Abstract

1 Introduction

Life expectancy and its variants (such as healthy life expectancy) are 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 is ~~indeed very~~ convenient, as it expresses the average remaining years of life in a given year (Preston et al. 2001). However, life expectancy can camouflage other important characteristics of a population. One of such characteristics is the variation in the age at death, also known as lifespan variation. Lifespan variation, which describes the 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 ~~historical and contemporary~~ 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). ~~Therefore, by only focusing on life expectancy, we miss a fundamental inequality in age at death.~~ ~~Indeed,~~ 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). In fact, although individuals may be unaware of mortality statistics, they experience age at death variability through the deaths of relatives and friends and derive inferences which follow known trends of inequality, even realistically weighting the imact of unhealthy behaviour in some cases (Hurd and McGarry 1995; Dormont et al. 2018).

Studies on lifespan variation have mostly focused on populations with continued improvements in mortality or analysed differences by social determinants, such as SES or educational level (Edwards and Tuljapurkar 2005; Lariscy et al. 2016;Permanyer et al. 2018; van Raalte et al. 2011). More recently, some studies have started shedding light on the pattern 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, this 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. A number of factors separates the results of our analyses from being directly applicable to modern mortality crises. This pandemic and many other contemporary crises have milder effects than the ones we examine here. Moreover, an interesting factor to consider today would be the potential effect of socioeconomic status, which has been shown to matter even under extreme circumstances (Zoraster 2010). Given the data at our disposal, we cannot examine this aspect of mortality crises. However, our results could still 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.

This text is divided in four sections. First, we review some trends which have already been highlighted by the literature on lifespan variation and mortality crises and we briefly delineates the contexts of the mortality crises, which represent our case studies, by specifying their place in time and space as well as the causes identified by historical research. The second section presents the research questions which guide our analyses, while in the third we describe the data and methods used. In our last section, we present our main results and we conclude by taking stock of our analyses and results.

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 also highlighted for non-human primate populations by Colchero et al. (2016), who have calculated that when comparing two primate (human or non-human) 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. In fact, while a negative relationship between life expectancy and lifespan variation has been repeatedly found for primates, in many species these two measures remain independent or only weakly correlated (Jones et al. 2014; Baudisch et al. 2013). In human populations themselves, 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 mortallity changes occur (Aburto and van Raalte 2018; García and Aburto 2019;Aburto et al. 2020). Even when there is a correlation between life expectancy and a measure of lifespan variation, the former does not completely predict the latter (Van Raalte et al. 2011). ~~These latest results underline the importance of studying life expectancy and lifespan variation as two distinct characteristics of a population.~~

As patterns of lifespan variation have been studied before, so have mortality patterns during times of crises. The definition of what constituted 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 ~~(as indeed happens most of the time, since individuals rarely die of hunger alone).~~ Finally, crises can have other causes, such as wars or natural phenomena. These latter usually present ~~very~~ specific dynamics, so it might be difficult to compare them with the other two. 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 decreases drastically and quite suddenly. For the purposes of this article, we also 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 pattern can be expected, as age and gender specific mortality 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 "harvested" the weaker individuals, leaving only the most robust behind. However, age and gender specific mortality patterns can 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 (of Health and Welfare 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 traditionally affects children, but also non-immunised adults, common in isolated communities previously spared by the virus. Regarding gender differences, measles typically take a greater toll among female children in terms of mortality, although females usually suffer lower prevalence and severity from viral infections (Muenchhoff and Goulder 2014; Garenne 2015).The last crisis we analyse was a typhus and dysentery epidemic, 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).

Now that we have given a general outlook ~~at~~ the trends of mortality in famines and epidemics, let us consider our specific cases. We consider five 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. After reviewing some of the work, which has been done on famines and epidemics in general, we will briefly outline the context of each of our cases.

