Abstract

1 Introduction

Life expectancy is often used as a summary measure to describe the state of a population in terms of mortality, as well as in terms of health (Cao et al. 2017; Hay et al. 2017; Bilal et al. 2019). In this sense, life expectancy convenently expresses the average remaining years of life in a given year (Preston et al. 2001). However, it can camouflage other important characteristics of a population. One such characteristic is the variation in the age at death, also known as lifespan variation. Lifespan variation, which describes uncertainty ~~of~~ the timing of death at the individual level and ~~at the aggregate level~~ underlies heterogeneity in population health, has been decreasing as life expectancy and, more recently, the modal age at death have increased (Colchero et al. 2016; Kannisto 2001; Smits and Monden 2009; Vaupel et al. 2011; Aburto et al. 2020). Yet, life expectancy and lifespan variation have been shown to follow different trends, so that an increase in life expectancy does not necessarily imply a decline in lifespan variation (Aburto and Raalte 2018; Brønnum-Hansen 2017; Sasson 2016; Wilmoth and Horiuchi 1999). Greater lifespan variation has concrete implications on lifecycle investments and consumption, as individuals assess their chances of benefiting from such decisions in the future (van Raalte et al. 2018; Tuljapurkar 2011), and poses ethical dilemmas for the organisation of pension and health systems(Brønnum-Hansen et al. 2017).

Studies on lifespan variation have mostly focused on populations with continued mortality improvements or analysed differences by social determinants, such as SES or educational level (Edwards and Tuljapurkar 2005a; Lariscy et al. 2016;Permanyer et al. 2018; van Raalte et al. 2011). More recently, some studies have started shedding light on ~~the behaviour~~ of lifespan variation when life expectancy stagnates or decreases, for the entire population or for some socioeconomic subgroups (Permanyer and Scholl 2019; García and Aburto 2019; Sasson 2016; Aburto and Beltrán-Sánchez 2019). Yet, to the best of our knowledge, lifespan variation has rarely been studied in circumstances where mortality increases sharply. ~~An exception is the study by~~ Colchero et al. (2016)~~, which finds~~ that the gender gap in lifespan variation, which usually favours females, remains even in crisis situations. ~~Despite this finding, that paper does not focus on lifespan variation during mortality crises, but rather on the overall relationship between life expectancy and lifespan variation across a number of human and nonhuman primate populations.~~ We contribute to filling this gap by focusing specifically on populations that have experienced a mortality crisis.

This analysis aims at making three main contributions. First ~~of all~~, studying the patterns of lifespan variation can shed light on the behaviour of mortality under such extreme circumstances and give additional information on mortality in general. For example, Zarulli (2018; 2013) has used mortality crises to explore the biological underpinnings of mortality. By comparing lifespan variation trends for men and women separately, we can examine if the higher resilience of the former in terms of life expectancy translates to lifespan variation as well. Secondly, by using different measures of lifespan variation, we can study how each captures the considerable changes tied with mortality crises and better understand their properties in such situations. Finally, these analyses can contribute to laying the fundation for studying the consequences of mortality crises nowadays. Although such crises are not a current event in contemporary Europe, recent evidence suggests that they will likely become an ever more pressing question throughout the world as extreme weather events increase in frequency with climate change, potentially bringing natural catastrophes and food shortages in their wake, as well as increasing the risk of epidemics (Cynthia et al. 2001; Li et al. 2019; Mweya et al. 2016; Tirado et al. 2010). The vulnerability of countries across the world

to such events was made evident by the ongoing pandemic of COVID-19.

2 Background

A strong negative relationship between life expectancy and lifespan variation has been highlighted by numerous studies on the subject, across time, countries and social groups (Wilmoth and Horiuchi 1999; Sasson 2016; Van Raalte et al. 2011; Vaupel et al. 2011). The same result was highlighted for different primate populations by Colchero et al. (2016), who have calculated that when comparing two such populations, the difference in life expectancy is, on average, 28 times the difference in lifespan equality, a value closely tracked by the 25.4 times found by Aburto et al. (2020). However, the relationship is not as simple as this might suggests. Greater life expectancy does not necessarily imply lower lifespan variation. Since the 1980s, for example, while all strata of the Danish population have enjoyed longer lives on average, lifespan disparity actually increased for the least educated, a trend similar to that found in recent years for Finland, Spain and the USA (Brønnum-Hansen 2017; van Raalte et al. 2014; Sasson 2016; Permanyer et al. 2018). In fact, the correlation between the two measures very much depends on the threshold age and on the ages at which mortality changes occur (Aburto and van Raalte 2018; García and Aburto 2019; Aburto et al. 2020 and even when there is a correlation, life expectancy does not completely predict lifespan variation (Van Raalte et al. 2011).

Mortality crises themselves have also been examined. The definition of what constitutes a mortality crisis has been debated, with a number of indices ~~variously~~ based on duration, intensity of mortality and/or comparisons with previous years (for an interesting introduction to the issue, see for example Charbonneau (1970)). Despite these discussions, Sogner (in Charbonneau (1970)) classified mortality crises in four main categories, depending on their causes. Subsistence crises (or famines) are brought about by a lack of food, or by its unavailability to a considerable part of the population, while epidemics are caused by diseases. Some crises combine famine and epidemic components, most often in this order as the famine weakens the population leaving it vulnerable to pathogens. Finally, crises can have other causes, such as wars or natural phenomena. These latter usually present specific dynamics, so it might be difficult to compare them with the others. For this reason, we focus on the first three kinds. More specifically, we adopt a straightforward definition of mortality crises, as periods where life expectancy at birth e_0 decreases drastically and quite suddenly. For the purposes of this article, we only consider relatively short crises, spanning one or two years. ~~Although longer crises would certainly be interesting to analyse, we postpone such work to a later date.~~

No single mortality pattern can be expected during crises, as age and gender specific rates vary, ~~not only for "other-cause" crises, but also~~ between famines and epidemics. For example, male adults are more at risk during some epidemics such as those caused by HIV/AIDS (Gaylin and Kates 1997; Hosegood et al. 2004), while natural disasters seem to affect more women, children and the elderly (Bern et al. 1993; Frankenberg et al. 2011; Neumayer and Plümper 2007). Regarding famines, Bongaarts and Cain (1982, in Kane 1987) hypothesised that in some cases mortality would increase during a famine to reach a peak at its end. Afterwards, mortality rates would gradually decrease as the long-term consequences of food deprivation took their toll. Finally, after a year or so, mortality could even drop below its pre-famine levels, as the famine finished "harvesting" the

weaker individuals, leaving only the most robust behind. However, age and gender specific mortality patterns vary depending on the cultural and social environments. Some information about Scandinavian trends can be found in Bengtsson et al. (2009), who analysed historical data linked with increased food prices. They found that infants were generally less affected by increased food prices, as they mostly depend on breast-feeding, while older children are much more sensitive to external conditions. Because of breastfeeding and pregnancy, women are more vulnerable to food deprivation, which may also affect them more in case of an unequal distribution of food in the household, which often favours males. In fact, Zarulli et al. (2018) found that the life expectancy gender gap advantages females at almost all ages even in populations experiencing extremely high mortality, suggesting that females might benefit from advantageous biological characteristics. However, they also found signs that this gap can reverse because of social preferences and the incidence of gynaecological diseases or childbirth complications. Finally, the elderly are also affected, but show little differences in terms of gender and socio-economic status, possibly as a result of the selection of the most robust individuals into old age (Bengtsson et al. 2009).

When looking at epidemics, trends become even more complicated, as each disease is characterised by a set of age and gender specific ~~mortality~~ rates. For example, the risk of dying of a cardiovascular disease increases with age (AIHW 2010), while malaria, an infectious disease, affects predominantly young children (WHO 2018). Moreover, age can interact with gender, adding to the complexity of mortality patterns (Garenne 2015). Finally, social characteristics can also be determinants of morbidity and mortality, because of the prevalence of certain ways of transmission or different access to health care, as was the case, for example, during the AIDS/HIV epidemics of the 1980s in the USA (Gaylin and Kates 1997).Of the three epidemics we consider, two were caused by measles, which usually affects children, but also non-immunised adults, common in isolated communities previously spared by the virus. Measles also typically take a greater toll among female children in terms of mortality, although females usually suffer lower prevalence and severity from other viral infections (Muenchhoff and Goulder 2014; Garenne 2015).The last epidemic we analyse was caused by typhus and dysentery, diseases which kill especially weakened individuals, such as children and the elderly (typhus especially the latter), while no clear gender differences have been found (Castenbrandt 2014; Taylor et al. 2015; Goble and Konopka 1973).

