Lifespan Variation During Mortality Crises

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Abstract

Lifespan variation has been increasingly gaining attention as a measure of population health and mortality. Many studies have analysed periods of steady mortality decline highlighting a strong inverse relationship between lifespan variation and life expectancy. More recently, 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 in times of significant mortality increase. Analysing three epidemics and two famines in Europe from the XVIII to the XX century, we find that during these events, relative lifespan variation increases, while absolute variation declines, both quickly coming back to pre-crisis levels afterwards. Using decomposition techniques, we show that absolute and relative indicators diverge because mortality at older ages leads to a temporary increase in the former , but not in the latter. Moreover, female lifespan variation is less affected by the crises than ~~the males~~', a discrepancy ~~mostly~~ explained by the higher impact of infant and child mortality ~~among males~~. This paper offers insights into the effect of mortality crises, showing strong, but nuanced and short-lived consequences on lifespan variation. Contrary to what is often asserted, we show that the choice of lifespan variation indicator is not always inconsequential, namely in times of mortality crisis.

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1 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 population experienced the current conditions throughout its life (Preston et al. 2001). However, it can camouflage other important characteristics, such as the variation in length of life, also known as lifespan variation. Lifespan variation, which describes 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; Vaupel et al. 2011; Smits and Monden 2009; Kannisto 2001). 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 analysed socioeconomic differences (Permanyer et al. 2018; Lariscy et al. 2016; van Raalte et al. 2011; Edwards and Tuljapurkar 2005). 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 ~~the~~ female~~, advantage in lifespan variation remains even in crisis situations,~~ there is ~~very little knowledge~~ about trends in lifespan variation during mortality schocks. We contribute to filling this ~~knowledge~~ gap by focusing on populations ~~experiencing~~ mortality crises, such as famines and epidemics.

~~The contribution of this analysis twofold. Firstly, to better understand the mortality distribution and its trends during historical mortality shocks,~~ by using a novel (in its application) kind of summary indicator. ~~Zarulli et al. (2018) and Zarulli (2013) have used mortality crises to explore the biological underpinnings of mortality, finding support for a higher resilience of women. Our analyses can show whether this translates to lifespan variation as well. Secondly, we hope to enlighten further the characteristics of lifespan variation indicators,~~ by comparing the patterns of a diverse set of measures.

2 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; García and Aburto 2019; Aburto and van Raalte 2018) 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 intial 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)). Still, mortality crises can be classified depending on their causes (Sogner in Charbonneau and Larose (1970); Goubert 1958). Subsistence crises (or famines) ~~depend on~~ a lack or unavailability of food to a considerable part of the population (1), while epidemics are caused by diseases (2). Some crises combine both components, as a famine weakens the population leaving it vulnerable to pathogens (3). Finally, crises can have other causes, such as wars or natural phenomena (4). These latter present specific dynamics, making it difficult to compare them with others. Therefore, we focus on the first three kinds. We adopt a straightforward definition of mortality crises, as periods ~~where~~ 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, as age and gender specific rates vary. In an absolute sense, male adults are more at risk during some epidemics such as those caused by HIV/AIDS (especially in the past), but females may be more vulnerable in proportional terms (Hosegood et al. 2004; Gaylin and Kates 1997), and they are also more affected by natural disasters, toegther with children and the elderly (Frankenberg et al. 2011; Neumayer and Plümper 2007; Bern et al. 1993). Bongaarts and Cain (1982, in Kane 1987) hypothesised that in some cases mortality would reach its peak at the end of a short famine and then gradually decline. After about a year it could even drop below pre-famine levels, as the harvesting effect only left the most robust individuals behind (as was found by Goubert (1958)). However, age and gender specific mortality patterns vary depending on the cultural and social environments. Bengtsson et al. (2009) found that ~~in~~ Scandinavia infants were generally less affected by food availability, as they depend on breastfeeding, while older children ~~are much~~ more sensitive. Because of breastfeeding and pregnancy, women might be more vulnerable to food deprivation, in addition to potential disciminations in the household food distribution (Klasen 1998). Still, females experience lower mortality at almost all ages even in populations experiencing 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 the elderly are more vulnerable to influenza and, in developed countries, to tubercolosis (Yoshikawa and Norman 2009). Age can also interact with gender (Garenne 2015) and social characteristics can affect morbidity and mortality, as during the HIV/AIDS~~V~~ 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 especially weakened individuals (Taylor et al. 2015; Castenbrandt 2014; Goble and Konopka 1973).

2.1 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 concerned, children between ages 1 and 14 suffered the most, while infants witnessed a relatively small 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 are also the diseases involved in the 1808-1809 epidemic, which followed troop movements involved in the Finnish War. Despite the war, the increase in mortality is thought to have resulted mainly from the epidemic (Glei et al. 2019). As a consequence, mortality follows the same age-pattern as in 1773.

Icelandic history is fraught with bad years, "due to cold winters, icefloes, failures of fisheries, shipwrecks, inundations, volcanic eruptions, earthquakes, epidemics and contagious diseases among men and animals" (Magnùs Stephensen, in Tomasson (1977, p. 410)). In 1846 and 1882 measles struck. Both years, particularly cold springs and summers forced fishermen to remain ashore, facilitating the spread of the disease, 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 following epidemic in 1882, which affected ages under 40. This epidemic lasted only from June to August and led to the death of around 2% of the population (Shanks et al. 2015; Cliff et al. 1983).

The context of the Ukrainian famine of 1932-1933 is more difficult to describe, as data was not public until the end 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 implemented from 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 Naumenkoherself maintains). Finally, a third strand of research argues that the famine was caused by the decision of the central government to retain food distribution in order to quell anti-government sentiments in Ukraine sparked by collectivisation policies (Graziosi 2015). Still, 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 (Rudnytskyi et al. 2015, Meslé and Vallin 2012).

3 Research questions

Our first research question looks at the trends of 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 of mortality at adult ages, together with some convergence at older ages (2013) 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 for children and the elderly; for the second a greater increase for the 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 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 where contributions go from positive to negative (and vice-versa when mortality declines). This is called the threshold age (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 at 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 during the crisis there is selection at the vulnerable ages, 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 determines whether some ages ~~especially~~ contributed 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 subgroups are physically less equipped to deal with extreme conditions and children might receive less care in situations where resources are scarce. This phenomenon is context-specific (Lanau and Fifita 2020), but Bengtsson et al. (2009) have shown it might have existed in Sweden. Gender inequality can also exacerbate this uneven allocation of resources for boys and especially for girls. Moreover, deaths at the extremes of a distribution will ~~more heavily~~ affect variation. Therefore, we expect that these age groups will ~~largely contribute to the expected~~ increase ~~in~~ lifespan variation. However, because of the reasons outlined earlier, the contribution of older age groups might be smaller than that of young individuals.

4 Methods and data

4.1 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 gauge (van Raalte and Caswell 2013). ~~Therefore,~~ 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 1955b); the relative and absolute Gini coefficients, G_0 and G_0^{abs} respectively (Shkolnikov et al. 2003, Hanada 1983). Each pair is constituted by 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 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 $e(x) = \frac{1}{l(x)}\int_0^\omega l(a)da$ 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 each individual's age 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 1955a). 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 at 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 tied with 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 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 ~~Horiuchi~~ method. Horiuchi et al. (2008) show that a change in a continuous function can be expressed discretely as

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

where $c_i = \int_{x_{i1}}^{x_{i2}} \frac{\partial y}{\partial x_i 1} dx_i$ is the total change in the value y by changes in the i-th covariate x_i, y_1 and y_2 are the values of the function under analysis at times t_1 and t_2 respectively and x_{i1} and x_{i2} are the values of the i-th covariate x_i, again 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.

4.2 Data

We use two data sources. The first is the Human Mortality Database (Barbieri et al. 2015). It gathers several mortality-related data by 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, only Scandinavian countries have data series which include mortality crises.

We use data for Iceland and Sweden, which were collected by parishes since the XVIII 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. Death rates at older ages were smoothed to correct excessive fluctuations and to separate data included into 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 account for 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 has 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.