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, compared to the others and crude death rates doubled in central Sweden. Although all age-groups were affected, 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 epidemics 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)). In 1846 and 1882 measles were the cause. 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. ~~Indeed,~~ in 1883 excessive ice off the coasts of Iceland lowered the temperature, leading to famine 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 these 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 depending on estimates~~ (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

Zarulli (2013) presents three possible effects of a mortality shocks on an adult population's age-specific ~~log-mortality curve~~. The shock can affect all ages proportionally, thus shifting the curve upwards, without notable modifications of the slope ~~(in agreement with Vaupel's hypothesis of a biologically determined rate of ageing)~~. Otherwise, mortality could increase proportionally more for weaker age groups, or cause the same additive increase for all ages. In both cases, the slope would change: in the first, the slope should become steeper, as older individuals' mortality grows disproportionately; in the second, the slope should decrease, as the same additive increase to mortality rates would cause greater proportional increases at younger ages, thus changing the slope at the logarithmic level. In this paper, Zarulli finds evidence in support of the first hypothesis for two mortality shocks: a Japanese internment camp during the II World War and the 1933 Ukrainian famine, in this second case also finding indications of a selection of robust individuals at the older ages.

Based on the findings we have reviewed, which pattern of age-specific mortality can we predict to find in our five cases? We can of course expect results similar to Zarulli's for the 1933 Ukrainian famine, although, as we consider the total, and not just the adult~~,~~ population, we would also have to take into consideration the smaller proportional increase at young ages. The 1773 Swedish famine was also severe and spanned a similar time framework. However, while we know that mortality in this case was mostly due to typhus and dysentery, we could not ascertain the specific causes of death in Ukraine. Thus, these two crises could still present different patterns. In fact, as Dribe et al. (2015) have found, mortality increased proportionally much more for children than all other ages for our two Swedish crises. 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. Thus, we can expect to find three patterns of mortality: a proportional increase of mortality at all adult ages, with smaller increases for very young and old ages for Ukraine; a proportionally greater increase for children in the two Swedish crises and for the second Icelandic epidemic; a proportionately greater increase at the extremes of the age distribution for the first Icelandic epidemic.

In order to translate these patterns into expected changes of lifespan variation, we should keep in mind that changes in mortality at each age do not affect lifespan variation equally. First of all, increased mortality at younger ages, i.e. below the threshold age, increases lifespan variation, while increased mortality over this age has the opposite effect. 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 can be expected to have a greater effect on measures of lifespan variation, since the lifetable population is necessarily greater at younger ages than at older ones. Moreover, children typically have a longer life expectancy and their age at death is usually more distant from the average than that of older individuals, thus further increasing their weight (although this might not necessarily be the case during mortality crises). Therefore, we can expect to see an increase of lifespan variation for the Icelandic and Swedish crises. In the case of Ukraine, it is more difficult to make a prediction. Indeed, Zarulli's (2013) log-mortality distributions show a greater relative increase in the middle of the age-distribution, leading to a slightly flatter log-mortality distribution. However, the additive change in mortality rates was greater for the elderly. If this increase ware greater enough, it could offset the increase at the other ages, thus leading to a decrease in lifespan variation. However, it seems unlikely that this could be the case, since the population, subjected to the previous mortality would have necessarily dwindled by the time it arrives at these specific ages, therefore giving them less weight.In conclusion, we expect that lifespan variation will increase for each of our crises. We also expect that lifespan variation will decrease gradually to usual levels, as the consequences of the crisis continue to affect weakened individuals. However, this trend could be balanced by a selection, during the crisis, of the more robust individuals in the vulnerable ages, who would experience lower mortality than their frailer counterparts, as was found in the Ukranian by Zarulli (2013). 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) find that the gender gap in lifespan variation, which generally favours females (van Raalte 2011), does not change in high mortality situations. Accordingly, 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-specifcity 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 techniques 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, 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, when the lifetable radix is 1, its relative counterpart becomes

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

We have chosen to use multiple measures and to consider both relative and absolute variation for three main reasons. First of all, by using different measures, we can identify the patterns identified by all (or most) of them as more robust, compared with results obtained using a single indicator. Secondly, relative and absolute measures of variation each have their own advantages. Relative measures were brought to the forefront by 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 different species Jones et al. 2014). Here, we use relative measures to compare human populations in starkly different circumstances, specifically in normal vs crisis times. Thus, relative variation can highlight changes beyond modifications of life expectancy. On the contrary, absolute measures of lifespan variation are inextricably tied with the length of life. For this reason, absolute measures of variation can be more easily interpreted, as they are expressed in years, whereas relative measures of variation are dimensionless. This feature becomes particularly convenient when faced with the prospect of interpreting the contributions of different groups to an overall change, as we will do in the decomposition part of our analysis. Moreover, some research has suggested that a mix between relative and absolute measures of variation could be a better fit for the conception of variation, at least 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, ~~which might give us futher insight in their properties.~~

~~We also prefer these specific indicators to other measures, because they are easily interpretable.~~ 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 life years lost at death. 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 coefficient of variation is the average squared distance from the mean 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.