2.1 Context

~~Now that we have given a general outlook of the trends of mortality in famines and epidemics, let us consider our specific cases. First of all,~~ we look at the Swedish famine of 1772-1773 and at the typhus and dysentery epidemic, which struck this same country in 1808-1809. Then, we turn to the two measles epidemics Iceland experienced in 1846 and 1882. Finally, our most recent crisis is represented by the Ukrainian famine of 1933.

Dribe et al. (2015) describe the mortality response to the 1772-1773 famine and the 1808-1809 epidemic in Sweden. Crop failures in large regions of Sweden caused by unusual weather in 1772 exacerbated already high food prices and led to a famine which peaked the following year. In 1773 mortality 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 1 and 14 years of age 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 epidemics, which followed troop movements involved in the Finnish War. However, the increase in mortality is thought to have resulted from the epidemic rather than from war itself (Glei et al. 2019a). As a consequence, mortality follows the same age-pattern as in 1773, although the difference between children over 1 year and the other age groups is even greater.

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)). 1846 and 1882 were years ~~of~~ measles epidemic. In both years, particularly cold spring and summer forced fishermen to remain ashore in villages, 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, because of their physiological weakness. The epidemic lasted from July to December and caused the death of around 3% of the ~~whole~~ population. The individuals who survived were better prepared to face the following epidemic in 1882, which mostly affected ages under 50. The immunisation of the population also meant that this 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). Although the epidemic of 1882 was less deadly than the previous one, it was followed by another difficult year. In 1883 excessive ice off the coasts of Iceland lowered the temperature, leading to hunger and a cholera outbreak (Tomasson 1977), which slowed the recovery of the population.

The context of the Ukrainian famine of 1933 is more difficult to describe, as data was not public until the end of the Soviet Union and historians themselves do not agree on its causes and specific circumstances. Naumenko (2019) reports three main explanations to this famine. First, bad weather would have affected harvest yields in 1932, leading to an output lower than expected and to famine in the following months (Tauger 1991). Other researchers consider that the collectivisation policies that were implemented from the late 1920s affected output by imposing the type of crop to be sown and by increasing the weight of managerial and supervision personnel. 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 to widespread famine in the following years (as Naumenko herself maintains). Finally, a third strand of research argues that the famine was not caused by poor production, but rather 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 millions (Rudnytskyi et al. 2015). Although this famine affected other parts of the Soviet Union, it claimed the highest number of victims in Ukraine, only surpassed in relative terms by Kazakhstan.

3 Research questions

~~This text is structured around three research questions.~~

The first looks at the shape of mortality increase during the crises and at the associated lifespan variation trends. Following Zarulli's (2013) findings, we expect to see a proportional increase of mortality at adult ages, together with some convergence at older ages for the two Swedish and the Ukrainian crisis. Dribe et al. (2015) have also found that mortality increased disproportionally for children during the two Swedish crises, a pattern, which could also appear for Ukraine. In Iceland, we can expect to find two different patterns: for the first epidemic, a proportionately greater increase for children and the elderly; for the second a greater increase for the children only, as the elderly had already acquired immunity. In order to translate these patterns into expected changes of lifespan variation, we should consider the concept of threshold age. While mortality improvements typically decrease lifespan variation, an age can be calculated above which mortality improvements actually increase variation. This is age is known as the threshold age (Gillespie et al. 2014; Aburto et al. 2019; Zhang and Vaupel 2009). Thus, if we expect children and the elderly to be particularly affected, the opposing contributions of these two populations could balance each other out. However, even if the magnitude of the change is similar, children mortality should have a greater effect on measures of lifespan variation, as the survival function is necessarily greater at younger ages and life expectancy is usually longer. Thus, we expect lifespan variation to increase during a crisis and to gradually decline as its consequences continue to affect weakened individuals. However, a selection of the more robust individuals in the vulnerable ages, which was found in the Ukranian case by Zarulli (2013), could lead to a faster decrease of variation after the crisis. We will test these hypotheses by comparing a number of lifespan variation measures for period lifetables computed for the crisis year(s), as well as for the five years before and after.

Our second research question looks at gender differences. Zarulli et al. (2018) have found that the gender gap in life expectancy remains during high-mortality regimes. In the same way, 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 that differences in the time trends of lifespan variation will differ between sexes according to the gender-specificity of the crisis in question, for example with starker trends for females during the two measles epidemics.

Our final question ~~asks~~ whether some ages especially contributed to the change in variation during and after the crisis~~, and if so which ones~~. We predict that a mortality crisis will particularly affect children and the elderly, as these sub-groups are physically less equipped to deal with extreme conditions and because their survival might become less of a priority in situations where resources are scarce. 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, as those individuals who reach advanced ages were probably selected throughout their lives, their contribution might be smaller than that of young individuals.

4 Methods and data

4.1 Methods

Lifespan variation can be measured using a wide range of indices and all of them have been found to be highly correlated in empirical datasets when measured from young ages (Wilmoth and Horiuchi 1999). They are not, however, completely interchangeable. As van Raalte and Caswell (2013) point out, they differ in their formal properties and in the underlying concept they gauge. Following the authors' analysis, we have decided to include six measures of lifespan variation in this work: 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 (Shkolnikov et al. 2003). Each pair of measures is constituted by an absolute measure and its relative counterpart. For example, ~~using standard lifetable notation,~~ lifespan disparity can be expressed as

$$e^\dagger = \int_0^{+\infty} \mu(x)l(x)e(x)dx \tag{1}$$

while the lifetable entropy is

$$\bar{H} = \frac{e^\dagger}{e_0} = \frac{\int_0^{+\infty} \mu(x)l(x)e(x)dx}{e_0} \tag{2}$$

In a similar way, the standard deviation is

$$S_0 = \sqrt{\int_0^{+\infty} (x - e_0)^2 l(x)\mu(x)dx} \tag{3}$$

and the coefficient of variation

$$CV = \frac{S_0}{e_0} = \frac{\sqrt{\int_0^{+\infty} (x - e_0)^2 l(x)\mu(x)dx}}{e_0} \tag{4}$$

Finally, the absolute Gini coefficient is

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

and, for a lifetable radix of 1, its relative counterpart becomes

$$G_0 = \frac{G_0^a bs}{e_0} = \frac{e_0 - \frac{1}{[l_0]^2} * \int_0^{+\infty} [l_x]^2 dx}{e_0} = 1 - \frac{1}{e_0} \int_0^{+\infty} [l(x)]^2 dx \tag{6}$$

S_0 is the square root of the variance, which is itself the average squared distance from the mean age at death. Lifespan disparity expresses the remaining life expectancy at death and 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 can be understood as their absolute counterparts expressed as a proportion of life expectancy (i.e. the relative Gini coefficient is the average distance between each individual's age at death relative to e_0). $\bar{H}$ also allows a different interpretation, as a measure of the elasticity of life expectancy to a proportional change in mortality (Aburto et al. 2019; Wrycza et al. 2015). While the measures of absolute variation we have chosen are expressed in years, this is not the case for G and $\bar{H}$, which are both expressed as a proportion of life expectancy.

We use multiple relative and absolute 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 (i.e. the scale on which mortality progresses) and the shape (i.e. how rates change with age) as two separate dimensions (Wrycza et al. 2015), this approach has allowed comparisons between species with very different ilfe expectancies (Jones et al. 2014). Here, we use relative measures to compare human populations in starkly different circumstances, specifically in normal vs crisis times. Absolute measures of lifespan variation are inextricably tied with the length of life. For this reason, they can be more easily interpreted, as they are expressed in years, whereas relative measures are dimensionless. This feature becomes particularly convenient when interpreting the contributions of different groups to an overall change, as we will. Moreover, some research has suggested that a mix between relative and absolute measures of variation could better describe the conception of variation in some disciplines (Asada 2010), so that both kinds should be considered in research. Finally, by using both absolute and realtive measures, we can observe the differences in their ~~behaviours~~ under such extreme mortality circumstances.

In the second part of our analysis, we delve further into understanding the change in lifespan variation by decomposing it by age. In order to do so, we apply the continuous change or 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 whether certain age groups contributed to the increase or decrease in lifespan variation. The differences that may arise in these results depending on the measure considered could indicate different sensitivities to specific ages. 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 considering changes across time.

