suggestion of a doubling of the number of deaths (Goubert 1958) to more complex indices (for an introduction to the issue, see Charbonneau and Larose (1970)). Nevertheless, mortality crises can be classified in four groups in relation to their causes (Sogner in Charbonneau and Larose (1970); Goubert 1958): i) subsistence crises (or famines) are brought about by a lack or unavailability of food to a considerable part of the population; ii) epidemics are caused by diseases (2); some crises combine both components, such as when a famine weakens the population, leaving it vulnerable to pathogens; iv) crises can have other causes, such as wars or natural phenomena. This latter class of crises presents specific dynamics, making it difficult to compare them with the others. Therefore, we focus on the first three types. We adopt a straightforward definition of a mortality crisis: a period when life expectancy at birth e_0 decreased drastically and quite suddenly. For the purposes of this article, we only consider relatively short crises, spanning up to two years.

No single mortality pattern can be expected during crises, because age and gender-specific death rates vary. Males and older individuals seem to be at higher risk of dying from COVID-19 (Sharma et al. 2020; Verity et al. 2020), while other crises – such as natural disasters – affect more women, children and the elderly (Bern et al. 1993; Frankenberg et al. 2011; Neumayer and Plümper 2007). Bongaarts and Cain (1982, in Kane 1987) hypothesised that, in the case of short famines, mortality would reach its peak at the end of the famine period and then gradually decline. After about a year it could even drop below pre-famine levels (as was found

by Goubert (1958)), as only the most robust individuals had survived. However, age and gender-specific mortality patterns vary depending on cultural and social environments. Bengtsson et al. (2009) found that Scandinavian infants were generally less affected by food availability, as they depend on breastfeeding, while older children were more sensitive to it. Because of breastfeeding and pregnancy, women might be more vulnerable to food deprivation, in addition to potential discrimination in the household food distribution (Klasen 1998). Notwithstanding this, females experience lower mortality at almost all ages, even in populations that experience extremely high mortality – possibly because of biological advantages. However, this gap can reverse because of social preferences and the incidence of gynaecological diseases or childbirth complications (Zarulli et al. 2018). Finally, the elderly are also affected, but show little differences in terms of gender and socio-economic status, possibly as a result of selection into old age (Bengtsson et al. 2009).

Epidemics are more complicated, as each disease presents specific characteristics. For example, malaria affects predominantly young children (WHO 2018), while today the elderly are generally more vulnerable to influenza and, in developed countries, to tuberculosis (Yoshikawa and Norman 2009). Age can also interact with gender (Garenne 2015) and social characteristics can affect morbidity and mortality, such as during the HIV/AIDS epidemic of the 1980s in the USA (Gaylin and Kates 1997). Here, we consider three epidemics, two caused by measles and one by typhus and dysentery. Measles typically affects children, but also non-immunised adults, and causes greater female mortality (Garenne 2015; Muenchhoff and Goulder 2014), while no clear sex differences have been found for typhus and dysentery, which kill, in particular, weakened individuals (Castenbrandt 2014; Goble and Konopka 1973; Taylor et al. 2015).

Context

We study five crises. The 1772-1773 famine and 1808-1809 typhus and dysentery epidemic in Sweden, the Icelandic 1846 and 1882 measles epidemics and, finally, the Ukrainian famine of 1933.

Dribe et al. (2015) describe the 1772-1773 famine and the 1808-1809 epidemic. Crop failures in large regions of Sweden caused by unusual weather in 1772 exacerbated high food prices and led to a famine which peaked the following year. In 1773, death rates were 86% higher in the most affected counties than in the others

and crude death rates doubled in central Sweden. Although all age groups were affected, children between ages 1 and 14 suffered the most in proportional terms, while infants witnessed a relatively small proportional increase in mortality. Mortality was mostly driven by nutrition-related diseases, specifically typhus and dysentery (which alone accounted for 50% of the excess mortality that year). Typhus and dysentery were, likewise, the diseases involved in the 1808-1809 epidemic, which followed troop movements involved in the Finnish War. Despite the impact of the war, the increase in mortality is thought to have resulted mainly from the epidemic (Glei et al. 2019). As a consequence, mortality followed the same age pattern as in 1772-1773.

Icelandic history is fraught with bad years, "due to cold winters, ice floes, failures of fisheries, shipwrecks, inundations, volcanic eruptions, earthquakes, epidemics and contagious diseases among men and animals" (Magnùs Stephensen, in Tomasson (1977:410)). In the years 1846 and 1882, measles struck. In both years, particularly cold springs and summers forced fishermen to remain ashore, facilitating the spread of the disease, which was brought by Danish sailors. In 1846, even the oldest Icelanders had never been in contact with measles, which spread rapidly through the unimmunised population. Although mortality increased for all ages, children and the elderly were affected more severely. The epidemic lasted from July to December and caused the death of around 3% of the population. The individuals who survived were better prepared to face the subsequent epidemic in 1882, which affected ages under 40. That epidemic lasted only from June to August and led to the death of around 2% of the population (Cliff et al. 1983; Shanks et al. 2015).

The context of the Ukrainian famine of 1932-1933 is more difficult to describe, as data were not publicly accessible until the dissolution of the Soviet Union. Naumenko (2019) reports three main competing explanations. First, bad weather would have lowered harvest yields in 1932, leading to famine in the following months (Tauger 1991). Others consider that the crop output declined because of the collectivisation policies that had been implemented since the late 1920s. A poor harvest in 1931 and the effort by the government to maintain food distribution in the cities caused hunger in the countryside, which evolved into widespread famine in the following years (as Naumenko herself maintains). Finally, a third strand of research argues that the famine was a result of the government's decision to curtail food distribution in order to quell anti-government sentiment in Ukraine, which had been sparked by collectivisation policies (Graziosi 2015). Nevertheless, it is agreed that, during these years, Ukraine experienced a harsh famine, which led to the death of between 2.1 and 3.9 million people (Meslé and Vallin 2012; Rudnytskyi et al. 2015).

Research Questions

Our first research question looks at trends in mortality increase and lifespan variation. We anticipate three main patterns of mortality change. For the two Swedish and the Ukrainian crises, we expect to see a proportional increase in mortality at adult ages, together with some convergence at older ages and a disproportionate increase of child mortality, at least in Sweden (Dribe et al. 2015). In Iceland, we anticipate two different patterns: for the first epidemic, a proportionately greater increase of mortality in children and the elderly; for the second, a greater increase in children only, as the elderly had already acquired immunity.

While increases in mortality at any age lower life expectancy, increases at different ages may have divergent effects on lifespan variation. Increases in mortality at young ages typically contribute to increasing lifespan variation. However, increases at older ages compress the mortality distribution, so that these ages contribute to a decline in lifespan variation. Each mortality distribution presents a unique and specific age at which contributions go from positive to negative (and vice versa when mortality declines). This is called the threshold age and it is usually close to life expectancy at birth (Aburto et al. 2019; Gillespie et al. 2014; Zhang and Vaupel 2009). Thus, the expected mortality increases for children and the elderly would contribute to the change in lifespan variation in opposite directions, as each age group sits on a different side of the threshold age. However, we expect children to be more strongly affected by the crises than older individuals, as only the more robust survive to advanced ages. Therefore, in all five crises, we expect lifespan variation to initially increase and then decline – either gradually, if the consequences of the crisis continue to affect weakened individuals – or sharply, if there is selection at the vulnerable ages during the crisis, as was found for Ukraine (Zarulli 2013).

The second research question analyses gender differences. Colchero et al. (2016) show that the gender gap in lifespan variation, which generally favours females (van Raalte 2011), does not change in high mortality situations. We expect similar results for our analyses. We also expect the trends to differ between sexes according to the gender specificity of the crisis in question; for example, with starker changes for females during the measles epidemics.

Our third question is to determine whether some ages contributed more than others to the change in variation during and after the crisis. We anticipate that a mortality crisis will particularly affect children (with the exception of infants,

protected by breastfeeding) and the elderly. These sub-groups are physically less equipped to deal with extreme conditions and children might receive less care in situations where resources are scarce, although the latter phenomenon is context-specific (Lanau and Fifita 2020). Gender discrimination can also exacerbate this uneven allocation of resources for girls. Moreover, deaths at the extremes of a distribution will have a greater effect on variation. Therefore, we expect that these age groups will increase lifespan variation. However, because of the reasons outlined earlier, the contribution of older age groups might be smaller than that of young people.

Methods and Data

Methods

Lifespan variation can be measured using several indices, which are highly correlated in empirical datasets when measured from young ages (Colchero et al. 2016, Wilmoth and Horiuchi 1999). They are not, however, interchangeable as they differ in their formal properties and in the underlying concept they measure (van Raalte and Caswell 2013). For this reason, we use six measures of lifespan variation: the standard deviation at birth S_0 and the coefficient of variation CV; lifespan disparity $e^\dagger$ (Vaupel and Canudas-Romo 2003) and lifetable entropy $\bar{H}$ (Keyfitz 1977; Leser 1955); the relative and absolute Gini coefficients, G_0 and G_0^{abs} respectively (Hanada 1983; Shkolnikov et al. 2003). Each pair comprises an absolute measure and its relative counterpart. For example, lifespan disparity can be expressed as:

$$e^\dagger = \int_0^\omega d(x)e(x)dx \tag{1}$$

while its relative counterpart lifetable entropy is

$$\bar{H} = \frac{\int_0^\omega d(x)e(x)dx}{e_0} = \frac{e^\dagger}{e_0} \tag{2}$$

Where ω is the highest age in the population, $d(x)$ is the deaths distribution

and $e(x) = \frac{1}{l(x)} \int_x^\omega l(a)da$ life expectancy at age x. e_0 represents life expectancy at birth, which equals $\int_0^\omega l(a)da$, for $l(0) = 1$.

In a similar way, the standard deviation is

$$S_0 = \sqrt{\int_0^\omega (x - e_0)^2 d(x)dx} \tag{3}$$

and the coefficient of variation

$$CV = \frac{\sqrt{\int_0^\omega (x - e_0)^2 d(x)dx}}{e_0} = \frac{S_0}{e_0} \tag{4}$$

Finally, the absolute Gini coefficient is

$$G_0^{abs} = e_0 - \frac{1}{l(0)^2} \int_0^\omega [l(x)]^2 dx \tag{5}$$

and, for a lifetable radix $l(0) = 1$, its relative counterpart becomes

$$G_0 = \frac{e_0 - \frac{1}{[l_0]^2} \int_0^\omega [l_x]^2 dx}{e_0} = 1 - \frac{1}{e_0} \int_0^\omega [l(x)]^2 dx = \frac{G_0^{abs}}{e_0} \tag{6}$$

where $l(x)$ is the survival function.

S_0 is the square root of the variance, which is the average squared distance from the mean age at death. Lifespan disparity can be interpreted as the average years lost at death and the absolute Gini coefficient is the average distance between any pair of individuals' ages at death (Shkolnikov et al. 2003). Relative measures express their absolute counterparts as a proportion of life expectancy (e.g., the relative Gini coefficient is the average distance between each individual's age at death relative to e_0). $\bar{H}$ can also be interpreted as a measure of the elasticity of life expectancy to a proportional change in mortality (Keyfitz and Golini 1975; Leser 1955). While measures of absolute variation are expressed in years, relative measures are dimensionless.