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 $c_i = \int_{x_i 1}^{x_i 2} \frac{\partial y}{\partial x_i 1} dx_i$ 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 $x_i 1$ and $x_i 2$ 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 especially 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 as we do.

All of these analyses will highlight trends in the variation within the overall population, however, the data we use, which we will describe in the next part of this section, include information on gender-specific mortality. We use these 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). This open-use dataset was developed by the University of Berkeley and the MPIDR in Rostock (Germany) and was launched in 2002. 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. Together with the selction of data sources, other procedures of the HMD protocol, such as comparing newly included data with other available sources and applying uniform methods to all country-periods in order to avoid the introduction of further biases (Wilmoth et al. 2019), ensure a high level of data quality.

More specifically, we use HMD for crises in 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, records which represent the

~~basis of the HMD data for these periods.~~ These raw data were cleaned of obvious mistakes, but a number of issues remain. We can observe 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 (although some heaping is still visible). 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 (INED), containing period lifetables covering the ~~whole~~ Ukrainian population from 1927 to 1939, 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 (e.g. censuses) using forward and backward projections. This makes metadata all the more important. Such a document is not available with the dataset, but the relevant information has been published in a number of articles and books (e.g. Vallin et al. 2002), 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 compensate for this lack by assuming 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 (**?**), 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 and discussion

This section will be divided in three parts. First, we will briefly analyse the age patterns of log-mortality rates in our five case studies (figures 1 and 2), to underline some overall and some more specific trends. Then, we will look at the behaviour of our lifespan variation indicators (figures 3 and 4) and, finally, we will examine the results of the age-decomposition, focusing on two measures: lifespan variation $e^{\dagger}$ and lifetable entropy $\bar{H}$ (figures 5 and 6).