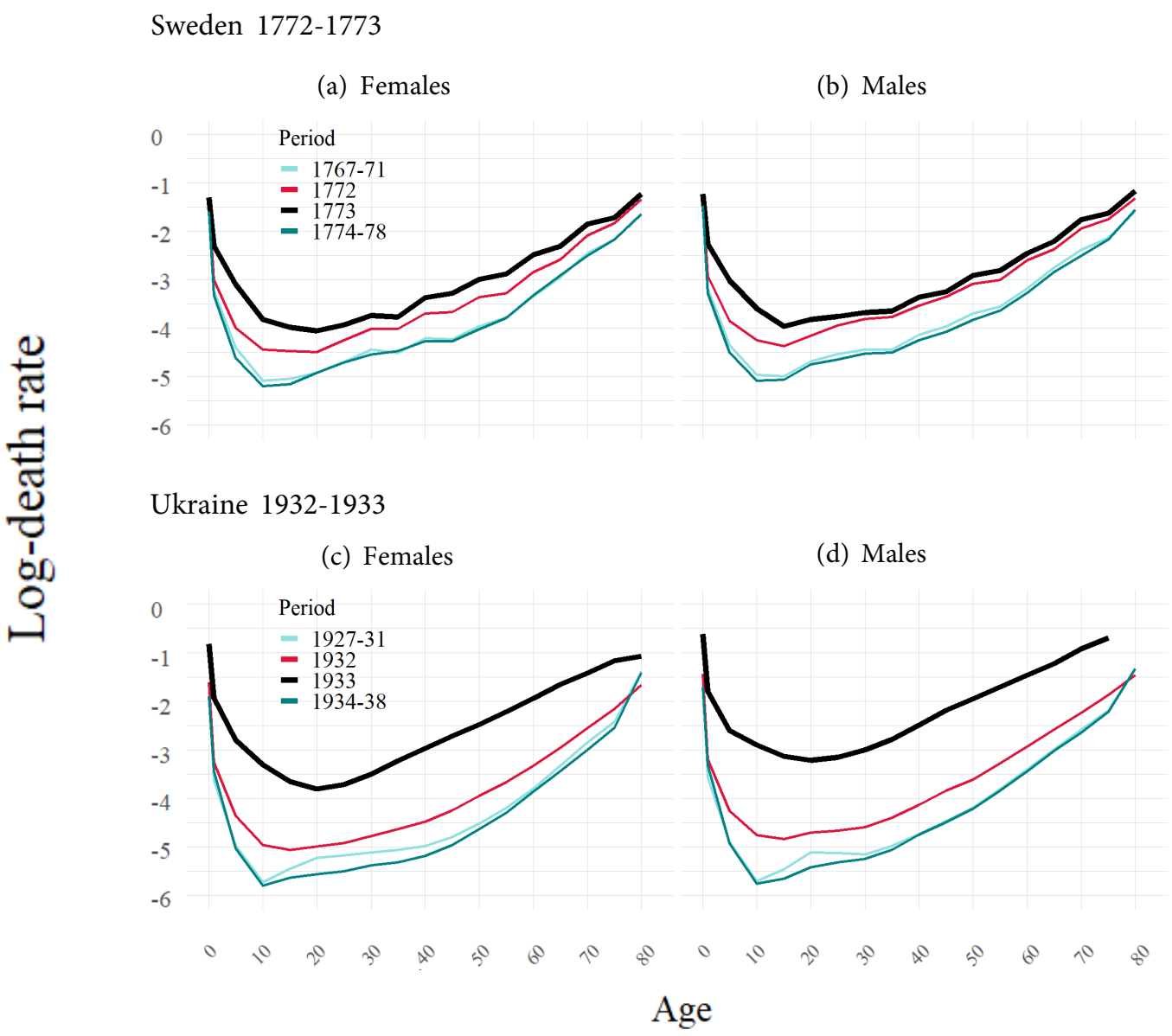
5 Results

5.1 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.

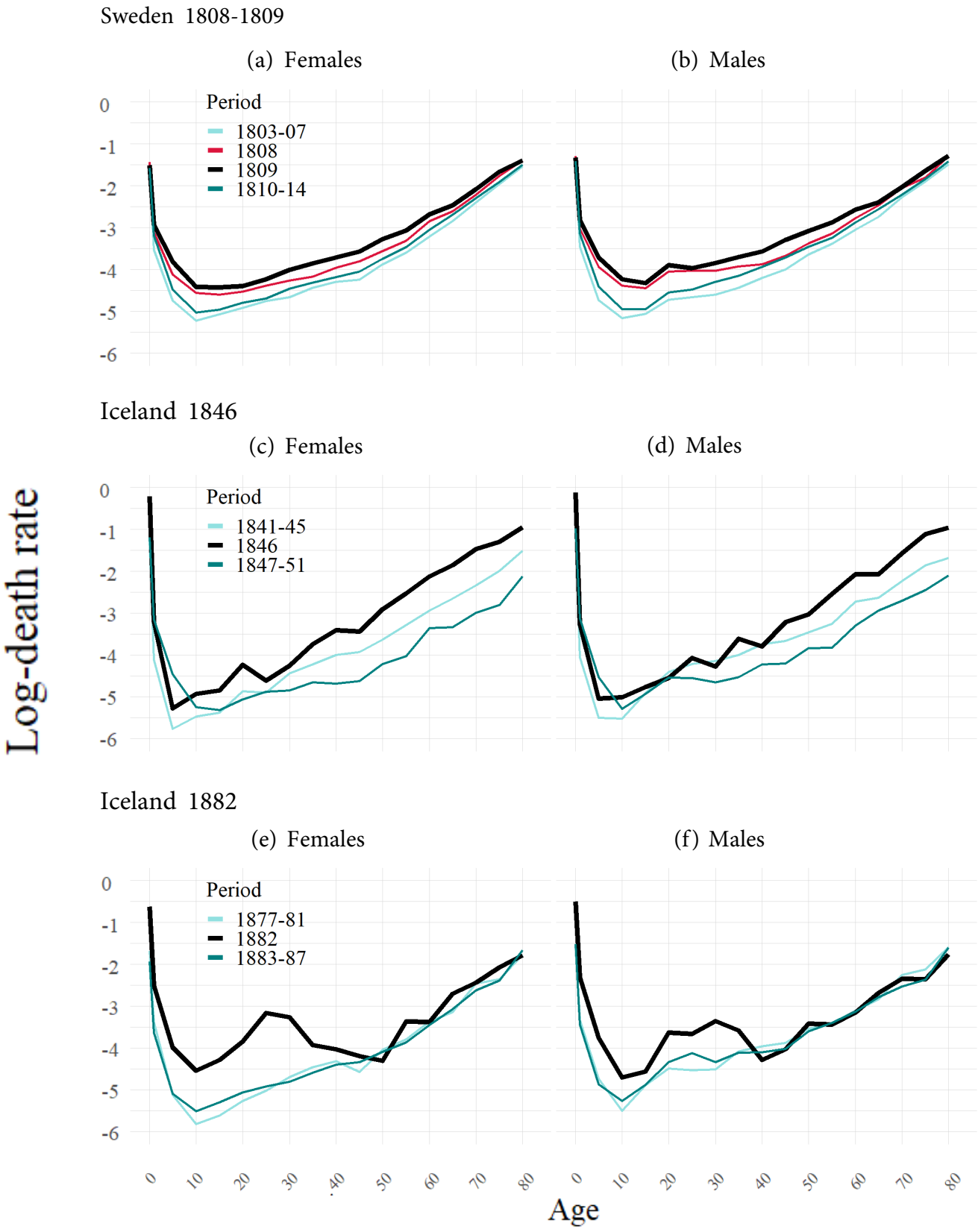
Despite some differences in magnitude and in the temporal dynamics of famines (Figure 1), there are many parallels. In both cases, death rates increased proportionally at adult ages. The increase is smaller at age 0 and there is evidence of convergence at the oldest ages, for both sexes in Sweden and for females in Ukraine (as the survival function for Ukrainian males in 1933

Figure 1: Log-death rates, famines



Source: HMD (Sweden) and Meslé & Vallin

Figure 2: Log-death rates, epidemics



Source: HMD (Sweden and Iceland)

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. In 1809 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 epidemics show significant variability, as can be expected from a smaller population. Thus, patterns should be considered with care, especially when they are seemingly idyosincratic. 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 under pre-crisis levels, while child mortality was higher even than during the crisis itself. Finally, female mortality during the crisis is higher than the males', 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.

Figures B1 and B2 show the difference in log-death rates between the (main) crisis year and the other periods for all five crises.