We use multiple measures for three reasons. First of all, by comparing outcomes we can make more robust conclusions. Secondly, relative and absolute measures of variation each have their own advantages. Relative measures are the basis of the pace and shape framework, proposed by Baudisch (2011). By distinguishing the pace (the scale on which mortality progresses) and the shape (how rates change with age) as two separate dimensions (Wrycza et al. 2015), this approach allows comparisons between populations with different lengths of life (Jones et al. 2014), e.g., in normal vs crisis times. Absolute measures of lifespan variation are inextricably linked to the length of life and, therefore, easier to interpret. Moreover, some research has suggested that a mix between relative and absolute measures of variation could better describe the idea of equality in some disciplines (Asada 2010), so that both kinds of measure should be considered in research. Finally, by using both sets of measures, we can observe the differences in their behaviours under such extreme mortality circumstances.

We further decompose by age the changes over time of each measure of lifespan variation, using the linear integral method. Horiuchi et al. (2008) show that a change in a continuous function can be expressed discretely as:

$$y_2 - y_1 = \sum_{i=1}^{n} c_i$$

In our case, y indicates lifespan variation and y_1 and y_2 are its values at times t_1 and t_2, respectively. $c_i = \int_{x_{i1}}^{x_{i2}} \frac{\partial y}{\partial x_i 1} dx_i$ represents the age-specific contributions to the change in lifespan variation, with x_i being the death rate specific to age i; while x_{i1} and x_{i2} indicate the value of this death rate at times t_1 and t_2, respectively. Using this method, we aim to understand which age groups contributed most to the change in lifespan variation. Although other methods of decomposition have been developed and applied to measures of lifespan variation (see for example Appendix B of Wilmoth and Horiuchi (1999)), the Horiuchi method assumes that the covariates in a function change continuously, which is particularly appropriate when studying changes across time.

Data

We use two data sources. The first is the Human Mortality Database (HMD, Barbieri et al. 2015). It gathers mortality data by age and sex, for multiple populations and periods, collected from each country's statistical office. Only countries with virtually complete death registration and censuses are included in the database. Among these, Scandinavian countries offer excellent examples of mortality crises.

We use data for Iceland and Sweden, which have been collected by parishes since the eighteenth century (Andreeva and Dukhonov 2020; Glei et al. 2019). The raw data were corrected, but a number of issues remain, such as age heaping and age exaggeration. Moreover, in Sweden out-migration was not recorded, which could bias our analyses if the crises encouraged migration. This should not be an issue for Iceland, because of its isolation and the rapidity of the epidemics. At the same time, the Icelandic population is so small (a little under 60,000 in the 1840s (Tomasson 1977)), that it is likely to show a fair deal of statistical noise. Death rates at older ages were smoothed to exclude excessive fluctuations and to disaggregate open age intervals. These procedures caused some implausible patterns for these categories. Therefore, we reduce the number of age categories by creating an open-ended class at age 80, instead of 110. We also use abridged period lifetables to lessen the effect of digit preference. The other issues are more difficult to tackle and should be kept in mind when interpreting results.

The second dataset was compiled in the early 2000s by Meslé and Vallin (INED), containing single-age period lifetables covering the Ukrainian population from 1926 to 1959 (later extended to the early 2000s), by sex and age. Because of the limited data collection in Ukraine during this period, the lifetables in this dataset were imputed from various sources using forward and backward projections. The relevant metadata have been published in a number of articles and books (e.g., Meslé and Vallin 2012 or Vallin et al. 2002). These data are likely to underreport actual deaths, especially during the crisis years. Moreover, Meslé and Vallin assume zero net voluntary migration, taking into account the restrictions imposed on travel during this period in the USSR. Although this dataset clearly presents some quality issues, it is still considered the best available source of information for studying Ukraine during the 1933 famine (Zarulli et al. 2018), which provides a useful counterexample to the Scandinavian mortality crises. In order to increase comparability, we grouped these data in 5-year age categories and set the open-ended group at age 80, instead of 89.

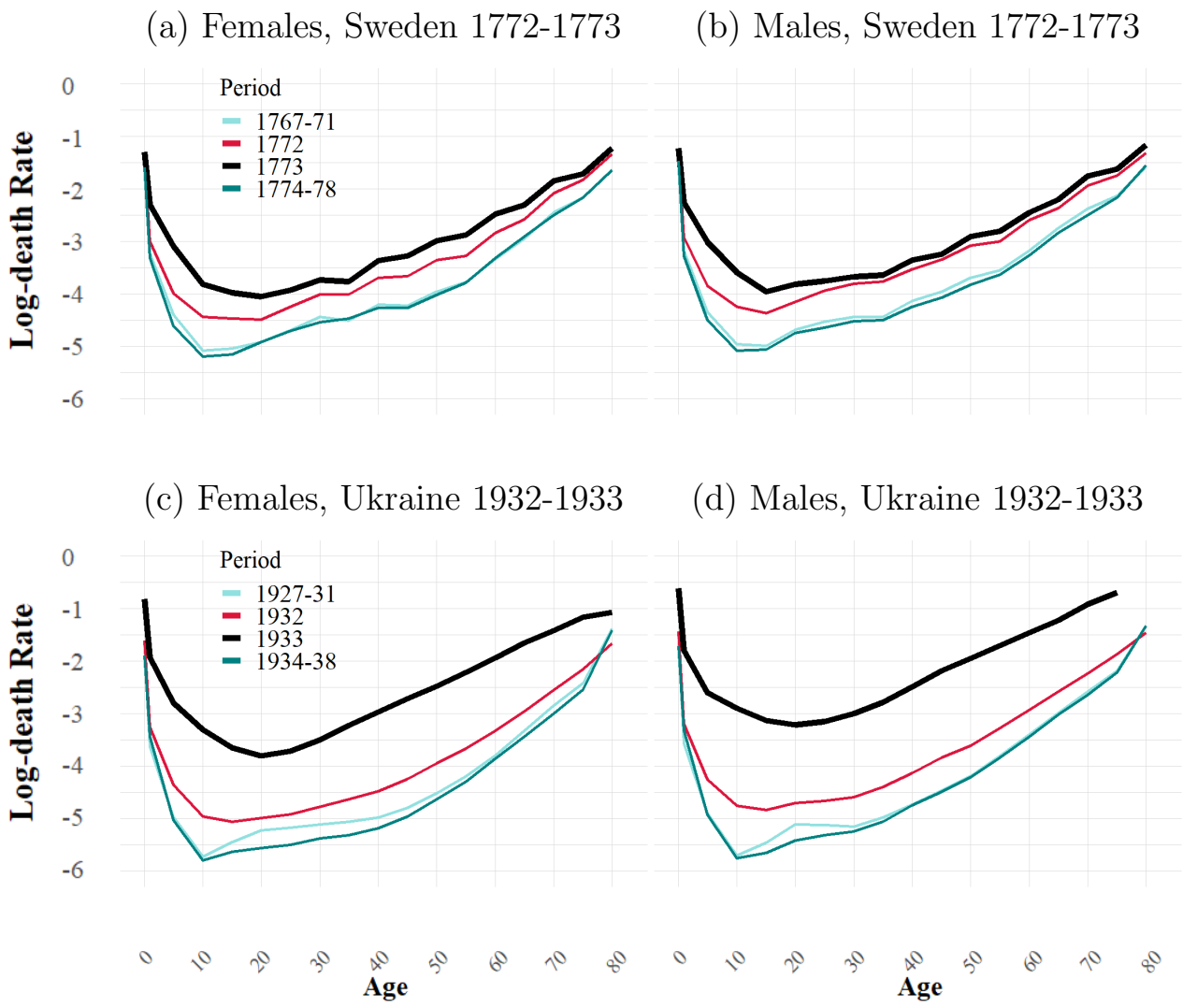


Figure 1: Log-death rates, famines. Source: HMD (Sweden) and Meslé & Vallin

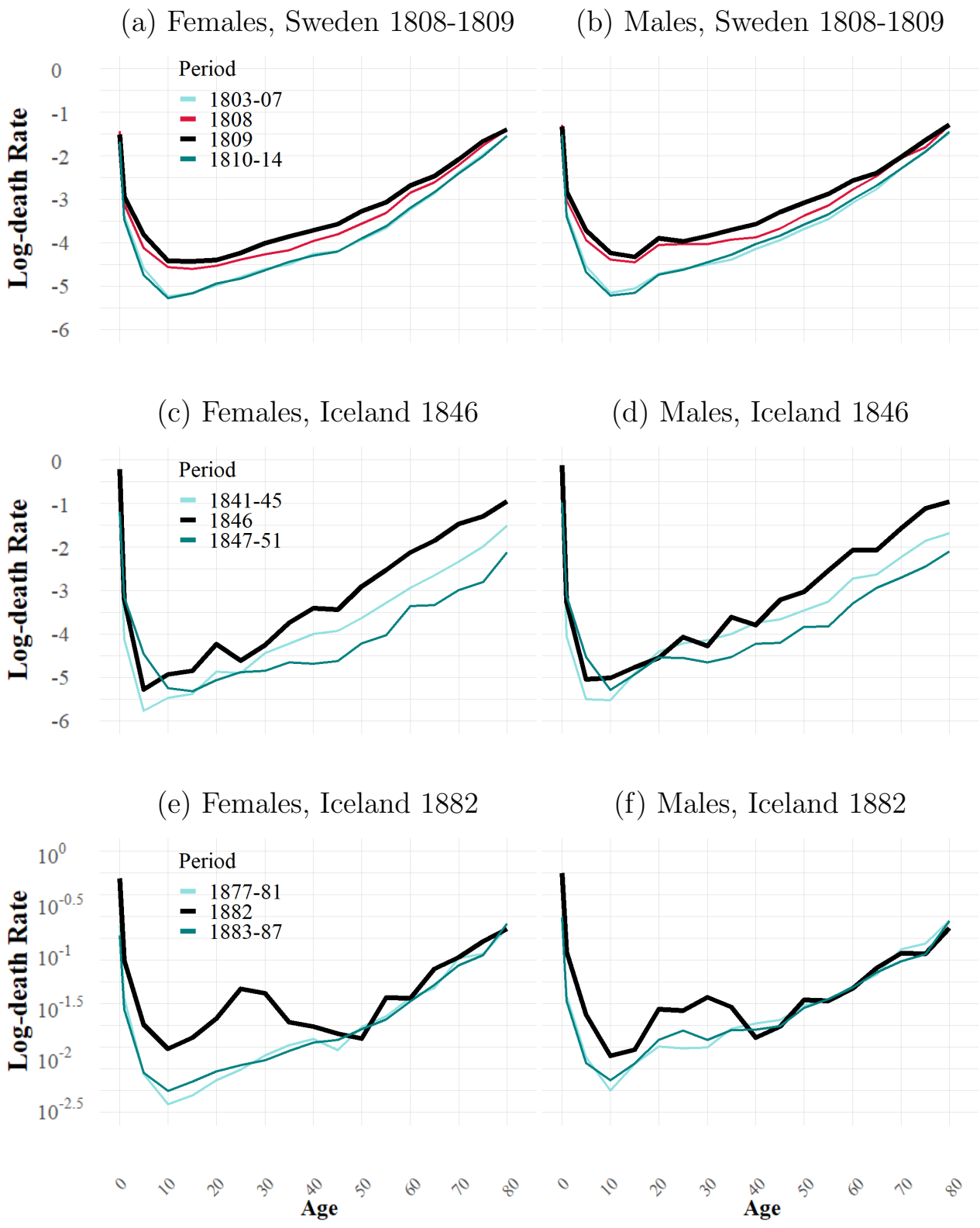


Figure 2: Log-death rates, epidemics. Source: HMD (Sweden and Iceland)

Results

Age Patterns of Mortality

Figures 1 and 2 show male and female log-death rates for each case study. In each panel, we plot the crisis year(s) and the averages of the five years before and after the crisis. (Appendix Fig. B1 and B2 show the absolute difference in log-death rates between the (main) crisis year and the other periods for all five crises).