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

5.1 Age patterns of mortality

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

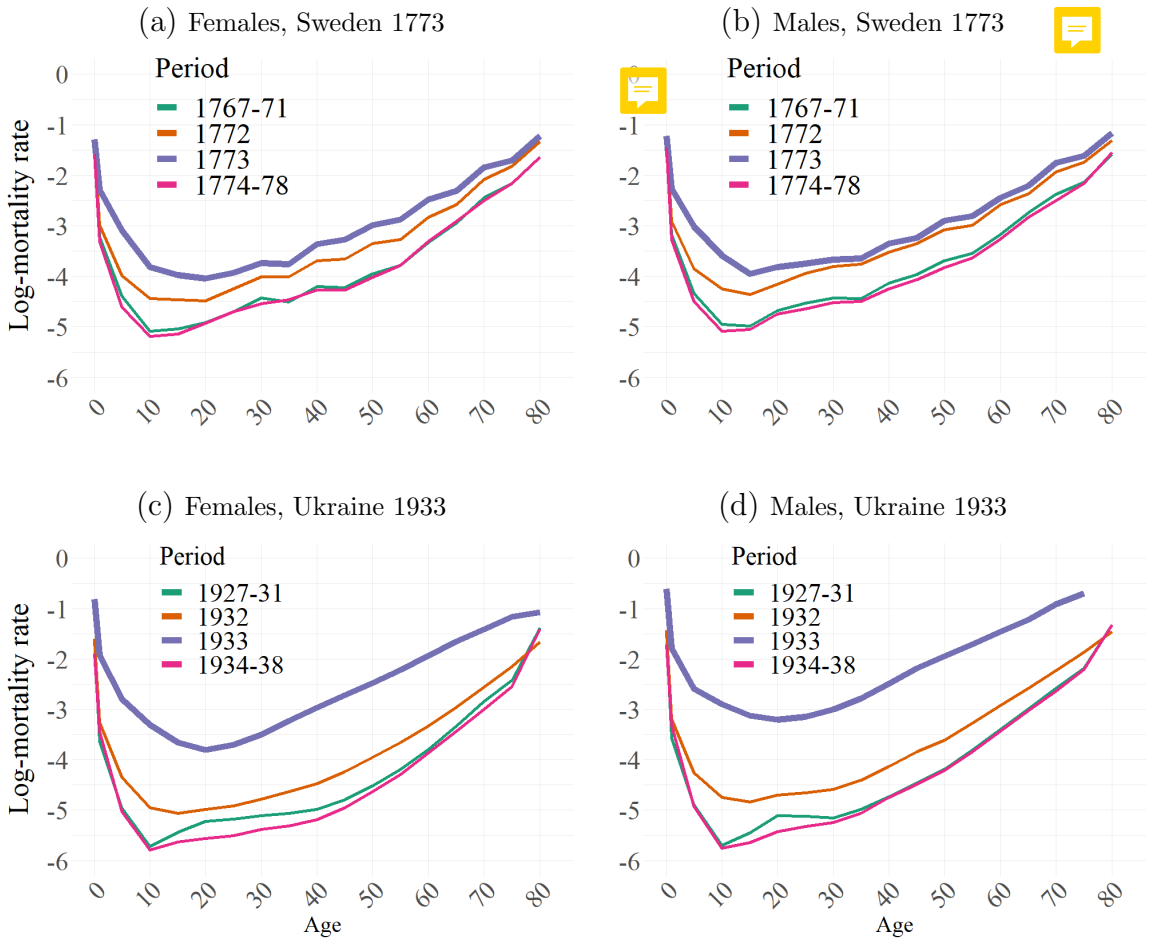
Let us first consider our two famines in figure 1. In both cases, mortality rates seem to have increased proportionally at adult ages, confirming the findings by **?**. The increase of mortality is smaller at age 0, which is consistent with the protective effect of breastfeeding. There is also evidence 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). This result is also consistent with the possibility of a selection of the more robust individuals during the crisis. Finally, sex differentials also behave similarly: not only are rates higher for males in all years, but they also increase more for males. Thus, both famines support a female advantage during mortality crises.

Despite these similarities across the two famines, 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. On theo ne hand, this could be due to the fact that the Swedish famine did not affect the entirety of the country severely and, on the other, that the Ukrainian famine might have been made worse by the central government response to the lack of food. Secondly, the increase in mortality seems to have especially affected children in Sweden, while this is not the case in Ukraine. Bengtsson et al. (2009) explains that non-productive children may have had a rather precarious place in households of the past, becoming expendable in times of crisis. However, this changes depending on the place and time and could have been true for Sweden in 1773, but not for Ukraine in 1933. Morevoer, the Ukrainian data have been extensively manipulated and smoothed, and might possibily be hiding a similar increase for children.

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. First of all, in 1809, infant mortality was not affected by the crisis. Again, this could be due to breast-feeding, which protects from the nutrion-related diseases active during this crisis (Livi-Bacci 1990). There are also some clear sex differences. Male mortality rates are higher for all periods, but even more so in 1808-09 for ages 15 to 40. We should remember that ths 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. Finally, there is a clear convergence at older ages, further supporting the possibility of selection.