~~All of these analyses will highlight trends in the variation within the overall population. However, the data we use include information on gender-specific mortality. We use it to apply our measures to the male an female populations separately. This will not only permit us to analyse changes in the gender gap across time, but also to uncover potential differences in the gender-specific trends of lifespan variation and in the role of different ages in explaining them.~~

4.2 Data

We use two data sources. The first is the Human Mortality Database (HMD, www.mortality.org). It gathers several mortality-related data~~, common and~~ separated by sex, for multiple populations and periods, collected from each country's statistical office. We use single-age period lifetables (from 0 to 110+). With the oldest data series starting at the end of the XVIII century, it is one of the few data sources, which allows the study of mortality crises, while ensuring a high level of quality and immediate accessibility. In order to maintain data quality throughout the time-series, only countries with virtually complete death registration and censuses, and therefore mostly wealthy and highly industrialised, are included in the database. For this reason, we can only use HMD data to analyse mortality crises in Scandinavian countries, whose data series go further back than other nations.

More specifically, we use data for Sweden and Iceland, described by Andreeva and Dukhonov (2020) and Glei et al. (2019b). In these countries, data collection on vital statistics by parishes became compulsory by the middle of the XVIII century. These raw data were ~~cleaned~~ of obvious mistakes, but a number of issues remain, such as age heaping and age exaggeration. Moreover, in Sweden out-migration was not recorded, which might represent a bias for our analyses, as the crises might have pushed individuals to leave the country. This should not be an issue for Iceland, because of the rapidity of the crisis (leaving little time for migration) and the relative isolation of the island. Swedish data was collected in five-year age groups, which were then splitted into single-year ages. Mortality rates at older ages were also smoothed to correct excessive fluctuations and to separate data included into open age intervals. These procedures, however, caused some implausible patterns for these categories. Some of these concerns can be addressed by conducting sensitivity analyses using the raw data instead of the smoothed ~~one.~~ We also use 5-year age categories[1] instead of single age ones, to account for digit preference to some extent. We also reduced the number of age categories by creating an open-ended age class at age 80, instead of 110. This also allows to address the smoothed rates and the paucity of individuals alive at these ages. The other issues are more difficult to settle and should be kept in mind when interpreting results.

The second data source is the dataset compiled in the early 2000s by Meslé and Vallin

[1] With the exception of ages 0 to 4, which were divided in two: age 0 and ages 1-4.

(INED), containing period lifetables covering the Ukrainian population from 1926 to 1959 (aperiod later increased 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. This makes metadata all the more important. The relevant information has been published in a number of articles and books (e.g. Vallin et al. 2002 or Meslé and Vallin 2012), together with some additional information on Ukraine during this period. More specifically, the data at the origin of this dataset are likely to underreport actual deaths, especially during the crisis years. Moreover, there is little data concerning voluntary migration. 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 also grouped these data in 5-year age categories and set the open-ended group at age 80, instead of 89.

5 Results

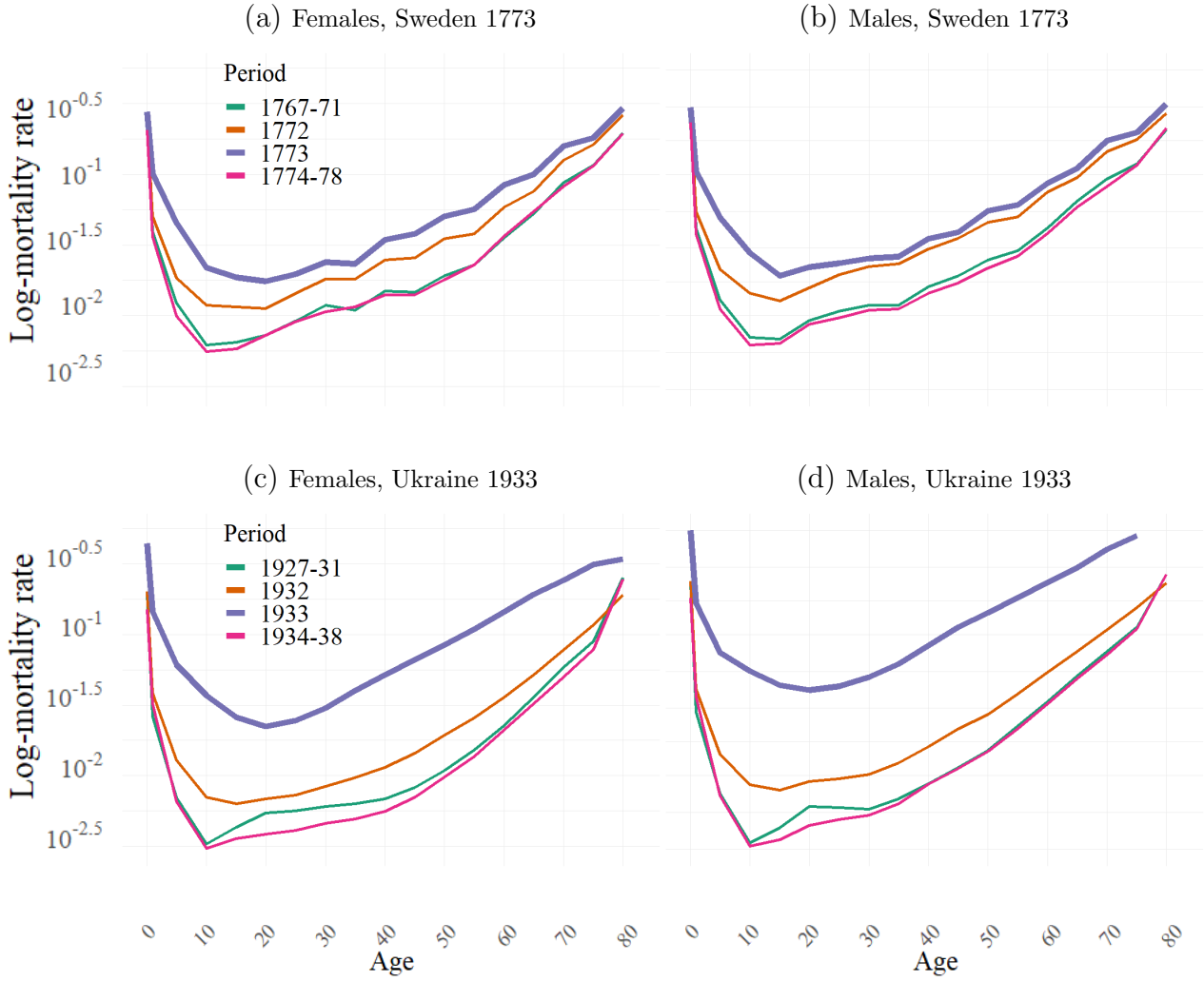
5.1 Age patterns of mortality

Figures 1 and 2 show log-mortality rates for each case study, for males and females separately. In each panel, we plot the crisis year(s) and the averages of the five years before and after the crisis.

Let us first consider our two famines in figure 1. In both cases, mortality rates seem to have 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's case~~ and for females in Ukraine~~'s~~ (as the survival function for Ukrainian males in 1933 reaches 0 at age 75, the rates stop short of age 80). Finally, sex differentials also behave similarly: not only are rates higher for males in all years, but they also increase more for males. Despite these similarities, some differences are also noticeable. The 1933 Ukrainian famine (bottom row) affected the population much more severly than the 1773 Swedish one (top row). Moreover, the first crisis year, when the famine was just starting, is much closer to the second crisis year for Sweden than for Ukraine, suggesting different temporal dynamics.