Despite some differences in magnitude and in the temporal dynamics of famines (Fig. 1), there are many parallels. In both cases, the increase in mortality is smaller at age 0 and there is evidence of convergence at the oldest ages, for both sexes in Sweden and for females in Ukraine (as the survival function for Ukrainian males in 1933 reaches 0 at age 75, the rates stop short of age 80). Sex differentials also behave similarly: not only are male rates always higher, but they also increase more.

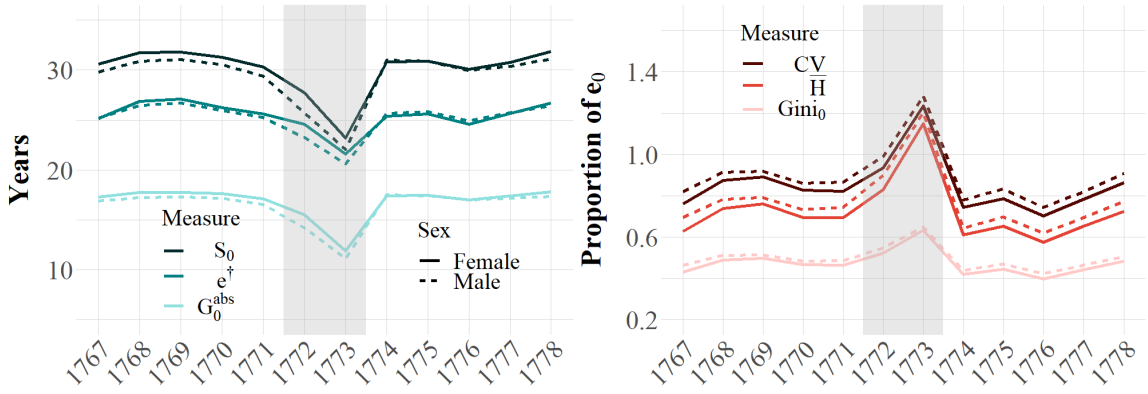
Figure 2 shows log-death rates for the three epidemics. Contrary to what happened for famines, each epidemic presents a distinct mortality pattern. During the 1809 typhus and dysentery epidemic, infant mortality was not affected. Male mortality is higher for all periods, but even more so in 1808-1809 for ages 15 to 40. The two Icelandic measles epidemics show significant variability, as can be expected from a smaller population. Thus, patterns should be considered with care, especially when they are seemingly idiosyncratic. During the 1846 epidemic, there was a greater increase in mortality among children, and after age 45, particularly for males, with no sign of convergence for older ages. However, after the crisis, adult mortality dropped to below pre-crisis levels, while child mortality was even higher than during the crisis itself. Finally, female mortality during the crisis is higher than male mortality, although during non-crisis years the opposite is true. The 1882 epidemic also affected females more than males. There is also a greater increase in infant and childhood rates compared to 1846 and a clear spike for young adults.

Trends of Lifespan Variation

In Fig. 3 and 4, we examine how absolute and relative lifespan variation measures behave (note that we use single years rather than 5-year averages for pre-crisis and

(a) Absolute measures, Sweden 1772-1773

(b) Relative measures, Sweden 1772-1773



(c) Absolute measures, Ukraine 1932-1933

(d) Relative measures, Ukraine 1932-1933

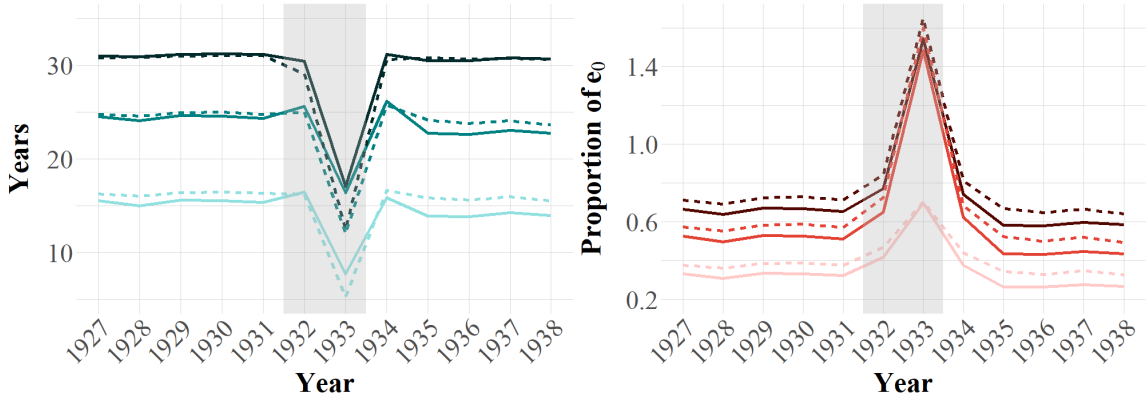


Figure 3: Lifespan variation, famines. S_0 indicates standard deviation, $e^\dagger$ indicates lifespan disparity and G_0^{abs} indicates the absolute Gini coefficient. CV indicates the coefficient of variation, $\bar{H}$ indicates lifetable entropy and G_0 indicates the relative Gini coefficient. Source: HMD (Sweden) and Meslé & Vallin

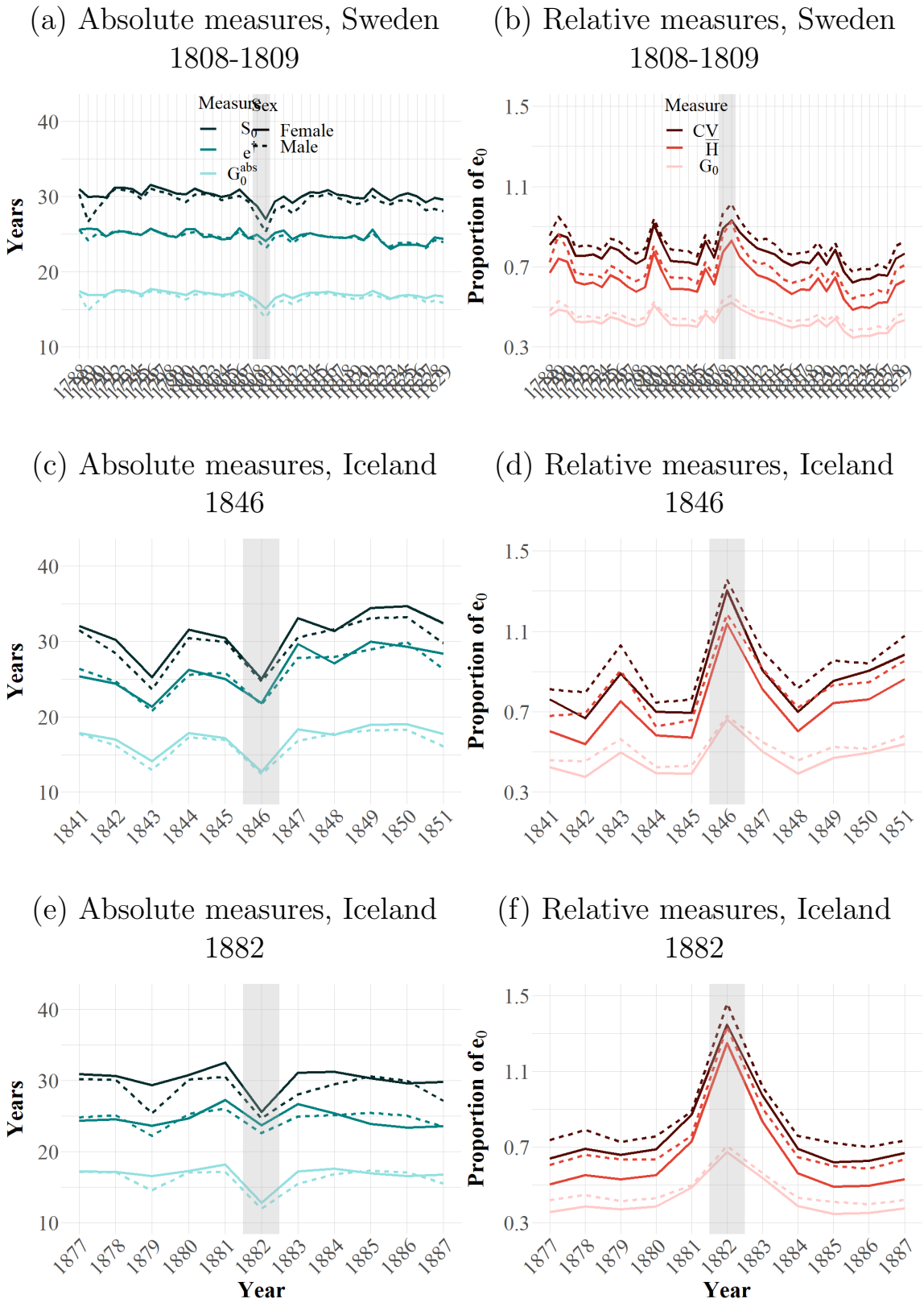


Figure 4: Lifespan variation, epidemics. S_0 indicates standard deviation, $e^\dagger$ indicates lifespan disparity and G_0^{abs} indicates the absolute Gini coefficient. CV indicates the coefficient of variation, $\bar{H}$ indicates lifetable entropy and G_0 indicates the relative Gini coefficient. Source: HMD (Sweden and Iceland)

post-crisis periods). A striking finding is that absolute and relative variations present contrasting dynamics: the former decreases during the crisis, while the latter goes up. Another difference is that absolute variation is usually higher for females than for males, whereas the opposite is true for relative measures. Male absolute variation also changes more during a crisis, but not relative variation. Both kinds of measures, however, show that the change in lifespan variation typically only lasts during the crisis itself, with little effect during the following years. This mirrors the trend of life expectancy, which also returns to pre-crisis levels right away; two notable exceptions being the 1809 Swedish epidemic, where e_0 increased slowly after the crisis, and the 1846 Iceland epidemic, where e_0 returned to its pre-crisis level for one year, but declined again soon after (see Appendix, Fig. C1).

We also find differences within each group of measures. The two Gini coefficients present the flatter curves, with smaller absolute changes. However, the lower proportional increase in its relative counterpart G_0 shows that G_0^{abs} is more sensitive in proportional terms to the changes in mortality. At the opposite end, we have $e^\dagger$, as shown by the greater proportional increase of $\bar{H}$ in all five cases.

Absolute variation starts decreasing right away in two of the three crises spanning two years. In the Ukraine, there is a noticeable drop only in 1933, with the small exception of male S_0. However, relative variation already starts rising (albeit slightly) in 1932, further underscoring the different behaviours of these measures. The two Icelandic epidemics also display interesting patterns. In 1843 for both sexes, and in 1879 for males only, absolute variation drops to the same levels as during the epidemic itself. This drop, however, was not accompanied by a similar decrease in e_0, meaning that the increase in relative variation is much less in 1843 and non-existent in 1879. Notwithstanding this, the same partly unexpected trend holds true for all five crises: absolute lifespan variation declines, while relative variation increases.