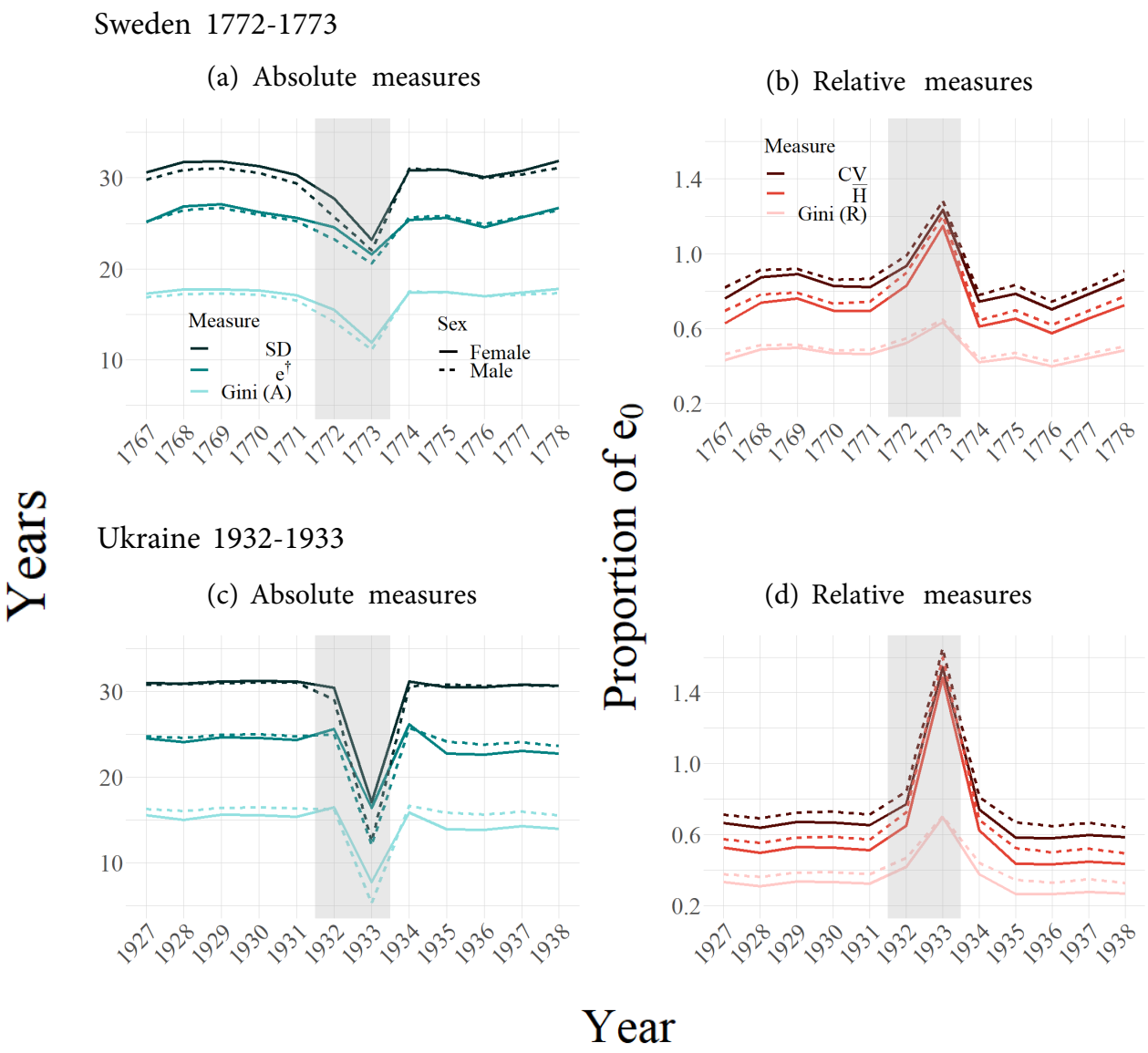
5.2 Trends of lifespan variation

In figures 3 and 4, we examine how absolute and relative lifespan variation measures behave (mark that here, each non-crisis year is handled separately and not averaged). A striking finding is that absolute and relative variations present opposite 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 figure C1).

We also find differences within each group of measures. The two Gini coefficients present the flatter curves, with smaller absolute changes. However, the lesser 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.

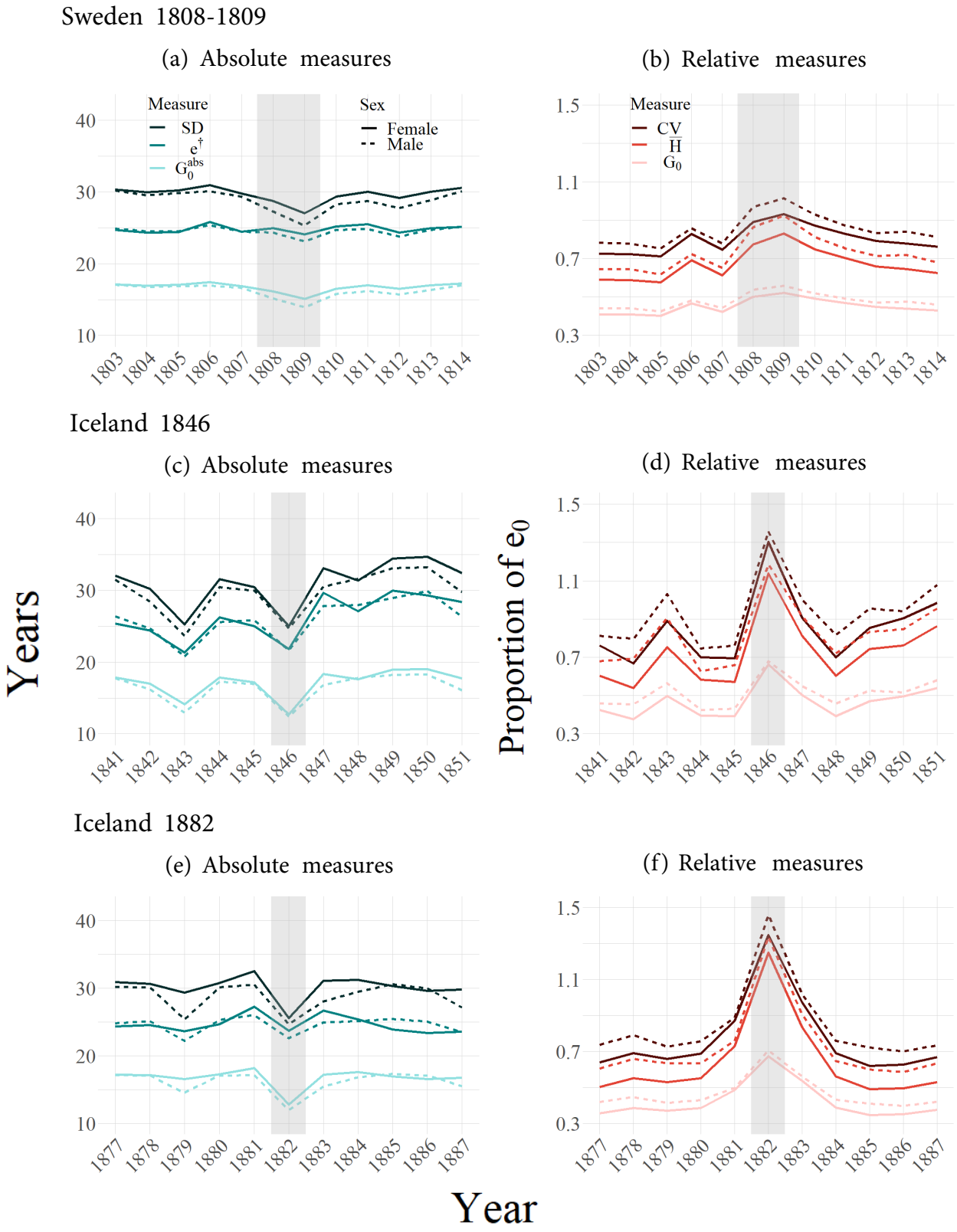
Absolute variation starts decreasing right away in two of the three crises spanning two years. In Ukraine, there is a noticeable drop only in 1933, with the small exception of male SD. However, relative variation starts rising (albeit slightly) in 1932 already, further underscoring the different behaviours 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

Figure 3: Lifespan variation, famines



Source: HMD (Sweden) and Meslé & Vallin

Figure 4: Lifespan variation, epidemics



Source: HMD (Sweden and Iceland)

in e_0, meaning that the increase in relative variation is much lesser in 1843 and non-existent in 1879. ~~Although these years are not part of our current focus, an analysis of their circumstances could shed light on the relationship between lifespan variation and life expectancy.~~ Still, the same partly unexpected trend holds true for all five crises: absolute lifespan variation declines, while relative variation increases.