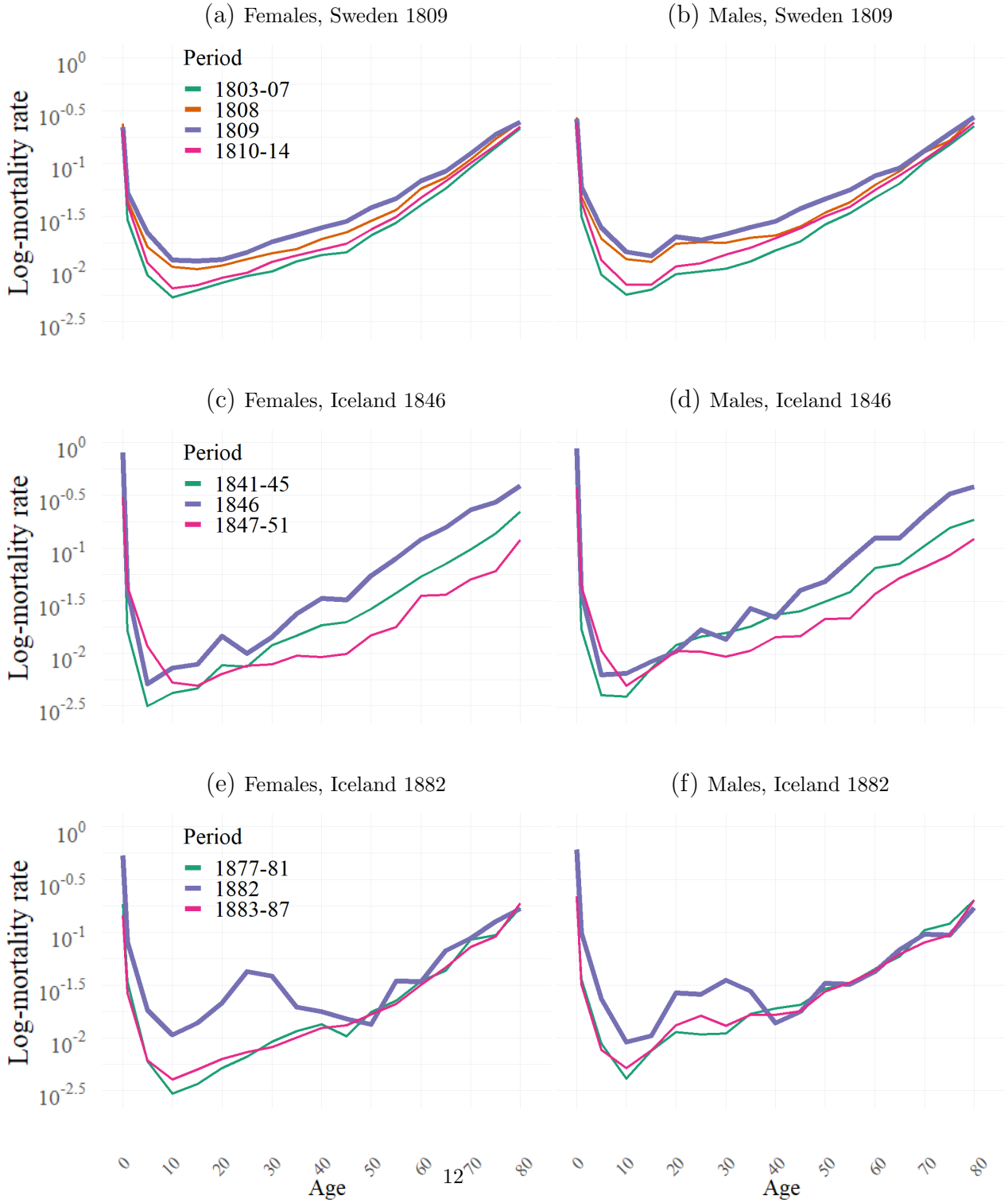
Figure 2 shows log-mortality rates for the three epidemics. Contrary to what happened for famines, each epidemic presents a distinct pattern of mortality, ~~highlighting their greater specificity~~. In 1809 infant mortality was not affected. Morevoer, male mortality is higher for all periods, but even more so in 1808-09 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 seems to be a greater increase in mortality among children and after age 45, a trend which is particularly visible for males, with no sign of convergence for older ages. However, in the years 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.

Figure 1: Log-mortality rates, famines



Source: HMD (Sweden) and Meslé & Vallin

Figure 2: Log-mortality rates, epidemics



Source: HMD (Sweden and Iceland)

More striking, however, is the complete convergence after age 40 for individuals who were immunised during the earlier epidemic. There is also a greater increase in infant and childhood rates compared to 1846 and a clear spike for young adults.

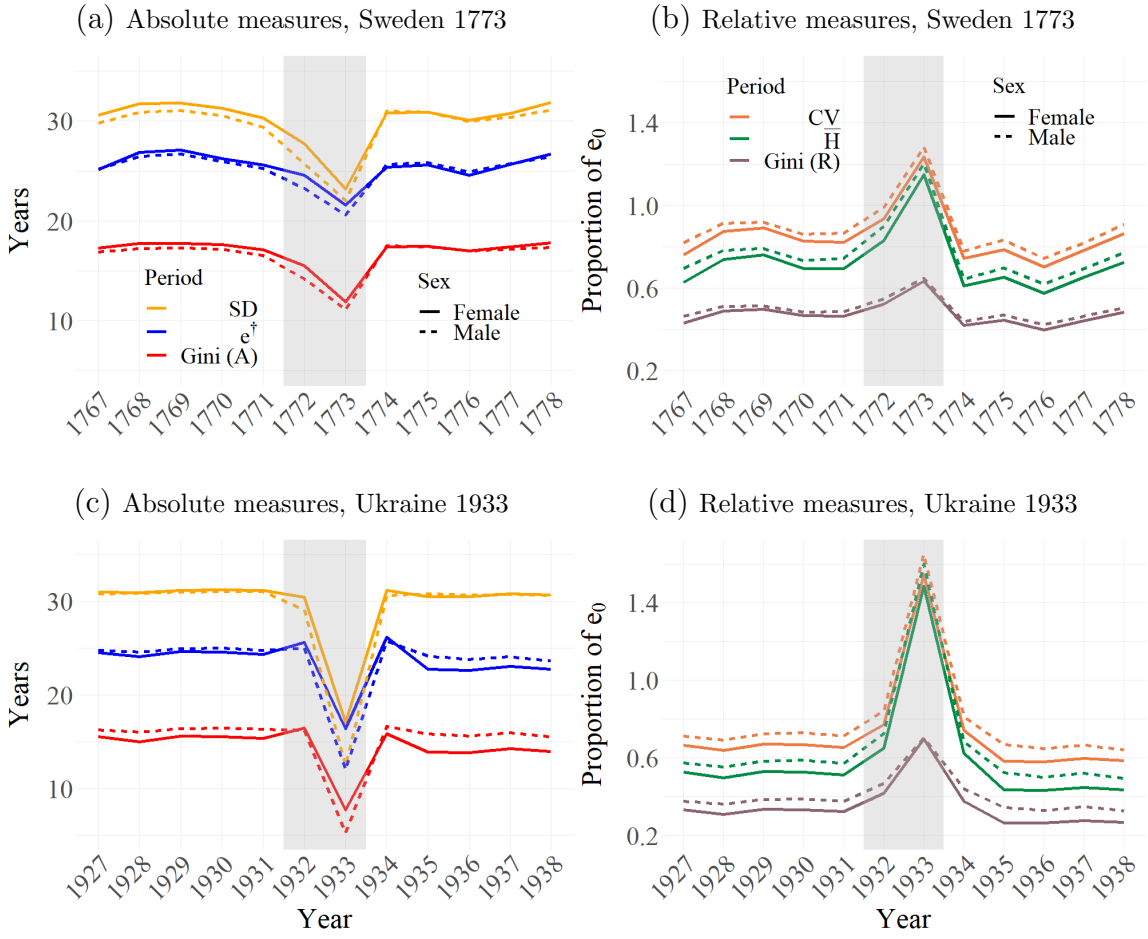
5.2 Trends of lifespan variation

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). The first striking feature, which reoccurs in all the graphs, is that absolute and relative variations present opposite dynamics: the former decreases during the crisis, while the latter goes up. A second persistent difference is that absoute variation is usually higher for females than for males, whereas the opposite is true for relative measures. Male absolute variability also changes more during a crisis, but this is not ususally the case for relative variation. Both kinds of measure, 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, when 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 A2).

We can also find some differences within the two groups 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.