Decomposition of Lifespan Variation

Figures 5 and 6 show the results of the decompositions for males and females. We report results for two measures, $e^\dagger$ and $\bar{H}$, but the same main trends appear for the others, as shown in Appendix, Fig.E1 to E4. Each column indicates how the change in mortality experienced by the corresponding age group contributed to the overall change in the indicator. For example, the change in Swedish female infant mortality between the period 1767-1771 and the beginning of the famine in 1772 contributed

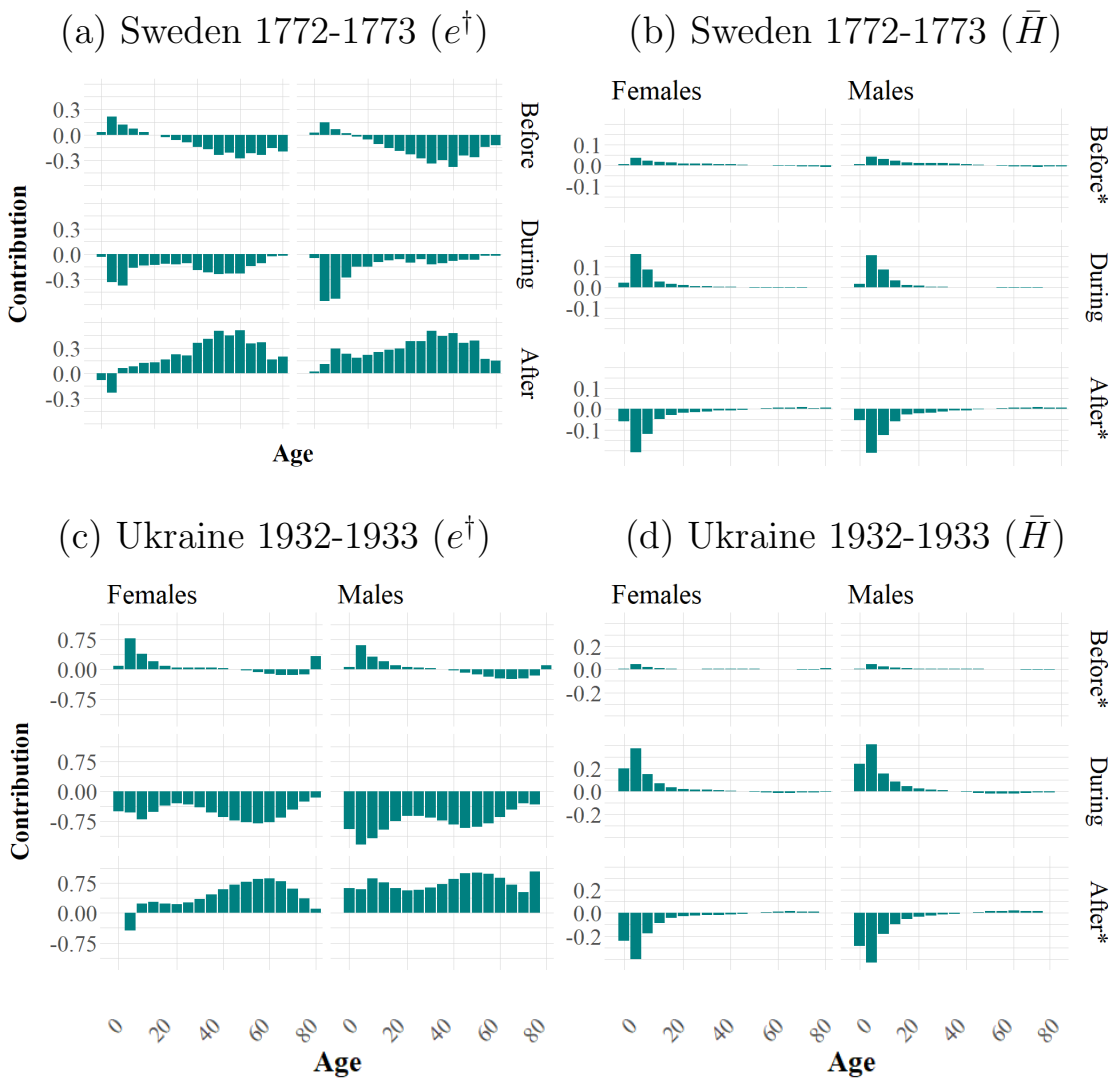


Figure 5: Lifespan variation decomposition, famines. * Before = average of previous 5 years , After = average of following 5 years. $e^\dagger$ indicates lifespan disparity, $\bar{H}$ indicates lifetable entropy. Source: HMD (Sweden) and Meslé & Vallin

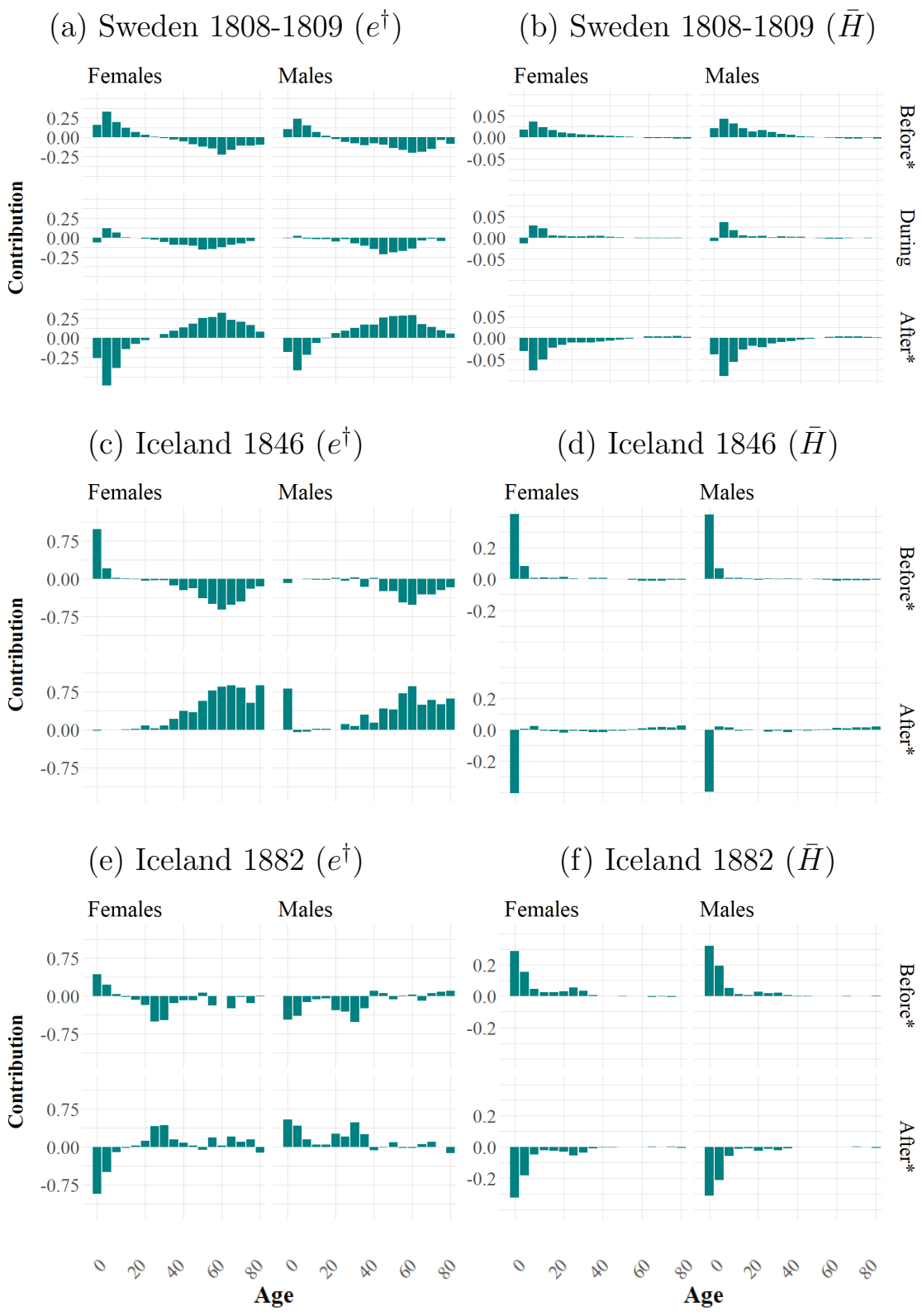


Figure 6: Lifespan variation decomposition, epidemics. * Before = average of previous 5 years , After = average of following 5 years. $e^\dagger$ indicates lifespan disparity, $\bar{H}$ indicates lifetable entropy. Source: HMD (Sweden and Iceland)

+0.034 years to the change in $e^{\dagger}$. Each age can contribute positively or negatively and the overall change is calculated by summing all contributions.

For the crises that lasted two years, we analyse three changes: between the average of the five years before the crisis and the first crisis year; between the two crisis years; and between the second crisis year and the average of the following five years. For the two Icelandic epidemics, we only look at the difference between the epidemic year and the averages of the previous and following five years. To ensure readability, the graphs have different scales.

Older ages have very little influence on the change in $\bar{H}$, even when they contribute significantly to the change in $e^{\dagger}$. Sex differences are more accentuated for the latter compared to the former, especially at young ages and for the Icelandic crises. While the uneven effect of measles can partly explain the exacerbated sex differences for Iceland, it cannot do so fully. Still, there are some common trends. The three nutrition-related crises (the two famines, plus the 1809 epidemic) saw small contributions of infants to the change in both $e^{\dagger}$ and $\bar{H}$, especially when compared to older children. Contributions to the change in $e^{\dagger}$ also decline after about age 60. The decomposition results for the Icelandic epidemics mirror age-specific patterns: significant contributions of infants (and older adults) in 1846, and of infants (and younger adults) in 1882.

All crises, apart from the Swedish famine, present ages which do not contribute to the change in lifespan variation in the expected direction. Usually, this is due to unexpected improvements in or aggravation of mortality from one period to the next (see Fig. 1 and 2). Table D1 shows the values of e_0 and of the threshold ages for $e^{\dagger}$ and $\bar{H}$.

Limitations

The data quality is not necessarily ensured. The sources of the HMD's death and population estimates do not record migrants. However, migrants are likely to have been healthier than those who stayed behind (Helgesson et al. 2019), so this recording issue would probably not influence our results to a significant extent. The Ukrainian data pose some specific issues. Namely, deaths are likely to have been underreported. This is especially true for infant deaths, which may in fact have been underreported in both Sweden and Iceland (Zarulli et al. 2018). Higher infant mortality would

concentrate deaths, thus accentuating the drop in absolute variation, but would also decrease e_0, so that relative variation might still increase. We addressed these issues through sensitivity analyses, changing the death rate by $\pm 10\%$ for every age and for infants only. We also changed the open-ended category in the lifetables between ages 70 and 90. Finally, we ran further sensitivity checks on a different dataset related to the Ukrainian famine, graciously provided by professor Wolowyna (Rudnytskyi et al. 2015). All of these confirmed our main results. We also tested sensitivity to the length of the non-crisis periods, comparing crisis years with 10, 15 and 20-year periods before and after each crisis[1]. These tests did not change our main results either.