5.3 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 figures 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 $+0.034$ 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 which 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, each graph has its own scale.

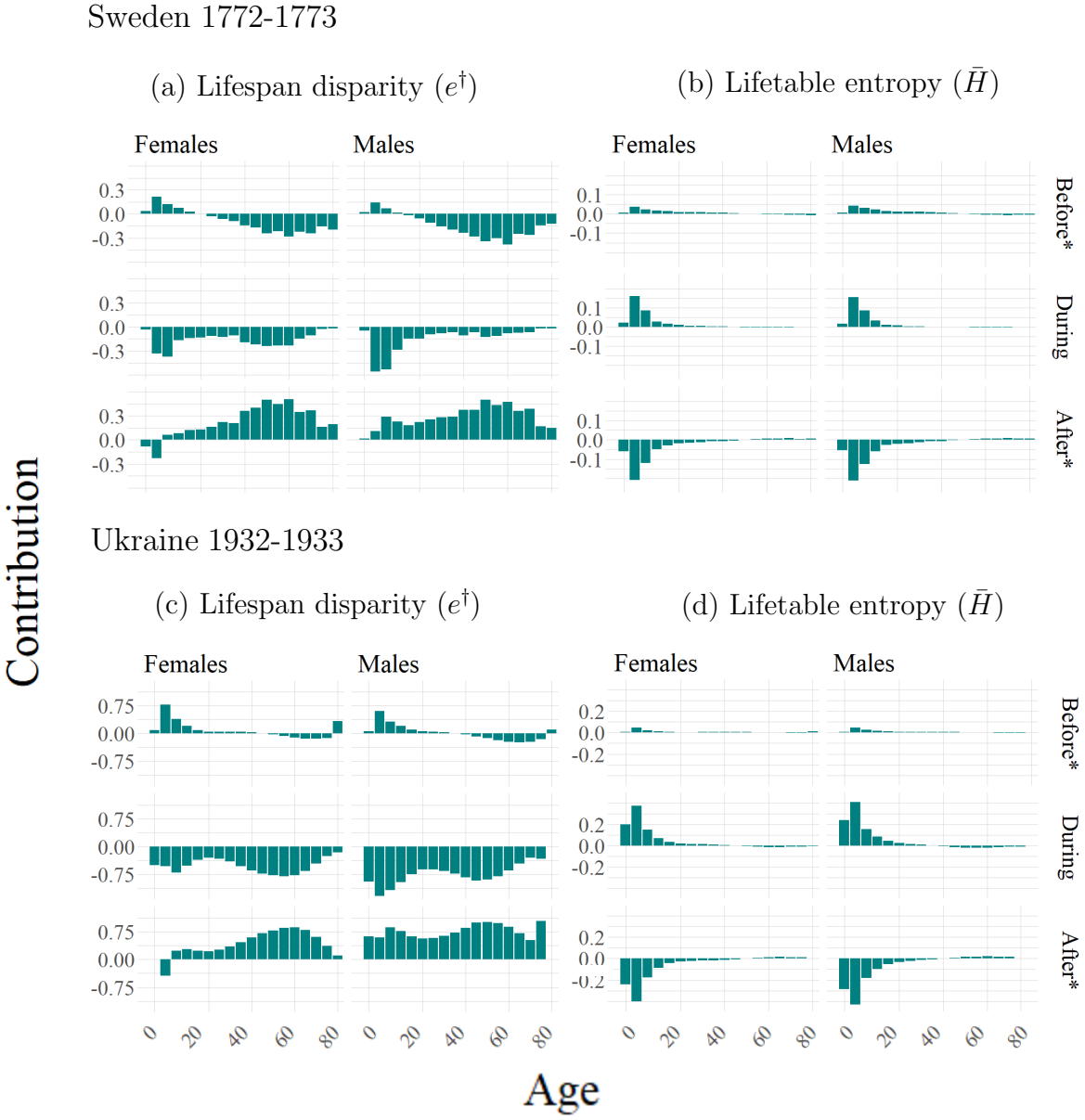
Older ages influence very little the change in $\bar{H}$, even when they contribute significantly to the change in $e^\dagger$. Sex differences are more accentuated for the latter compared to the former, especially at young ages and for the Icelandic crises. While the uneven effect of measles can partly 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 except for 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 or worsenings of mortality from one period to the next (see figures 1 and 2). Table D1 shows the values of e_0 and of the threshold ages for $e^\dagger$ and $\bar{H}$.

6 Limitations

Data quality is not necessarily assured. The parish registers at the base of the HMD's death and population estimates do not record migrants. However, these are likely to have been healthier than those who stayed behind (Helgesson et al. 2019), so that this gap should not overly influence our results. The Ukrainian data poses some specific issues. Namely, deaths are likley to have been

Figure 5: Lifespan variation decomposition, famines



* Before = average of previous 5 years , After = average of following 5 years

Source: HMD (Sweden) and Meslé & Vallin

Figure 6: Lifespan variation decomposition, epidemics

* Before = average of previous 5 years , After = average of following 5 years

Source: HMD (Sweden and Iceland)

underreported, especially infant deaths (in fact the latter may have been underreported in Sweden and Iceland as well (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. To address these issues, we changed the death rate by $\pm 10\%$ for every age and for infants only. We also changed the open-ended category in the lifetables between 70 and 90. Finally, we ran further sensitivity checks on an alternative dataset about the Ukrainian famine, graciously supplied by professor Wolowyna (Rudnytskyi et al. 2015). All of these confirmed our main results.

Even when of good quality, the data rarely provide information on social status or geographic location, for example, which could help refine the results (Zoraster 2010). Although we find reoccurring patterns for historic populations, these are not necessarily extendable to modern populations, 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 motality crises, such as the current pandemic, generally milder than the ones we study. Still, our results can act as a comparison term for future analyses looking at lifespan variation during contemporary crises, to understand if a regular pattern emerges, which trascends time. Methodologically, the translation of a continuous change into a discrete framework necessary for the decomposition analysis leads to some imprecisions, which can be exacerbated by extreme mortality change (as might be the case for the Icelandic crises).

7 Discussion

We have analysed five mortality crises, two famines and three epidemics, happening in three countries and across 160 years. Each case presented a pattern of mortality change through the crisis, which can be linked to its specific context. 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 weight of young and older ages between absolute and relative measures.

7.1 Mortality patterns

The five mortality crises we study were characterised by ~~an~~ 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 harvesting effect at adult ages, translating into a lower-than-pre-crisis mortality after the crisis itself. Some trends deviate from this model, but they are mostly attributable to the context in which the crisis happened and 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, being net consumers of resources, might

have become expendable in times of crisis. As this attitude changes across time and space, it could clarify the dispropotionate increase of child mortality in the Swedish famine, which did not happen in Ukraine. Moreover, the Ukrainian data have been extensively manipulated and smoothed, and might be hiding a similar increase for children.

The peculiar pattern of male rates for the 1808-1809 Swedish epidemic can be tied with 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 of 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 with the lasting effects of exposure to infectious diseases in early childhood (Stoermer 2011; Fridlizius 1989), 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 searched in a specific behaviour exposing this age group.

7.2 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 affect negatively both e_0 and variation, but the drop of absolute measures nuances this interpretation. The crises we have studied seem to have compressed the mortality distribution, by shortening lifespans for everybody. Thus, ages at death were 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 showing that the relationship between life expectancy and lifespan variation, typically found to be negative (Colchero et al. 2016; Sasson 2016; van Raalte et al. 2011; Smits and Monden 2009), is not so straightforward (Permanyer et al. 2018; van Raalte et al. 2014; Edwards and Tuljapurkar 2005). 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 concerning the sex gap, to the societal structure or to some other factor. ~~Unfortunately, the present research cannot illuminate the question further.~~

Despite the massive effect 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, figures 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.

7.2.1 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 (Pongou 2013; Department of Economic and Social Affairs, Population Division 2011; Drevenstedt et al. 2008). 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 the influence of older ages, while they contribute more in the other periods. Although the contribution of child mortality to the change in $\bar{H}$ are not identical across the sexes, the differences are less pronounced. An exception to these trends are the two Icelandic epidemics, where lifespan variation changed equally or more for females. These differences seem again to be mostly explained by differences in the contribution of young ages. Thus, young ages determine existing sex differences in lifespan variation change.