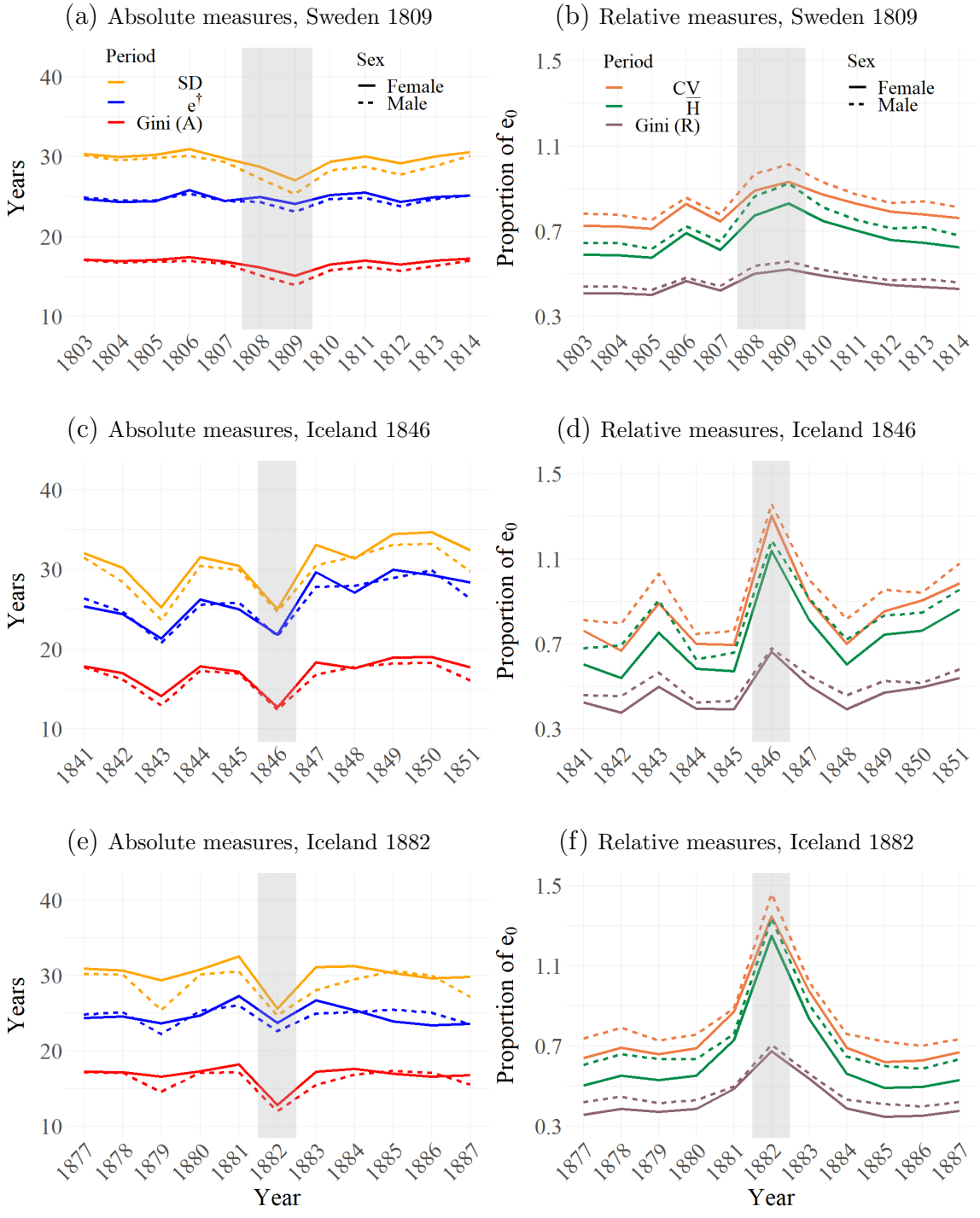
Out of the three cases where the crisis spans more than one year, absolute variation starts decreasing right away in only two. In Ukraine, there is a noticeable drop only in 1933, with the small exception of male SD. However, relative variability starts rising (albeit slightly) in 1932 aready, further 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 variability drops significantly, in fact reaching 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 varation is much lesser in 1843 and non-existent in 1879. Although these two years are not part of our current focus, an analysis of their circumstances could help shed light on the relationship between lifespan variation and life expectancy. Despite these particularities, however, the same partly unexpected trend holds true for all five crises: during these absolute lifespan variation declines, while relative lifespan variation increases.

Figure 3: Lifespan variation, famines



Source: HMD (Sweden) and Meslé & Vallin

Figure 4: Lifespan variation, epidemics



Source: HMD (Sweden and Iceland)

5.3 Decomposition of lifespan variation

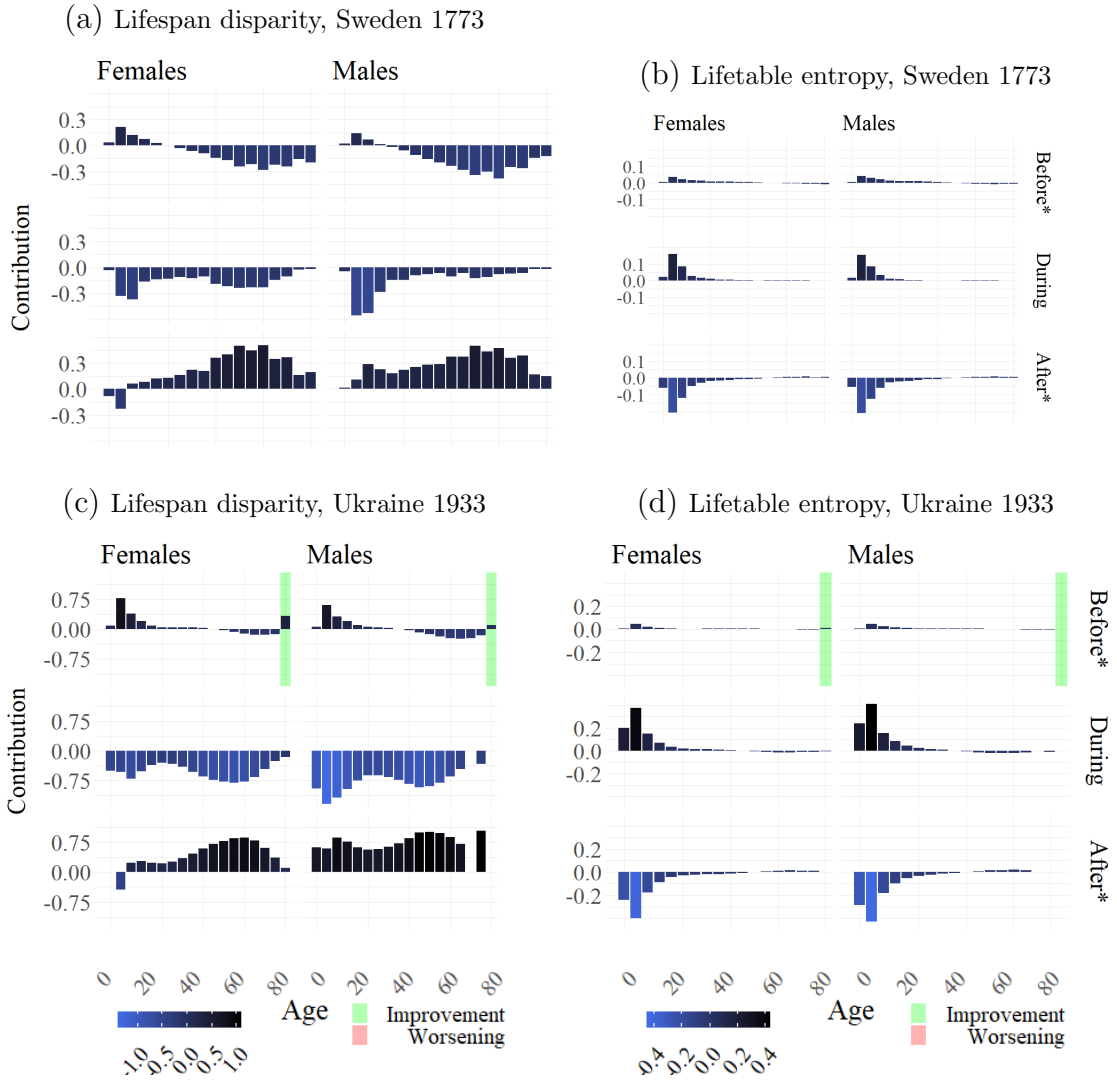
Figures 5 and 6 show the results of the decompositions for males and females. We have chosen to focus on two measures, $e^\dagger$ and $\bar{H}$, for conciseness, since the same trends appear for all absolute and relative measures. Each column indicates how the change in mortality experienced by the corresponding age group contributed to the overall change in the indicator, with the colour going from light blue for the smaller contributions to black for the highest. For example, the change in mortality female infants experienced between the period 1767-1771 and the beginning of the famine in 1772 contributed +0.034 years to the change in $e^\dagger 2$. 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 or following five years. We have shaded in green the ages where mortality unexpectedly improved, while it increased for all other ages. We have shaded in red the ages, where mortality increased, while declining for all others. Finally, to ensure readability, each graph has its own scale.