Even when of good quality, the data used do not provide information on, e.g., social status or geographic location, which could have helped refine the results (Zoraster 2010). Moreover, although we find patterns for historic populations, they are not necessarily extendable to modern populations that are characterised by a different age profile of mortality, nor to the modern world, where easier migration and international aid could modify the mechanisms at play, nor to modern mortality crises, such as the current COVID-19 pandemic, which are generally milder than the ones we analysed here. Our results can nevertheless act as a baseline for future analyses that look at lifespan variation during contemporary crises, to understand if a regular pattern emerges, which transcends time. In fact, Aburto et al. (2021) have recently found that in 2020 absolute variation has gone down together with life expectancy, because of the greater toll COVID-19 has taken on the elderly, suggesting more recent periods of mortality increase may indeed present similar patterns of lifespan variation. Methodologically, the translation of a continuous change into a discrete framework necessary for the decomposition analysis leads to some imprecision, which can be exacerbated by extreme mortality change (as might be the case for the Icelandic crises).

Discussion

We have analysed five mortality crises – two famines and three epidemics – that occurred in three countries and spanning 160 years. Each case presented a pattern of mortality change through the crisis, which can be linked to its specific context.

[1] We could not run these tests for the first Iceland epidemic and for the Ukrainian famine, as our data do not span enough years.

Despite these differences, each crisis was accompanied by a drop in absolute lifespan variation and a spike in relative variation. Decomposing by age showed sex differences, especially at younger ages, and a different effect of mortality change at young and older ages between absolute and relative measures.

Mortality Patterns

The five mortality crises we study were characterised by extreme and sudden drops in life expectancy at birth. In similar contexts, Zarulli (2013) found that mortality increases proportionally at adult ages, with convergence for older individuals when a crisis lasts more than one year. We observe comparable trends in our five cases. Even for the short Icelandic epidemic of 1846, there is evidence of a potential mortality displacement at adult ages, translating into a lower-than-pre-crisis mortality following the crisis itself. Some trends deviate from this model, but they are mostly attributable to the context in which the crisis happened, and to its characteristics. The lower increase of infant mortality for the two Swedish and the Ukrainian crises is consistent with the protective effect of breastfeeding against nutrition deficiency and nutrition-related diseases (Livi-Bacci 1990). Bengtsson et al. (2009) argue that in the past, children – as net consumers of resources – might have become expendable in times of crisis. As this attitude changes across time and space, it could clarify the disproportionate increase in child mortality in the Swedish famine, which did not happen in the Ukrainian famine. Moreover, the Ukrainian data have been extensively manipulated and smoothed, and might be masking a similar increase for children. In the Ukrainian case, death rates also increase proportionally less for young adults than for older ones, especially for females. This could be due to a greater resilience of these ages in the circumstances of the Ukrainian famine or, again, to the manipulation of the data.

The peculiar pattern of male rates for the 1808-1809 Swedish epidemic can be tied to the war context. Since the epidemic spread *via* troop movements, soldiers may have been more exposed. This also accounts for the different patterns in 1808 and 1809. In the first year, the increase in male death rates is much lower after age 40, while female mortality increases uniformly. Before the epidemic spread to the whole population, it is possible that males not directly involved in the war could have been less exposed than females, who took care of the sick, although more research into the practices of the time would be needed to verify this hypothesis.

The sex-specific patterns of the Icelandic epidemics are consistent with the literature, showing a greater vulnerability of females to measles (Garenne 2015; Muenchhoff and Goulder 2014). In 1846, the increase in rates also follows age-specific vulnerability to measles, while the surprising increase in child mortality *after* the epidemic could be tied to the lasting effects of exposure to infectious diseases in early childhood (Fridlizius 1989; Stoermer 2011), although a cohort study could better discriminate whether this is a stable pattern. The spike in mortality experienced by young adults in 1882 remains unexplained. It is unclear why these individuals should have disproportionally suffered from a, typically, childhood disease and which showed no such pattern just 40 years before. It is unlikely that such a definite trend would be due to sheer variation and the explanation is rather to be found in a specific behaviour that exposes this age group.

Lifespan Variation Trends

Period lifespan variation gives us insight into the distribution of mortality in a given year. The rise in relative measures indicates that mortality crises negatively affect both e_0 and variation, but the drop of absolute measures nuances this interpretation. By shortening lifespans for everybody, the crises we have studied seem to have compressed the mortality distribution. Thus, ages at death would have been more concentrated in absolute terms. However, deaths increased at the extremes of this compressed distribution: fewer years separated individual ages at death, but they represented more of the average lifespan. Our results add to the literature on lifespan variation that shows that the relationship between life expectancy and lifespan variation – which is typically found to be negative (Colchero et al. 2016; Sasson 2016; Smits and Monden 2009; van Raalte et al. 2011) – is not so straightforward (Edwards and Tuljapurkar 2005; Permanyer et al. 2018; van Raalte et al. 2014). What this also suggests is that even a generalised increase in mortality affects individuals differently. This could be due to biological differences, as Zarulli et al. (2018) suggest in relation to the sex gap, to a societal structure, or to some other factor.

Despite the massive effect that our five crises had on mortality, we cannot see noticeable mid-term consequences. Not only does e_0 go back to pre-crises levels right away, but so does lifespan variation. In this sense, Fig. 3 and 4 are a testament to the resilience of populations. However, as Goldstein and Lee (2020) point out, period measures may overstate the impact of a short-lived mortality crisis, because they "implicitly [assume] that the epidemic is experienced each year over and over

again" (Goldstein and Lee, 2020, 22037). Despite this limitation, because lifetable measures are standardised for the structure of a population and they express the underlying risks of mortality, they make it possible to meaningfully compare the experiences of different populations in this area.

Sex Differences

Figures 3 and 4 show that absolute variation tends to decrease less for females than for males. Thus, male mortality is more concentrated. However, since e_0 also decreases more for males than for females, the increase in relative variation is more similar across sexes. Previous research has found that males are physiologically more vulnerable during infancy and early childhood (Drevenstedt et al. 2008; Pongou 2013; Department of Economic and Social Affairs, Population Division 2011). We also find that the difference in absolute variation trends is mostly due to differences in the weight of young ages: before the crisis, young males contribute less than young females to the change in $e^\dagger$, thus compensating less for the influence of older ages, while they contribute more in the other periods. Although the contribution of child mortality to the change in $\bar{H}$ is not identical across the sexes, the differences are less pronounced. An exception to these trends is represented by the two Icelandic epidemics, where lifespan variation changed either equally, or more for females. These differences again seem to be mostly explained by differences in the contribution of young ages. Thus, young ages determine existing sex differences in lifespan variation change.

Methodological Implications

Our analyses also show that relative and absolute measures are not interchangeable. While at mid-range levels of e_0, the relationship with absolute variation is indeed negative, when e_0 drops to very low levels, absolute variation also starts to decrease (as shown in panel b of Appendix, Fig. A1). Permanyer and Shi (2020) find a similar plateau in absolute lifespan variation for very high levels of e_0. They link this plateau to a recent slowdown in e_0 gains, compared with gains in longevity; however, it is more difficult to adopt this framework in our case, where longevity is to a great extent determined by previous and drastically different mortality structures. Decomposition results give us some clarifying insight. As expected, the greatest single contributions to the change in $e^\dagger$ come from very young ages. However, it is actually

older ages that lead the direction of this change. Since the threshold age for $e^\dagger$ ($a^\dagger$) trails e_0 closely (Aburto et al. 2019), it was quite low in the periods we considered (see Appendix, Table D1). Thus, even at relatively low ages, mortality increases contribute to decreasing $e^\dagger$ and their sum can offset the positive contribution of younger ages. A similar mechanism explains the sudden increase of $e^\dagger$ after the crisis. Zhang and Vaupel (2009) have shown that when $\bar{H}$ surpasses 1, which consistently happens during our crisis years, $a^\dagger$ becomes 0. Because of this and because, after the crisis, mortality largely improves, all ages contribute to an increase in $e^\dagger$, bringing it back to pre-crisis levels.

On the other hand, relative variation meets our expectations. Panel a of Appendix, Fig. A1 shows a monotonic and negative relationship between $\bar{H}$ and e_0 throughout the age distribution. The definition of relative measures is key to understanding this trend. e_0 is more sensitive to the crisis than absolute variation (Appendix, Fig. C1). Because the denominator decreases more than the numerator, relative variation increases, even as its absolute counterpart drops. Decomposition results show a clear predominance of the contribution of very young ages over all the others. Decomposing the change in e_0 (Appendix, Fig. C2) shows that the contributions of young ages are greater than for the change in $e^\dagger$, while older ages contribute about the same to both measures. Here, again, a disproportionate change in the denominator is bound to accentuate the contribution of young ages, while at older ages the ratio remains constant. Moreover, the threshold age for $\bar{H}$ (a^h) is much more stable than $a^\dagger$ (Aburto et al. 2019), so that it rarely drops below age 30, even during these crises. Therefore, the proportion of the age distribution above and below the threshold is more balanced.

While changes in relative variation are led by mortality at young ages, as we had expected, changes in absolute variation are to a great extent determined by older ages – a difference that leads to their opposing behaviours at the overall level. These results underline the contrasts between absolute and relative variation measures, which are deeply influenced by e_0 trends and by the behaviour of the threshold age, and show that mortality at older ages can be crucial, even when infant mortality is high.

Although lifespan variation has thus far been calculated through absolute and relative indices, new measures can be developed. Permanyer and Shi (2020) propose a new set of normalised measures, which it would be interesting to test against the classic ones. Other measures of longevity could also be used, besides e_0, so as to better understand the relationship between length of life and its variation.

Policy Implications

Greater lifespan variation poses ethical dilemmas for the organisation of pension and health systems (Brønnum-Hansen et al. 2017) and can have specific implications for lifecycle investments and consumption, because individuals assess their chances of benefiting from such decisions in the future (Tuljapurkar 2011). Previous research has already called for greater public awareness of lifespan variation and its consequences, at both the individual and societal levels (van Raalte et al. 2018). Our results highlight that the choice of measure also matters. Not only can absolute and relative measures yield different results (not only for very low levels of e_0, but, as Permanyer and Shi (2020) have found, also for higher ones), but each expresses equality differently. For example, a decrease in mortality at older ages might increase absolute variation, but if it also lengthens e_0 enough, relative measures might register a decrease in variation. Thus, different measures might lead to opposing conclusions about the state of a population's health and to different distributions of resources to the welfare system or to programmes to fight inequalities. The introduction of lifespan variation in the public debate should be accompanied by a conscious reflection on the meaning of the tools we use to measure it, so as to mirror the preferred conception of equality.

Conclusion

Lifespan variation can illuminate some of the mechanisms of mortality crises. In our analyses, we have found a common trend for the crises examined: by increasing relative lifespan variation and decreasing absolute variation, mortality crises shorten all (period) lifespans, but to unequal extents. The effect of crises on lifespan variation also differs across sexes, which is mostly due to discrepancies in the contribution of young ages. These results reflect the literature that has questioned the straightforward inverse relationship between e_0 and lifespan variation. Moreover, they underline the importance of older age mortality even in contexts with very high infant mortality. The reoccurrence of this pattern throughout our five cases suggests that it might exemplify the behaviour of human mortality under extreme conditions. However, the crises we have used cannot hope to encompass the whole temporal and geographical extent of human experience. More analyses should be carried out to look at different kinds of crises. Longer and milder crises could present an interesting mid-way case study and could better show long-term effects of adverse conditions, as could analyses

on the cohort dimension. Studying modern crises would show whether demographic, social and geo-political changes have affected lifespan variation trends and could provide data for more detailed analyses. By comparing different kinds of measures, we also hope to have contributed to the understanding of their characteristics and of their behaviours, so as to add to the debate around the use of absolute or relative measures of variation – although here more research is also needed.