7.2.2 Methodological implications

These 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 starts decreasing as well (as shown in figure A1b). Permanyer and Shi (2020) find a similar plateau in absolute lifespan variation for very high levels of e_0. Whilst they tie this plateau to a recent slowdown in e_0 gains, compared with gains in longevity, it is more difficult to adopt this framework here, because in our cases 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, which lead the direction of this change. Since the threshold age for $e^\dagger$ ($a^\dagger$) trails e_0 closely, it was quite low in the periods we consider (see table D1). Thus, mortality increases even at relatively low ages 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. Figure A1a shows a monotonous and negative relationship between $\bar{H}$ and e_0 throughout the age distribution. The definition of rel-

ative measures is key in understanding this trend. e_0 is more sensitive to the crisis than absolute variation (figure C1). Since 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 on all the others. Decomposing the change in e_0 (figure C2) shows that young ages' contributions 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, as we had expected, led by mortality at young ages, changes in absolute variation are to a great extent determined by older ages, a difference which 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.

7.2.3 Policy implications

Greater lifespan variation has concrete implications on lifecycle investments and consumption, as individuals assess their chances of benefiting from such decisions in the future (Tuljapurkar 2011), and poses ethical dilemmas for the organisation of pension and health systems (Brønnum-Hansen et al. 2017). Previous research has already called for greater public awareness of lifespan variation and of its consequences at both the 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 actually 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 reflexion on the meaning of the tools we use to measure it, so as to mirror the preferred conception of equality.

8 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 make everyone unequally poor. 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 connect with the literature, which 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 looking at different kinds of crises. Longer and milder crises could present an interesting midway case study and could better show long-term effects of adverse conditions, as could analyses at the cohort level. 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 as well more research is still needed.

9 Contributions

JMA and SV conceived and designed the study and carried out the analyses and coding. IP and VZ contributed with comments on the implementation of the analysis. SV wrote the paper with helpful comments and critical revisions from JMA, IP and VZ.

10 Acknowledgments

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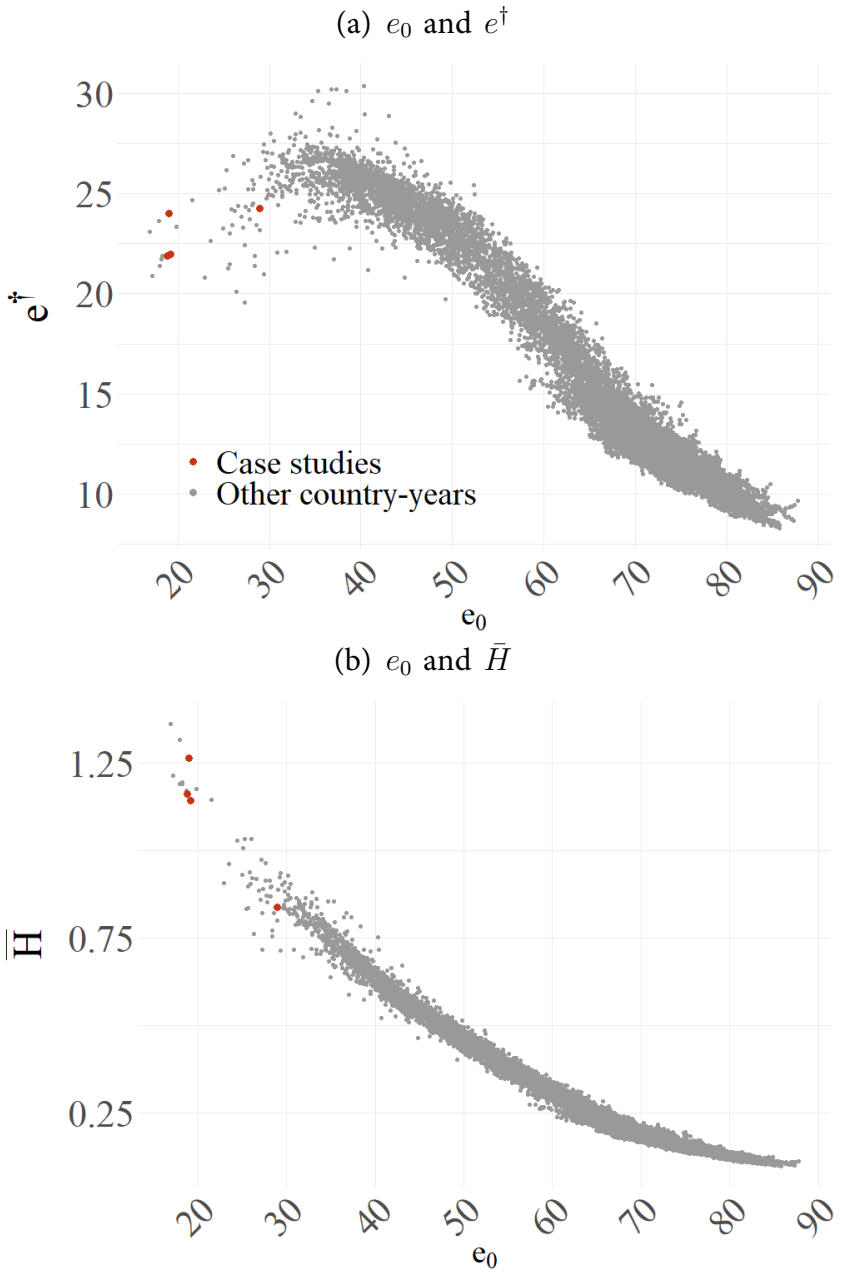
Appendix A