Comparing results for $e^\dagger$ and $\bar{H}$, we see that older ages influence very little the change in the latter, even when they contribute significantly to the change in the former. Examining our cases more in detail reveals some reoccurring trends. Our three nutrition-related crises (the two famines, plus the 1809 epidemic) see small contributions of infants to the change in both $e^\dagger$ and $\bar{H}$, especially when compared to older children. Contributions to $e^\dagger$ also decline after about age 60. The graphs for Ukraine and the Swedish epidemic both show unexpected improvements in mortality. In the first case, this could be due to bad data quality at older ages. In the second, this difference underlines how infants were protected from the epidemic. Since this crisis was the mildest of our collection, it is reasonable that it should have left infants more unaffected than the two famines.

The two Icelandic epidemics also show a number of unexpected changes in mortality, in both directions, which reflect patterns of mortality already visible in figures 1 and 2. The decomposition results also mirror age-specific patterns: significant contributions of infants and older adults in 1846, and of infants and younger adults in 1882. Finally, there are some interesting sex differences. In both cases, before the crisis female infants contribute positively to the change, while male infants contribute negatively or nothing at all, but the opposite is true after the crisis. While the uneven effect of measles can partly explain this pattern, it cannot do so fully. Instead, this result could show some shortcomings of the implementation of the Horiuchi decomposition. While the latter was developed in a continuous framework, it is carried out in a discrete one. This causes some imprecisions, which are exacerbate when the changes from one year to the other are particularly extreme, like in these cases. Thus, it is important, when interpreting these results, to keep the context in mind and distinguish plausible patterns from the others. More detailed data, for example describing causes of death, or a historian's persepctive would be needed to better analyse whether these could be actual patterns due to unaccounted for phenomena.

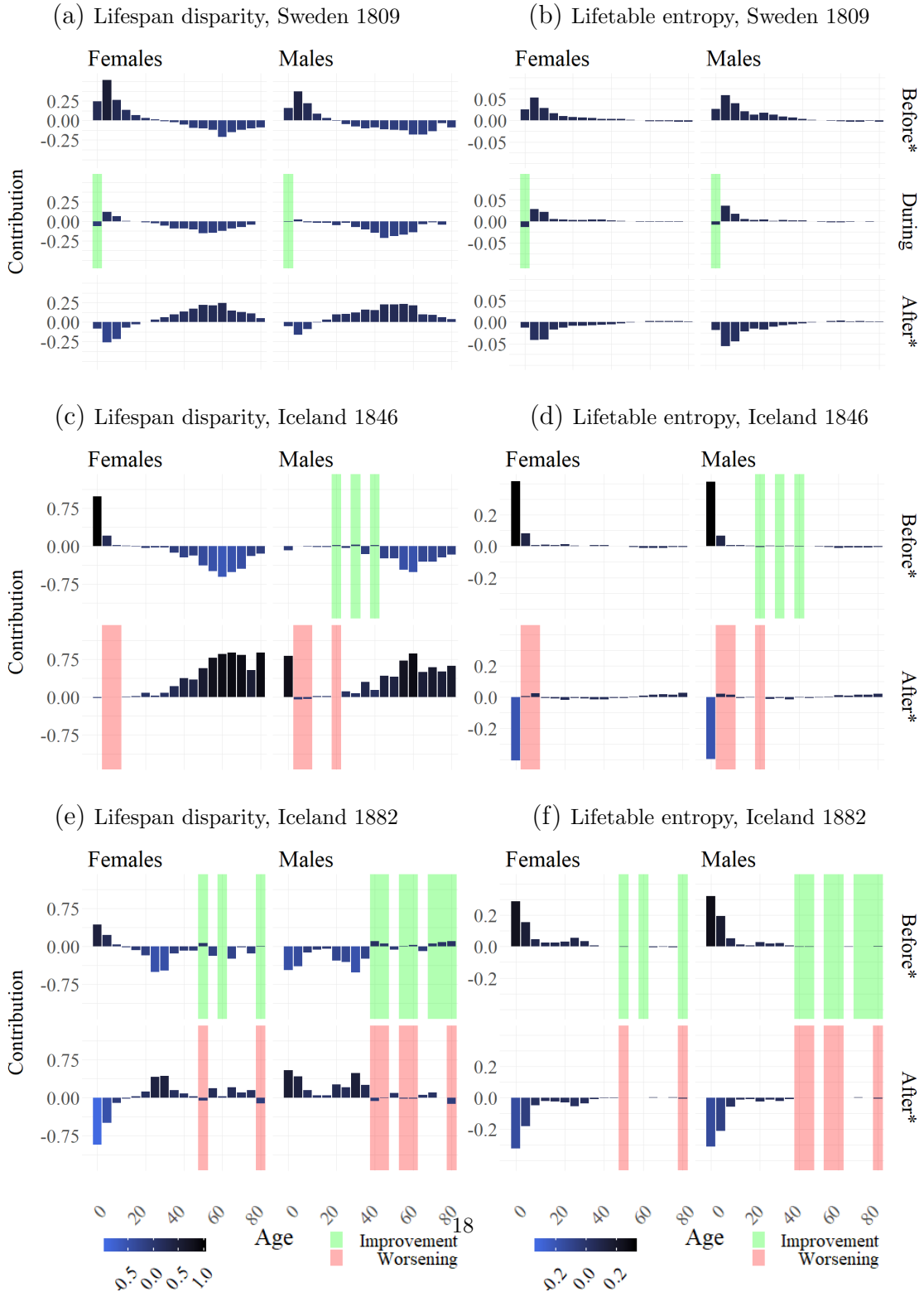
Figure 5: Lifespan variation decomposition, famines



* 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)

6 Discussion

The five mortality crises we study were characterised by an extreme and sudden drop in life expectancy at birth. In a similar context, 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 similar trends in our five cases. In fact, 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. This is not to say that all changes in mortality were proportional. However, deviating trends can be explained by the context in which the crisis happened and its characteristics, except for one. The lower increase of infant mortality for the two Swedish and the Ukrainian crises is consistent with the protective effect of brestfeeding against nutrition lack and nutrition-related diseases (Livi-Bacci 1990). Bengtsson et al. (2009) argues that in the past children, being net consumers of resources, might have held a precarious place in their household, becoming expendable in times of crisis. As this attitude changes across time and place, it could clarify the dispropotionate increase of child mortality in the Swedish famine, but not in the Ukrainian one. Moreover, the Ukrainian data have been extensively manipulated and smoothed, and might possibily be hiding a similar increase for children.

In interpreting the peculiar pattern of male rates for the 1809 Swedish epidemic, we should remember that this epidemic happened in the context of a war. Although mortality is thought to have mainly depended on the epidemic itself, the latter spread *via* troop movements, so that soldiers may have been more exposed. This also accounts for the different patterns in 1808 and 1809. In the first year, mortality rates for males after age 40 show no difference with post-crisis levels, while female mortality is significantly higher. Thus, before the epidemic spread to the whole population, males not directly involved in the war could have been less exposed than females, who took care of the sick.

The sex-specific patterns of the Icelandic epidemics are consistent with the literature showing a greater vulnerability of females to measles (Muenchhoff and Goulder 2014; Garenne 2015). 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 exposure to infectious diseases in early childhood affecting later mortality (Fridlizius 1989; Stoermer 2011), although a cohort study could better discriminate whether this is a stable pattern. The only unexplained trend is the spike in mortality experienced by young adults in 1882. It is unclear why these individuals should have disproportionally suffered from a disease which typically affects children and which showed no such pattern just 40 years before. It is unlikely that such a definite trend should be due to sheer variation and the explanation is rather to be searched in a specific behaviour exposing this age group.

Although these were not necessarily the trends we had expected, following the logic we detailed before they should still lead to an increase in lifespan variation. In the previous section, however, we have seen that this is not the case when using absolute measures. The relationship between life expectancy and lifespan variation has been studied using a range of absolute and relative measures, consistently revealing a negative association (Smits and Monden 2009; Colchero et al. 2016; van Raalte et al. 2011; Sasson 2016). However, our results add to the literature suggesting that this relationship is not so straightforward (Edwards and Tuljapurkar 2005b; Permanyer et al.

2018; van Raalte et al. 2014). In fact, while the usual relationship between lifespan variation and e_0 is monotonously and inversely proportional for all measures, at very low levels of e_0 absolute variation starts decreasing as well (as shown in figure A1). Permanyer and Shi (ming) find a similar plateau in absolute lifespan variation for very high levels of e_0. While this is true for absolute variation, the negative relationship between relative variation and e_0 holds at all levels of e_0, as we also see in our own results. Figure A2 shows that e_0 is more sensitive to the crisis than absolute variation. Since the denominator decreases more than the numerator, relative variation increases even as its absolute counterpart drops. Whilst Permanyer and Shi tie their plateau to a recent slowdown in e_0 gains, compared with gains in longevity, it is more difficult to adopt this framework in our case, because we consider mortality regimes which are only sustainable for an extremely short period, so that longevity is to a great extent determined by previous and drastically different mortality structures.