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Appendix A

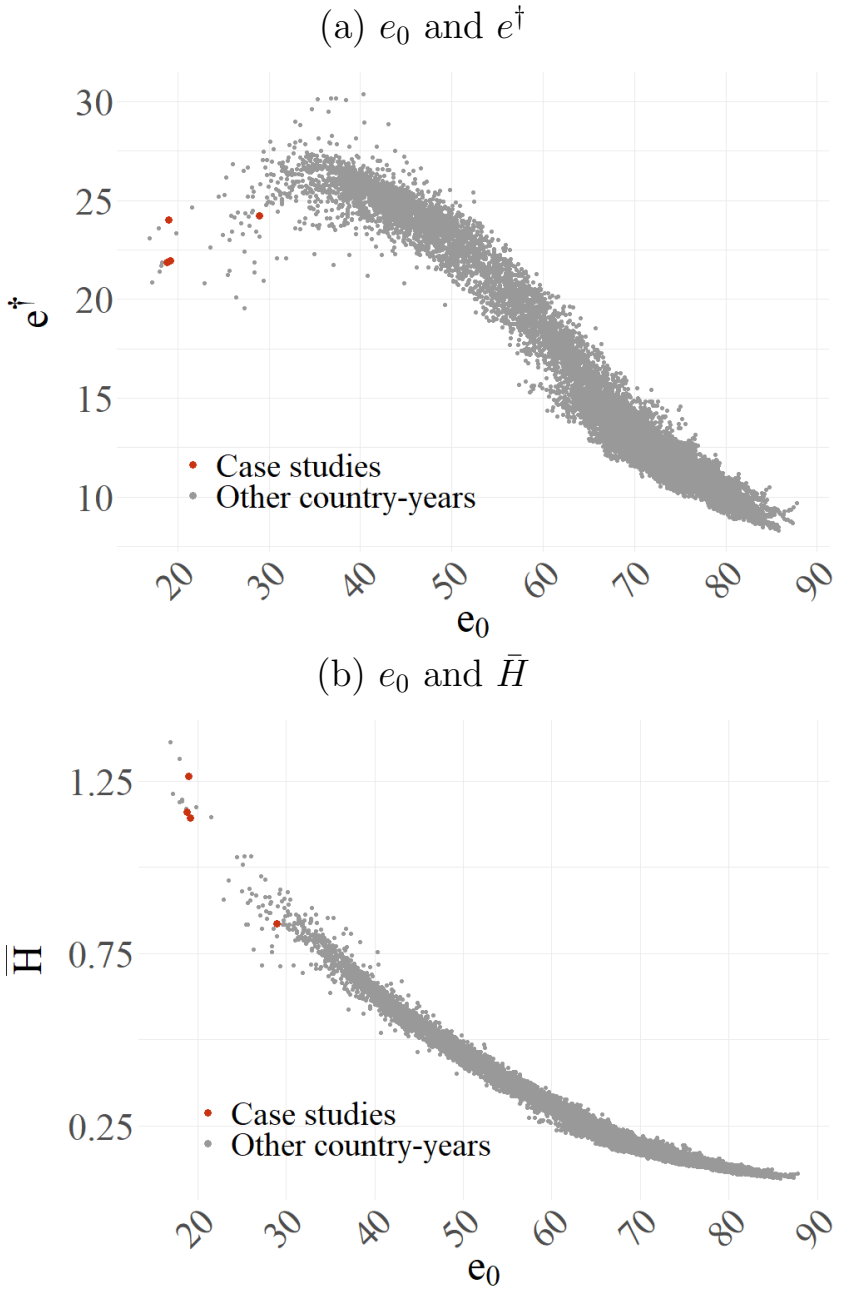

Figure A1: Life expectancy and lifespan variation. $e^\dagger$ indicates lifespan disparity, $\bar{H}$ indicates lifetable entropy and e_0 indicates life expectnacy at birth. Source: HMD, all countries

Appendix B

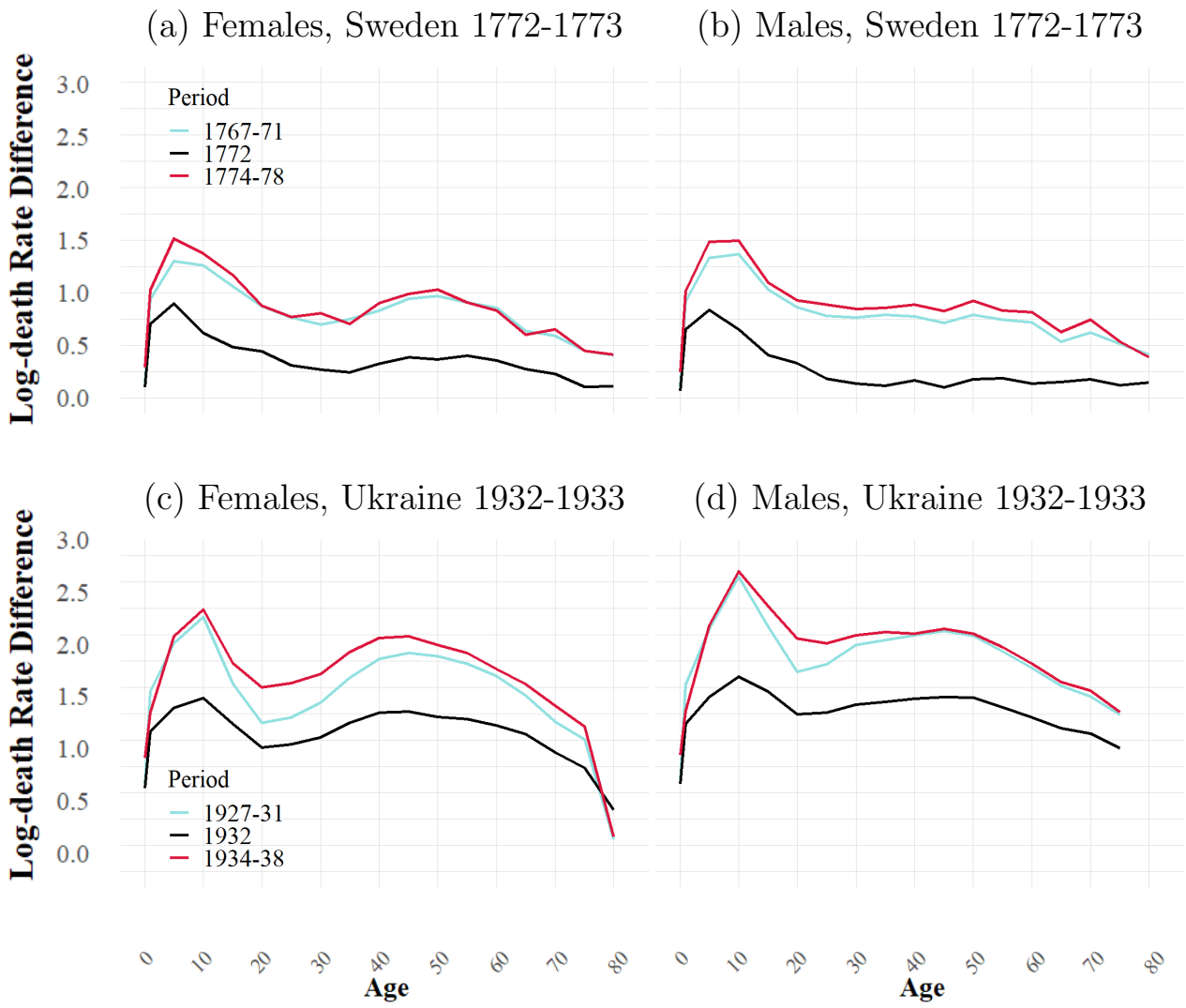

Figure B1: Absolute differences in log-death rates with main crisis year, famines. Source: HMD (Sweden) and Meslé & Vallin

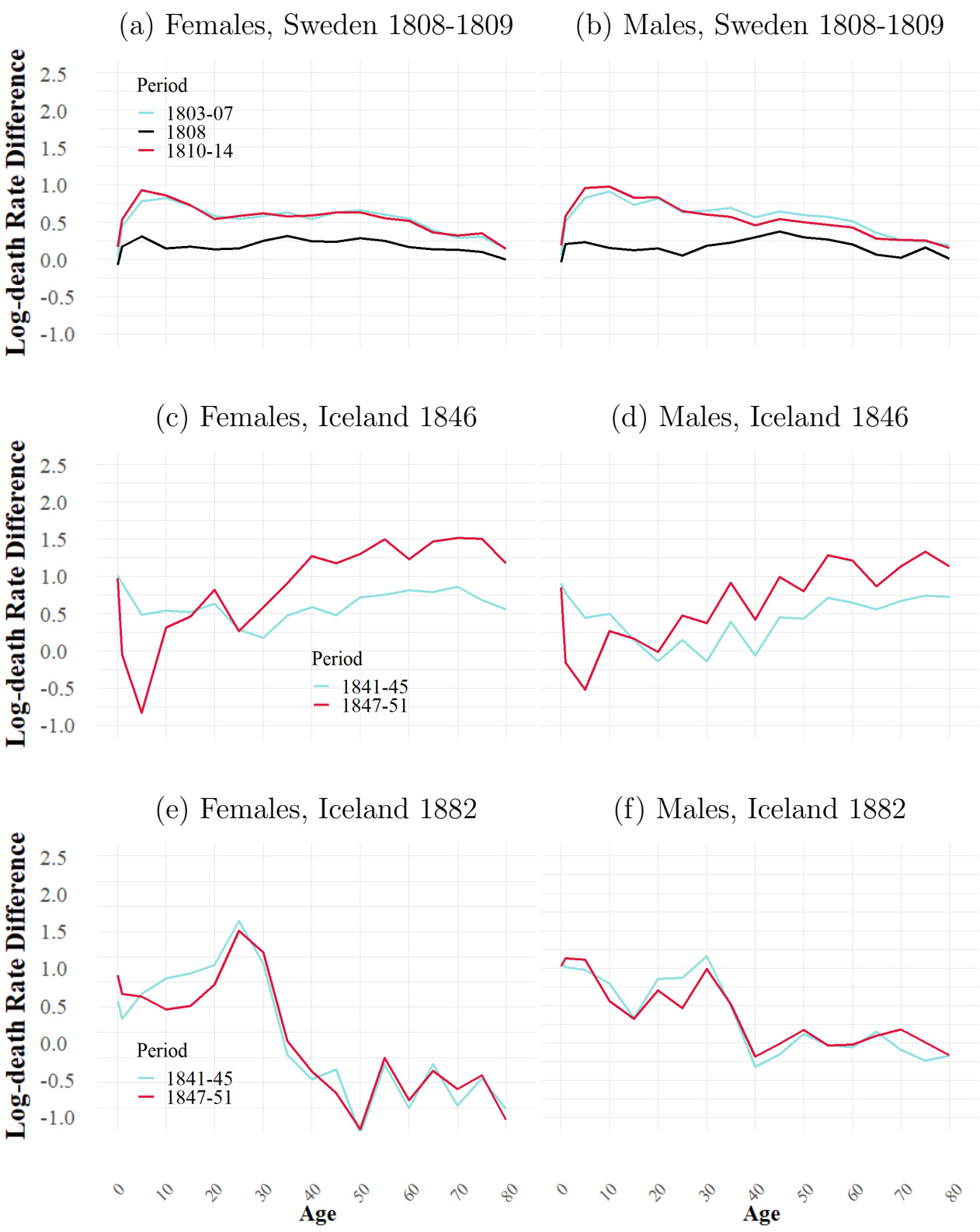

Figure B2: Absolute differences in log-death rates with main crisis year, epidemics. Source: HMD (Sweden and Iceland)