Figure A1: e_0 and lifespan variation



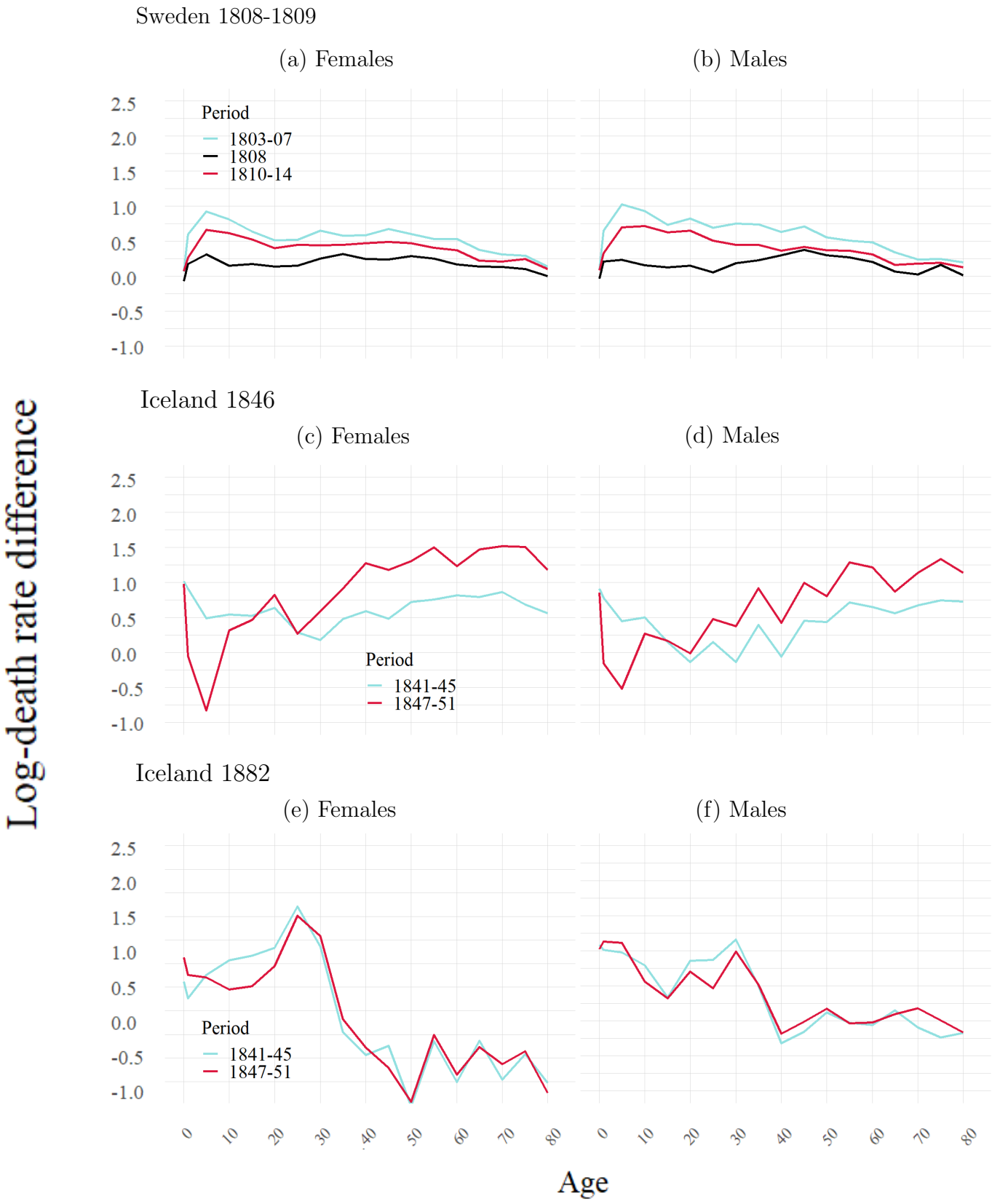
Source: HMD, all countries

Appendix B

Figure B1: Differences in log-death rates with main crisis year, famines

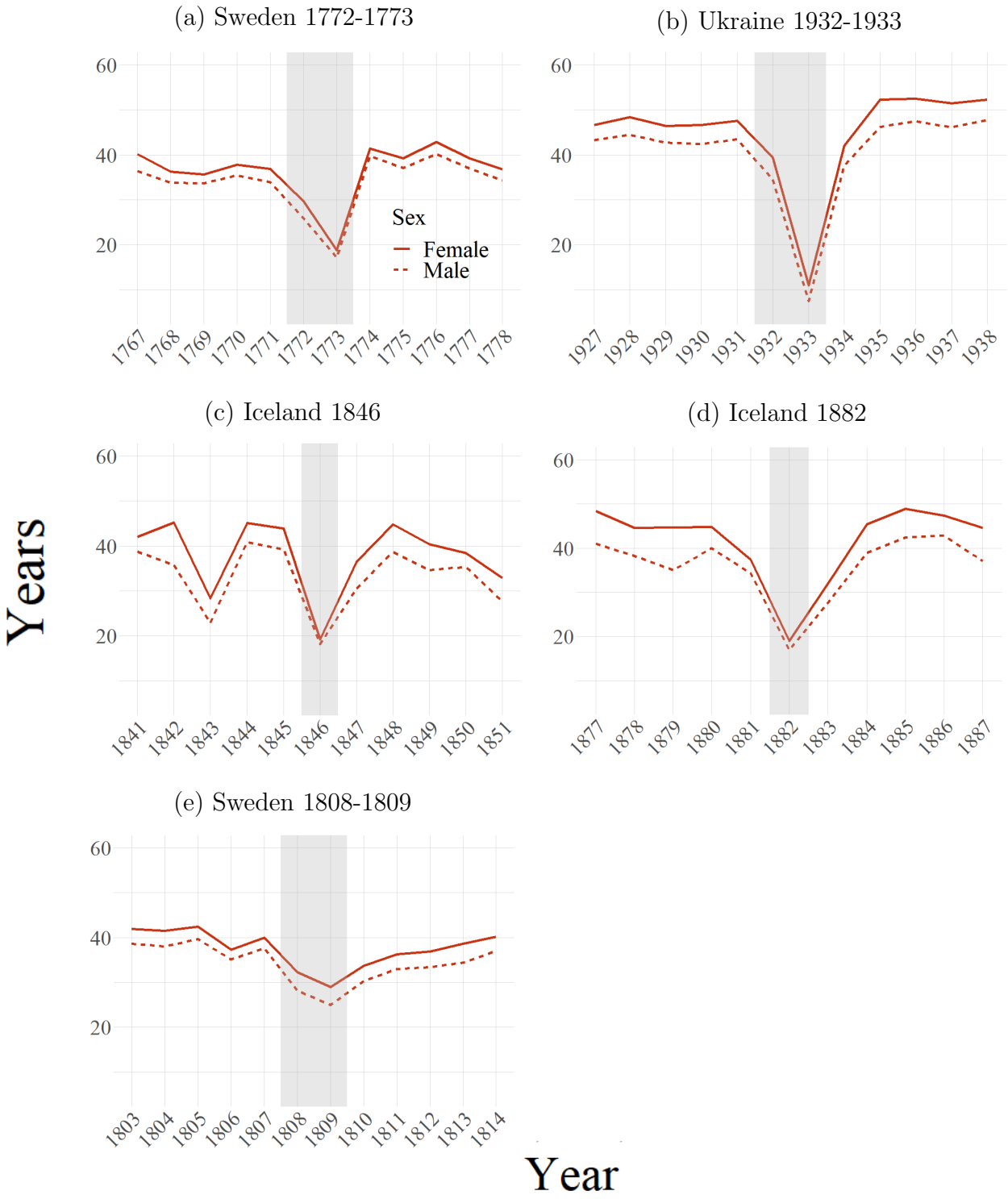
Source: HMD (Sweden) and Meslé & Vallin

Figure B2: Differences in log-death rates with main crisis year, epidemics



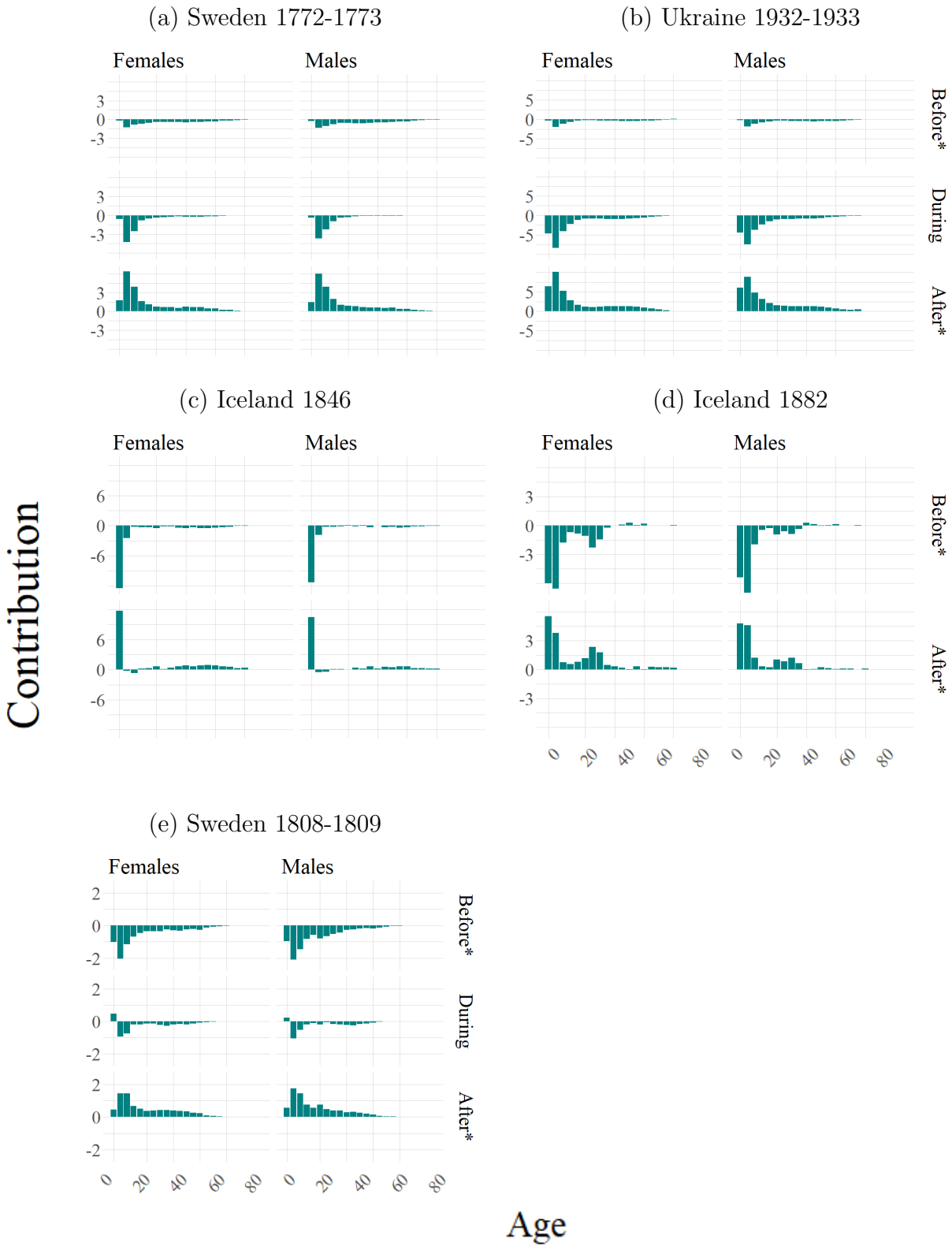
Source: HMD (Sweden and Iceland)