Decomposition results do give us some clarifying insight. Contrary to what we had expected, older ages contribute significantly to the change in $e^\dagger$. Since the threshold age for $e^\dagger$ ($a^\dagger$) trails e_0 closely, it was quite low in the historical periods we consider(table A1 shows threshold ages for our two measures, together with e_0 levels, with some imprecisions due to the transition from a continuous to a discrete framework). Thus, mortality increases even at relatively low ages contribute to decreasing $e^\dagger$ and their sum can offset the positive contribution of younger ages, although the magnitude of each contribution is often smaller for the former than for the latter. A similar mechanism explains the sudden increase of $e^\dagger$ from the crisis to the period afterwards. Zhang and Vaupel (2009) have shown that when $\bar{H}$ increases over 1, which consistently happens during our crisis years, $a^\dagger$ becomes 0. Because of this and because after the crisis mortality improvements are largely predominant for all of our cases, all ages contribute to an increase in $e^\dagger$, bringing it back to pre-crisis levels.

This does not happen for relative variation for two reasons. First of all, the threshold age for $\bar{H}$ (a^h), although not completely independent of e_0 trends, is much more stable than $a^\dagger$, so that it rarely drops below age 30 even during these crises (Aburto et al. 2019). Therefore, for mortality changes consistent across the ages, positive and negative contributions are more balanced. More importantly, we have seen that contributions to $\bar{H}$ changes are concentrated almost exclusively at young ages. Decomposition results for e_0 (figure A3) show that young ages' contributions are greater than for $e^\dagger$, while older ages contribute about the same to both measures. Relative variation being the ratio between absolute variation and e_0, a disproportionate change in the denominator is bound to accentuate the contribution of young ages, while at older ages the ratio remains constant. Therefore, while changes in relative variation are, as we had expected, led by mortality at young ages, changes in absolute varaition 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.

We would like to examine three additional results before going on to our concluding remarks. Figures 3 and 4 show that absolute variation tends to decrease less for females than for males. This trend highlights that males suffer more from mortality crises in terms of e_0 (**?**), but that their 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. This is mostly due to

differences in the weight of young ages in the decomposition results (although the magnitude of the contribution of older ages also differs across sexes): it is lower for males than for females before the crisis, while the opposite is true for the other periods. For $\bar{H}$ child mortality often contributes more to the change for males, although the difference is less pronounced. An exception to these trends are the two Icelandic epidemics, where lifespan variation change was equal across sexes or more marked for females. These latter differences seem again to be mostly explained by differences in the contribution of young ages (although as noted before we should consider these results carefully). Thus, despite the role of older ages in leading the direction of absolute variation change, young ages determine sex differences in this change for both absolute and relative measures.

Not only do the crises affect males and females differently, but the gender gap is switched between absolute and relative measures. While the latter show higher variation for males, as expected, in the former males are usually advantaged, during the crises and in normal years. Studies which have found higher variation for males have used both relative and absolute measures, so that this difference cannot lie in the indicator used. It would be interesting to study changes across time in the gender gap for lifespan variation, to understand whether the difference we see today developed in relatively recent times, as is thought to have happened for e_0 (Klasen 1998; Goldin and Lleras-Muney 2019). This could illuminate whether this gap is mainly determined by cultural or biological causes.

Since we use measures based on period lifetables and since the crises we study are necessarily short-lived, it is difficult to derive implications for individuals from the behaviour of lifespan variation during the crises themselves. However, its behaviour after the crises is interesting in this respect. The distribution of mortality is very quickly restored to pre-crisis levels, together with e_0. Thus, there seems to be no medium-term effect of crises at the individual or population level, at least as far as period measures are concerned.

7 Conclusion

In these analyses, we have found various patterns of mortality change. However, all of our cases show the same trend of lifespan variation: a drop in absolute variation, together with an increase in relative variation, a difference which is clarified by the contrasting weights of mortality change at young and older ages, together with the role of the threshold age for each kind of measure.

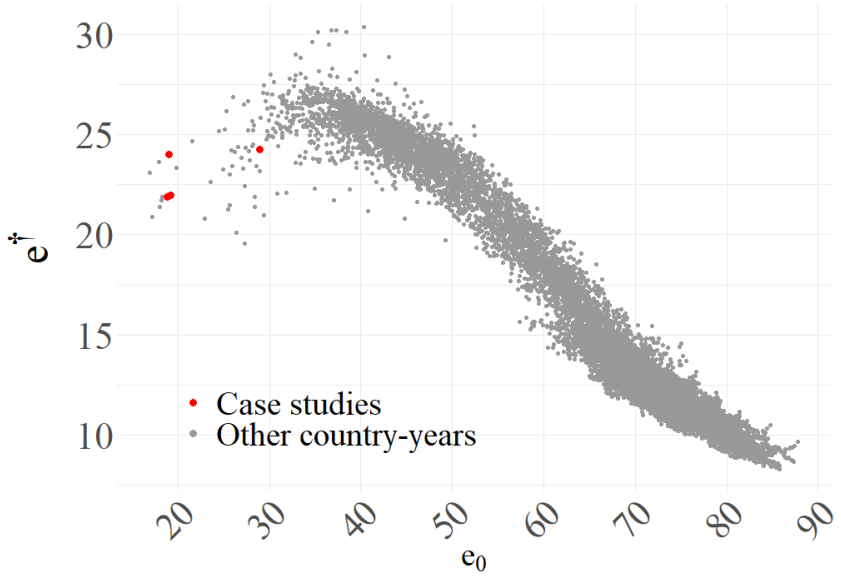
This text aims to contribute to the understanding of lifespan variation and mortality crises in three ways. First of all, it shows a common trend of lifespan variation for all five crises examined. This can advance the comprehension of human mortality under extreme conditions. Analyses into "other cause" crises could show whether this pattern is more widely true. Secondly, we have shown that the effect of crises on lifespan variation differs across sexes,which is mostly due to discrepancies in the contribution of young ages. Moreover, we have found evidence suggesting that the gender gap may not have always been in favour of females, depending on the measure used to capture variation. Finally, by comparing different kinds of measures, we hope to have given new insight into their characteristics and into the nature of their behaviours, so as to fruitfully contribute to the debate around the use of absolute or relative measures of inequality.

Naturally, this study also presents some limitaitons. A number of factors separates the results of our analyses from being directly applicable to modern mortality crises. Data quality is not necessarily assured, here in the case of Ukraine. Even when of good quality, the data rarely provide information on social status of geographic location, for example, which could help refine these results (Zoraster 2010). Finally, although we provide general patterns for such historic populations, these are not necessarily extendable to modern populations, characterised by older 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 studies looking at lifespan variation during contemporary crises, to understand if a regular pattern emerges, which trascends time. Methodologically, the discrete implementation of the decomposition analysis leads to some imprecisions, which can be exacerbated by extreme mortality change. However, to our knowledge more appropriate methods are not available and the interpretation of results should rely on the researcher's good sense.