Appendix C

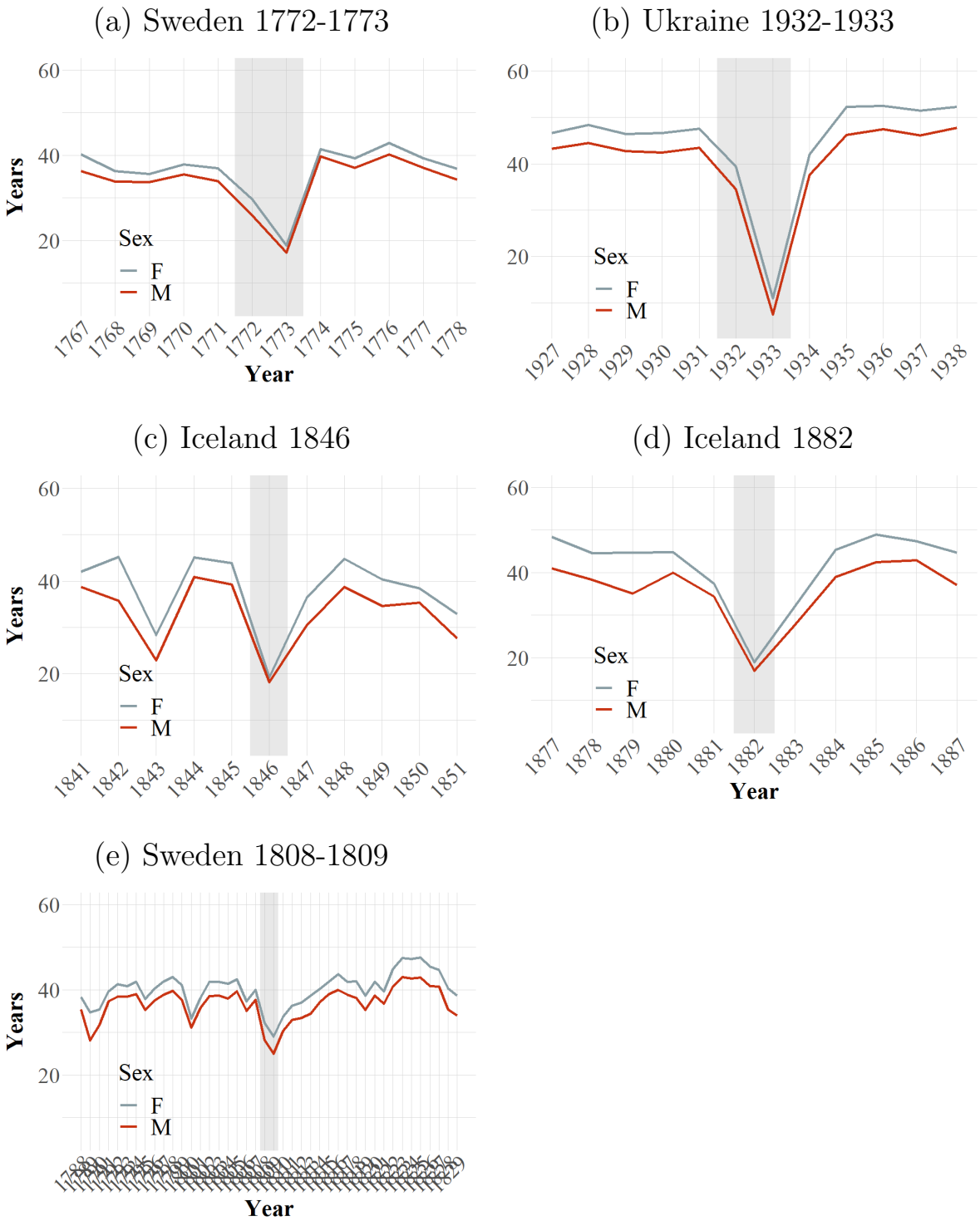

Figure C1: Trends of life expectancy. Source: HMD (Sweden and Iceland) and Meslé & Vallin

Figure C2: Decomposition of e_0. * Before = average of previous 5 years , After = average of following 5 years. Source: HMD (Sweden and Iceland) and Meslé &

Appendix D

Table D1: e_0 and threshold ages for $e^\dagger$ and $\bar{H}$

Measure	**Sweden 1772-1773**								
	Females					*Males*			
	Before	Pre-crisis	Crisis	After		Before	Pre-crisis	Crisis	After
e_0	37.31	29.65	18.83	39.85	34.61	25.91	17.18	37.59	
$a^\dagger$	19.59	8.75	0	24.16	15.97	3.2	0	20.83	
a^h	52.91	48	39.05	53	51.34	43.47	36.32	52.07	
	Ukraine 1932-1933								
	Females					*Males*			
	Before	Pre-crisis	Crisis	After		Before	Pre-crisis	Crisis	After
e_0	47.31	39.44	10.85	50.11	43.3	34.46	7.3	44.82	
$a^\dagger$	28.42	24.74	0	28.72	30.36	20.62	0	31.8	
a^h	53.86	53.86	25.92	53.49	30.36	20.62	12.24	31.8	
	Sweden 1808-1809								
	Females					*Males*			
	Before	Pre-crisis	Crisis	After		Before	Pre-crisis	Crisis	After
e_0	40.56	32.28	28.96	37.02	37.75	28.18	24.97	33.5	
$a^\dagger$	28.69	14.62	8.54	23.96	25.96	5.61	1.74	18.68	
a^h	53.45	50.67	47.19	52.7	52.38	47.42	42.84	50.13	
	Iceland 1846								
	Females					*Males*			
		Before	Crisis	After		Before	Crisis	After	
e_0		39.02	19.19	38.13	33.8	18.22	32.85		
$a^\dagger$		28.62	0	8.49	19.74	0	8.49		
a^h		50.94	38.43	52.39	48	36.43	51.05		
	Iceland 1882								
	Females					*Males*			
		Before	Crisis	After		Before	Crisis	After	
e_0		43.74	19.01	46.42	37.35	16.94	37.1		
$a^\dagger$		28.28	0	30.17	22.69	0	19.01		
a^h		52.82	38.1	52.33	50.23	35.06	48.96		

Source: HMD (Sweden and Iceland) and Meslé & Vallin

Appendix E

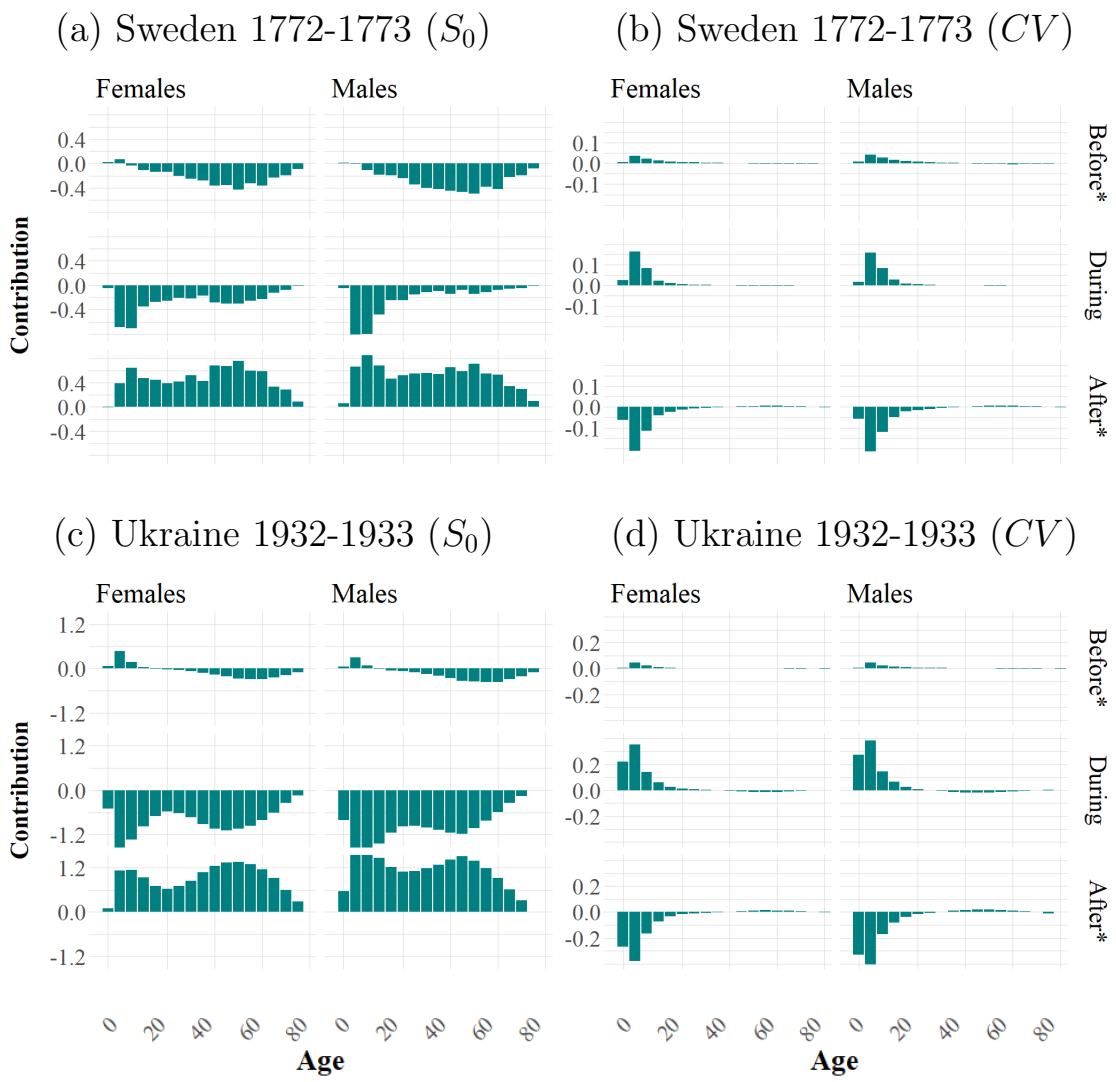

Figure E1: Standard deviation and coefficient of variation decomposition, famines. * Before = average of previous 5 years , After = average of following 5 years. S_0 indicates standard deviation, CV indicates the coefficient of variation. Source: HMD (Sweden) and Meslé & Vallin