Figure C1: Trends of e_0



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 & Vallin

Appendix D

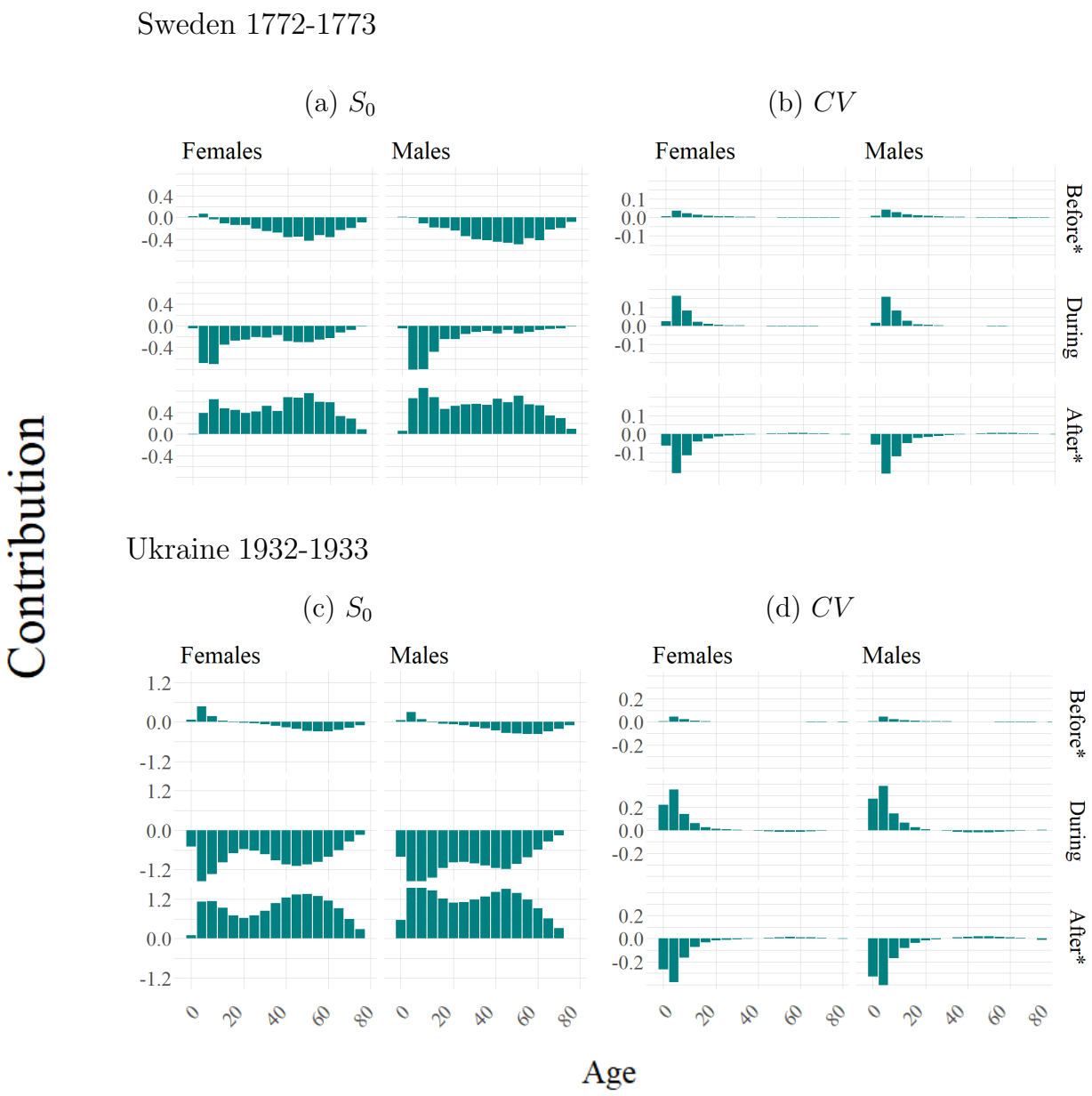
Table D1: e_0 and threshold ages for $e^\dagger$ and $\bar{H}$

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

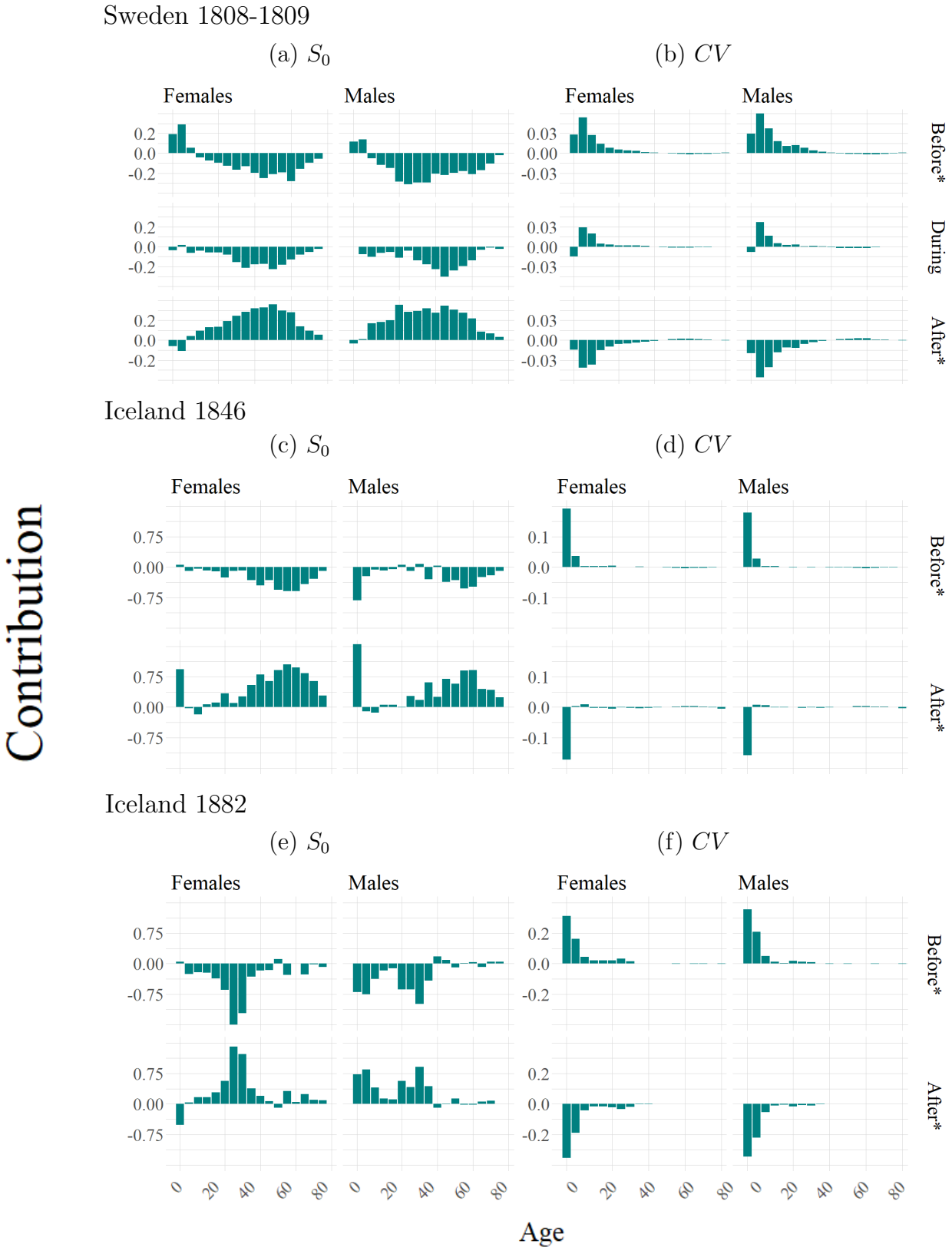
Figure E1: Standard deviation and coefficient of variation decomposition, famines