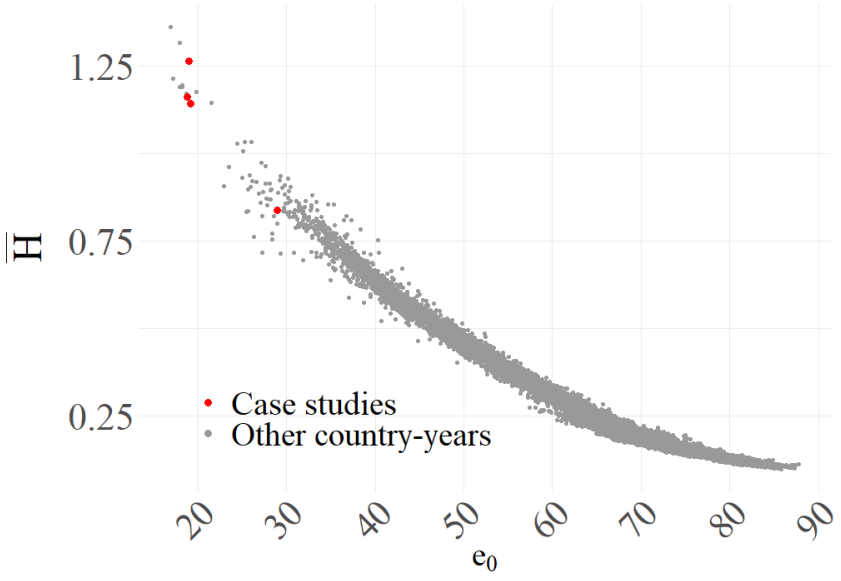
Appendix A

Figure A1: e_0 and lifespan variation

(a) e_0 and $e^\dagger$

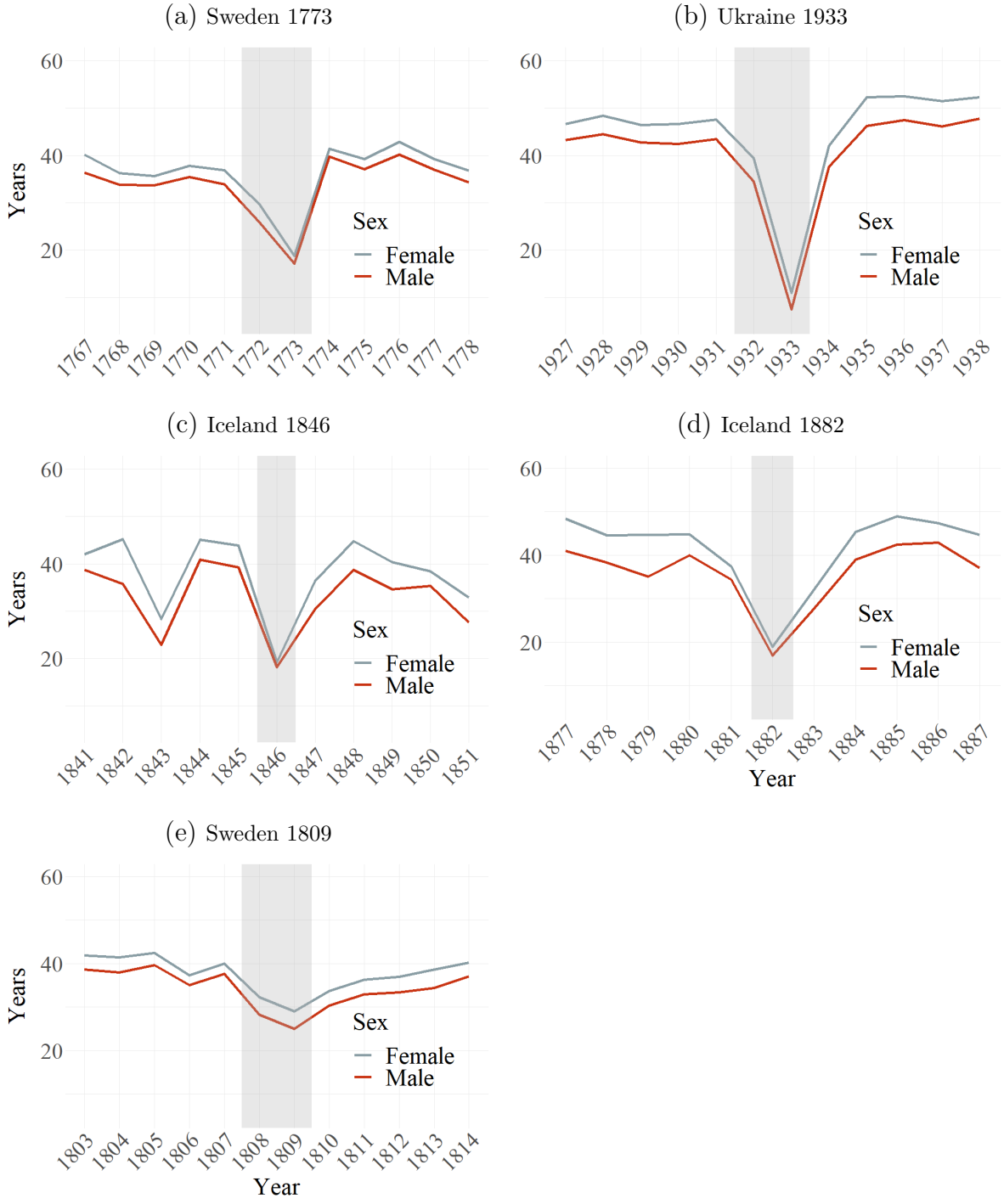


(b) e_0 and $\bar{H}$



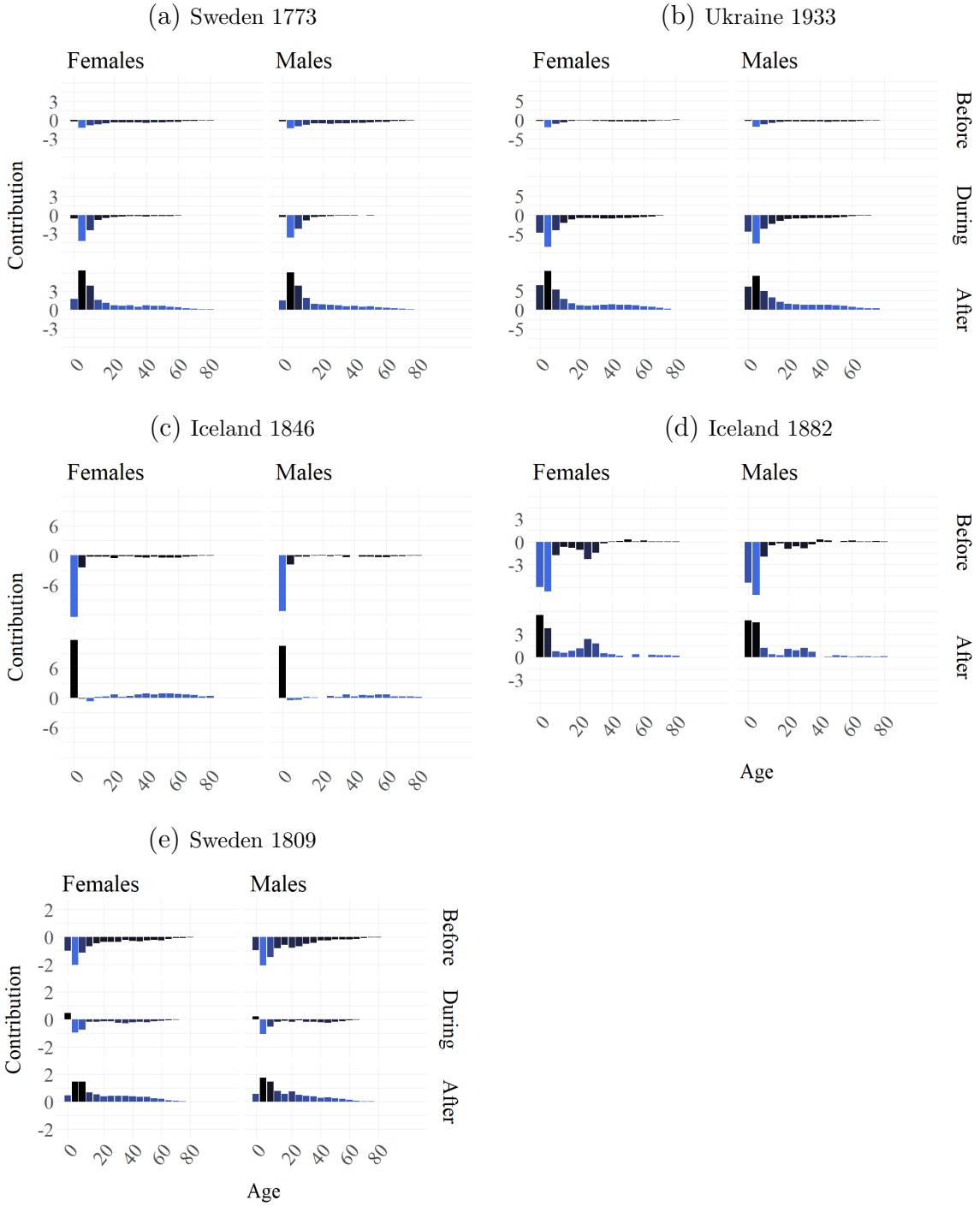
Source: HMD, all countries

Figure A2: Trends of e_0



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

Figure A3: Decomposition of e_0



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

Table A1: e_0 and threshold ages for $e^\dagger$ and $\bar{H}$

Measure	**Sweden 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

Measure	**Ukraine 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

Measure	**Sweden 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

Measure	**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

Measure	**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

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