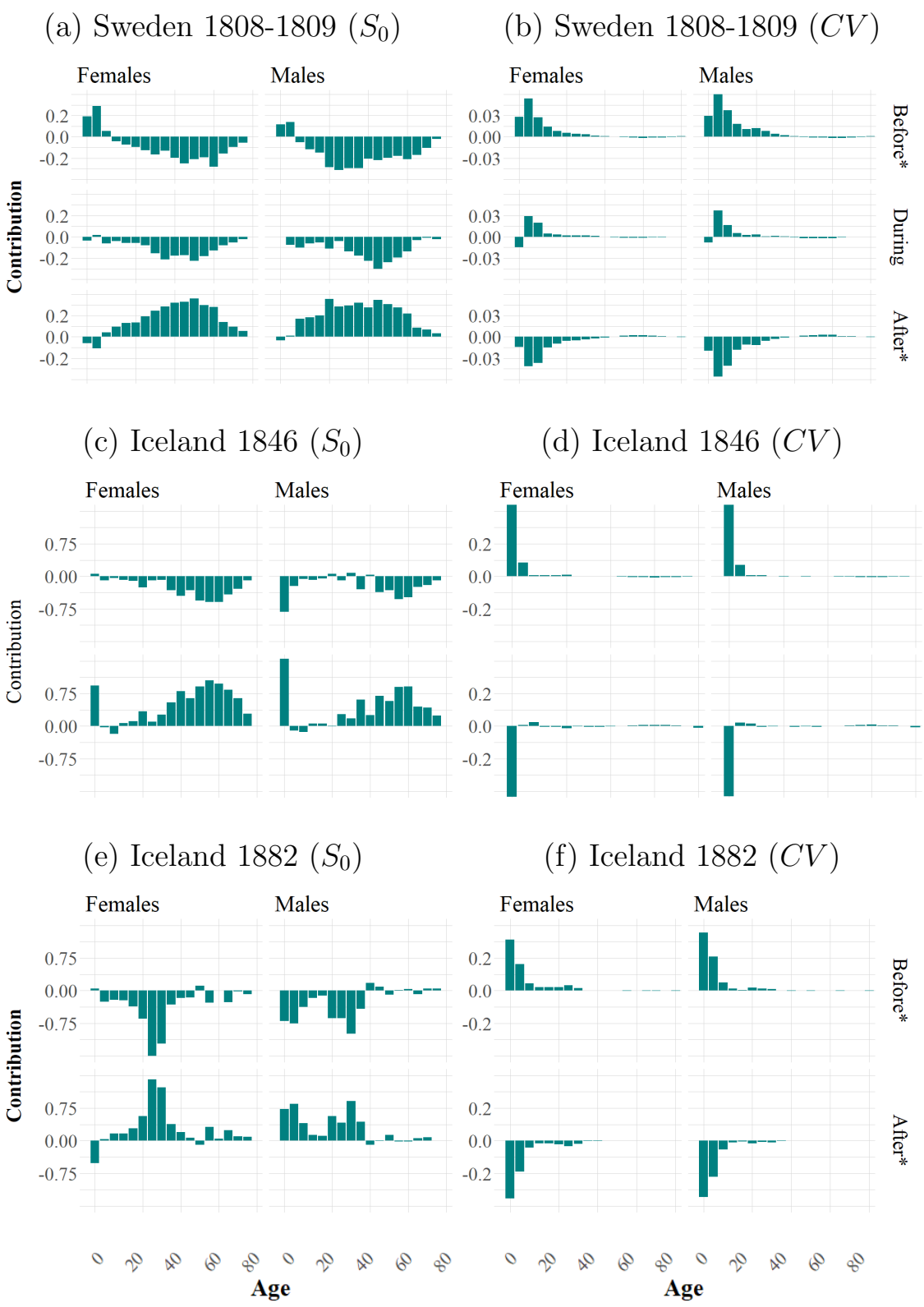

Figure E2: Standard deviation and coefficient of variation decomposition, epidemics. * Before = average of previous 5 years , After = average of following 5 years. S_0 indicates standard deviation, CV indicates the coefficient of variation. Source: HMD (Sweden and Iceland)

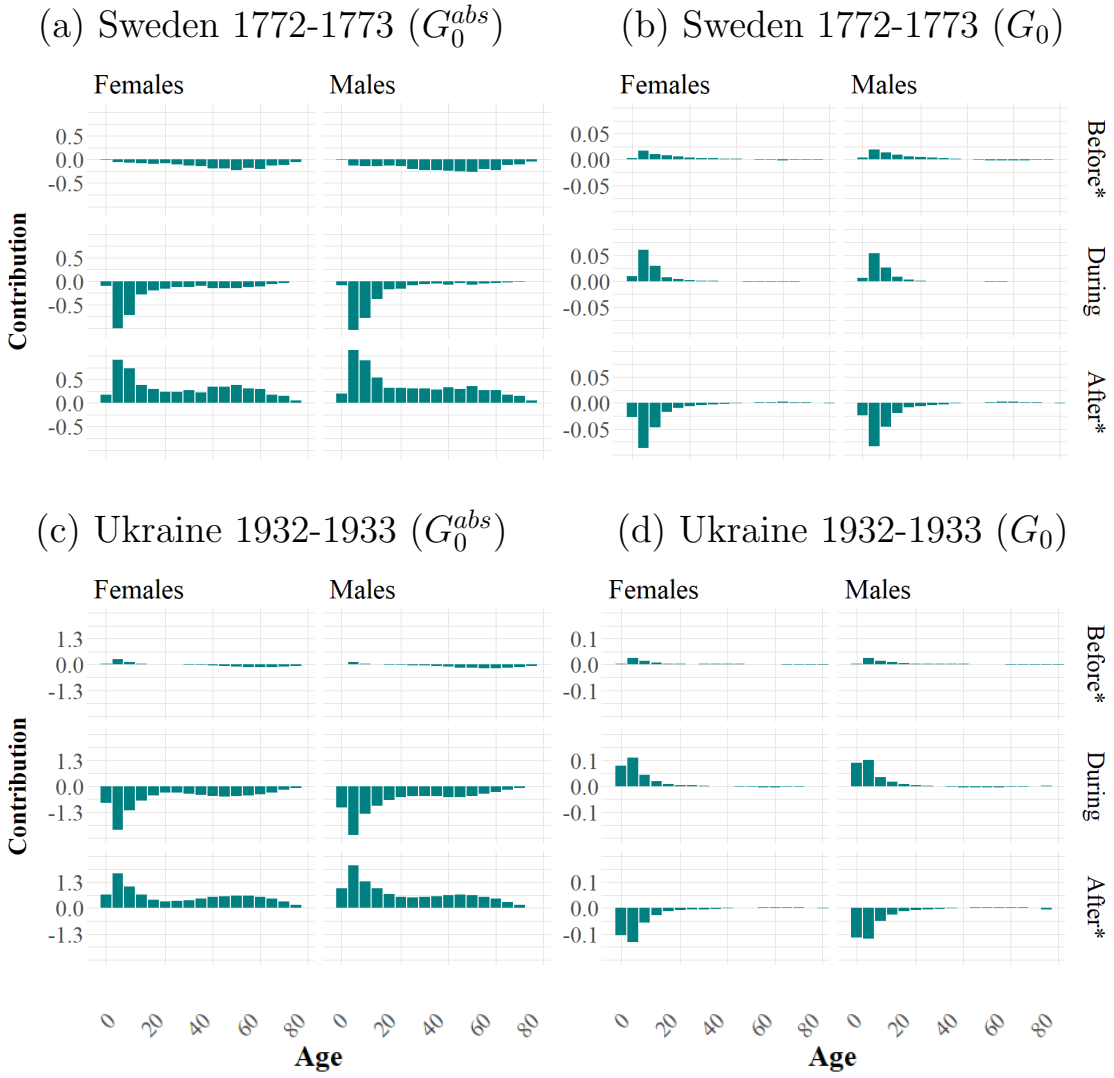

Figure E3: Absolute and relative Gini decomposition, famines. * Before = average of previous 5 years , After = average of following 5 years. G_0^{abs} indicates the absolute Gini coefficient, G_0 indicates the relative Gini coefficient. Source: HMD (Sweden) and Meslé & Vallin

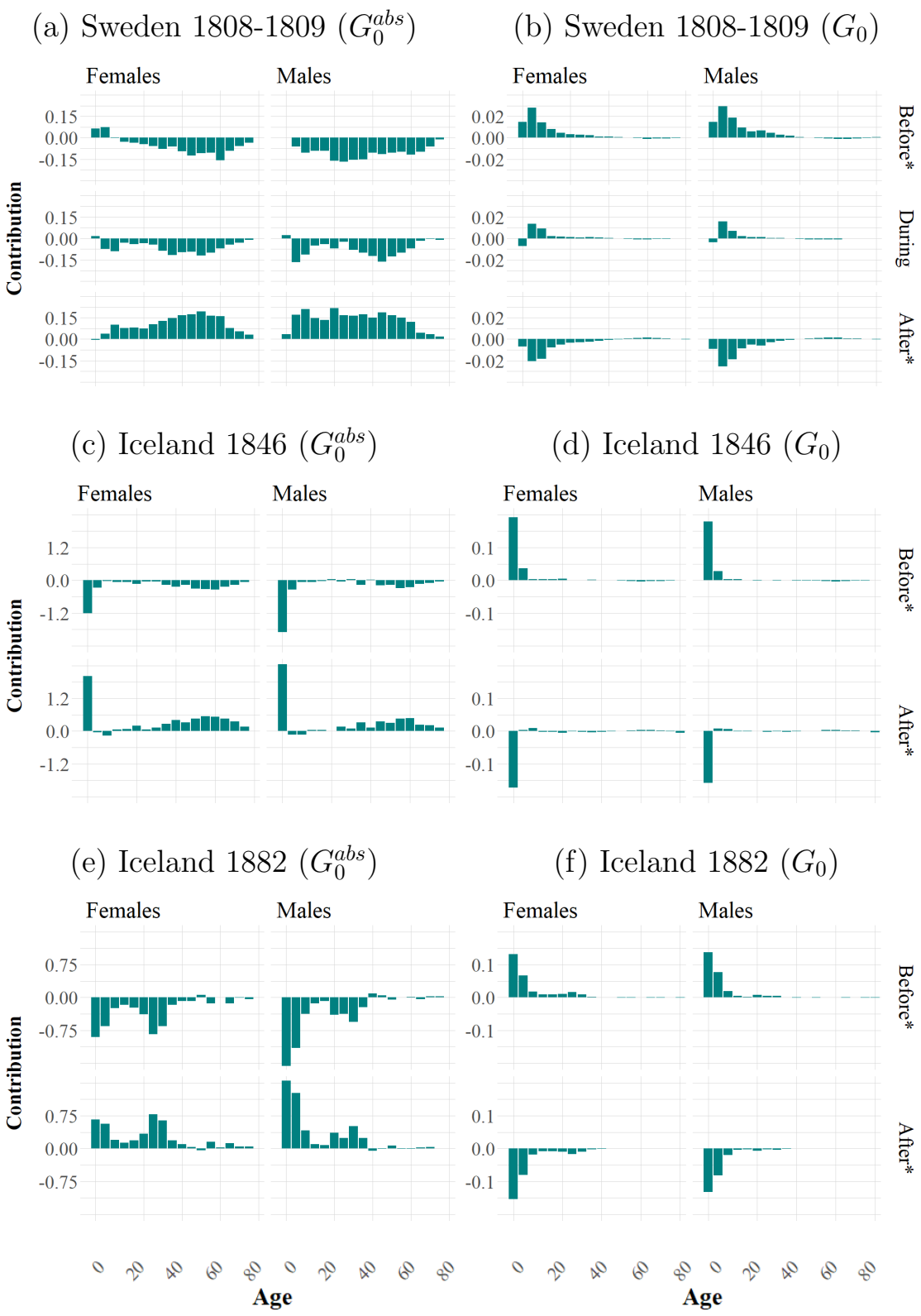

Figure E4: Absolute Gini and relative Gini decomposition, epidemics. * Before = average of previous 5 years , After = average of following 5 years. G_0^{abs} indicates the absolute Gini coefficient, G_0 indicates the relative Gini coefficient. Source: HMD (Sweden and Iceland)