* Before = average of previous 5 years , After = average of following 5 years

Source: HMD (Sweden) and Meslé & Vallin

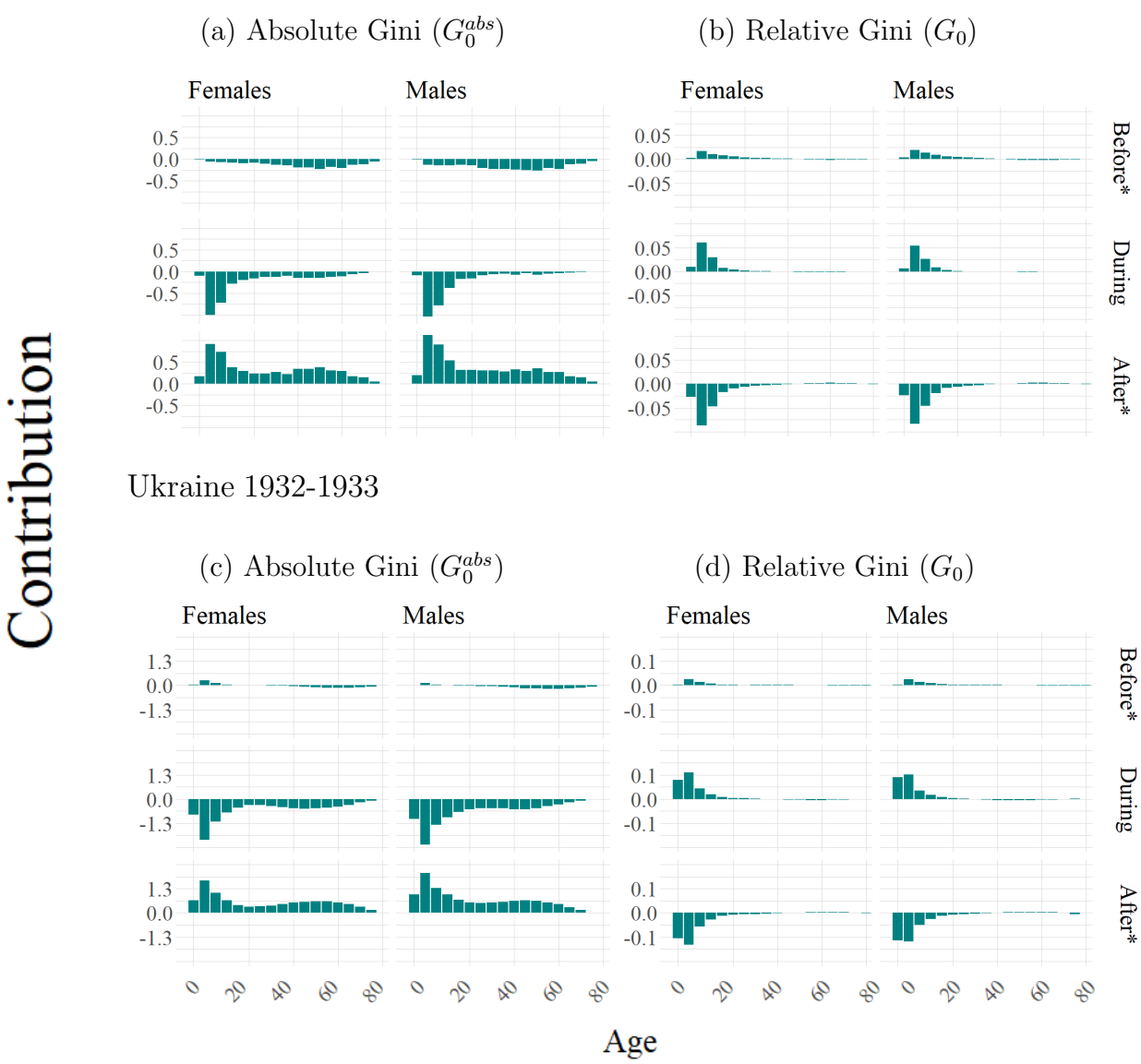
Figure E2: Standard deviation and coefficient of variation decomposition, epidemics



* Before = average of previous 5 years , After = average of following 5 years

Source: HMD (Sweden and Iceland)

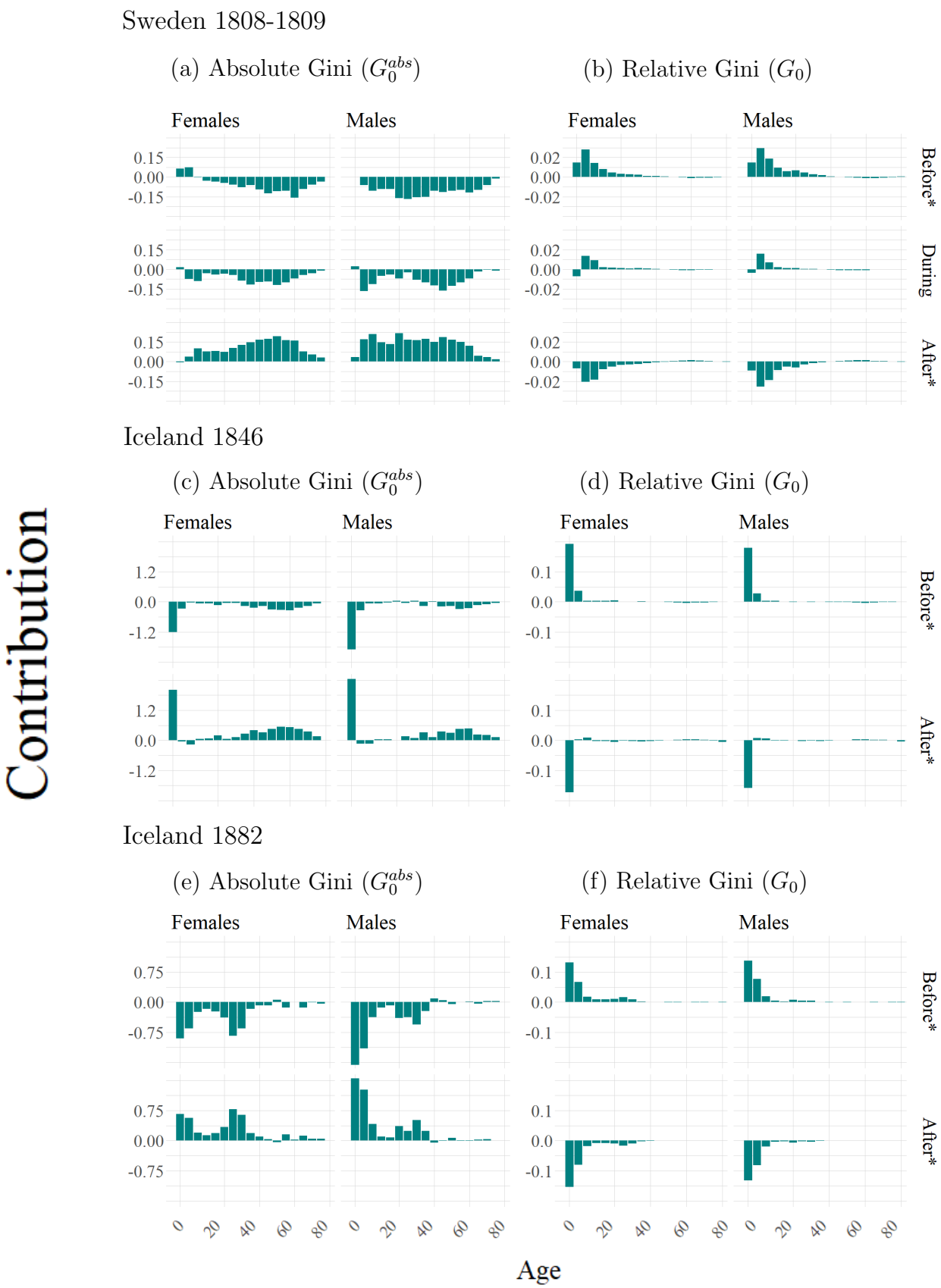
Figure E3: Absolute and relative Gini decomposition, famines



* Before = average of previous 5 years , After = average of following 5 years

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

Source: HMD (Sweden and Iceland)

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