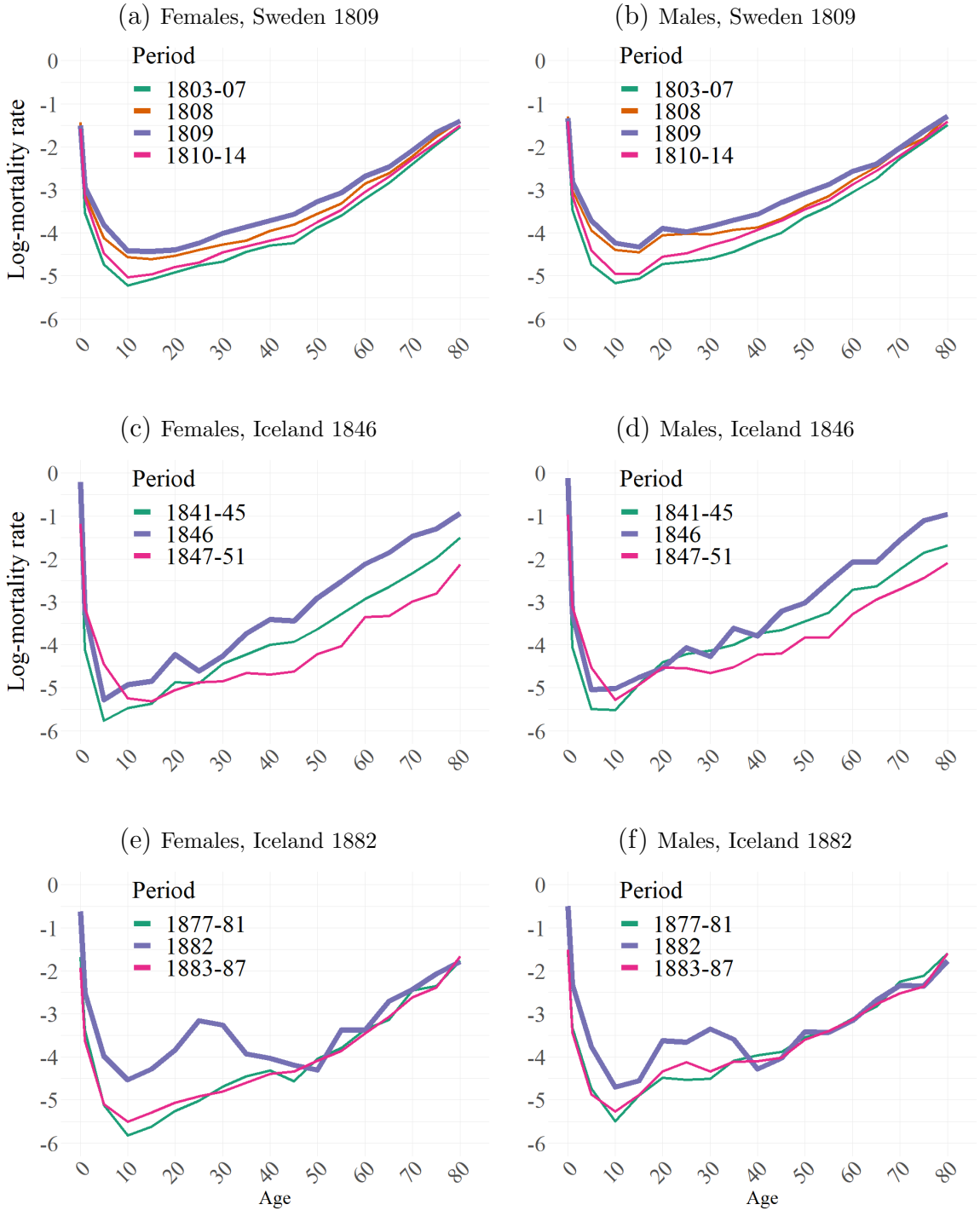
The two Icelandic epidemics show greater variation, as can be expected from a smaller population. Thus, patterns should be considered with care, especially when they are seemingly idyosincratic. With this in mind, for the 1846 epidemic there seems to be a greater increase in

Figure 1: Log-mortality rates, famines



Source: HMD (Sweden) and Meslé & Vallin

Figure 2: Log-mortality rates, epidemics



Source: HMD (Sweden and Iceland)

mortality among children and after age 45, a trend which is particularly visible for males. These are the ages which should be more vulnerable to measles. At these ages, however, mortality rates seem to have increased proportionally, with little evidence of convergence even at the oldest ages. As Zarulli (2013) notes, a crisis lasting a single year might not be enough for selection to take effect. However, in the years after the crisis, mortality rates for these ages dropped under pre-crisis levels, suggesting a harvesting effect during the epidemic, which affected all adult ages. On the contrary, child mortality is higher even than during the crisis itself. This increase could be tied with exposure to infectious diseases in early childhood affecting later mortality, a connection which might be stronger for males (Fridlizius 1989; Stoermer 2011), although a cohort study could better discriminate whether this is a stable pattern. Finally, female mortality during the crisis is higher than the males', although before and after the crisis the opposite is true. This is consistent with the literature which has found a greater vulnerability of females to measles (Muenchhoff and Goulder 2014; Garenne 2015).

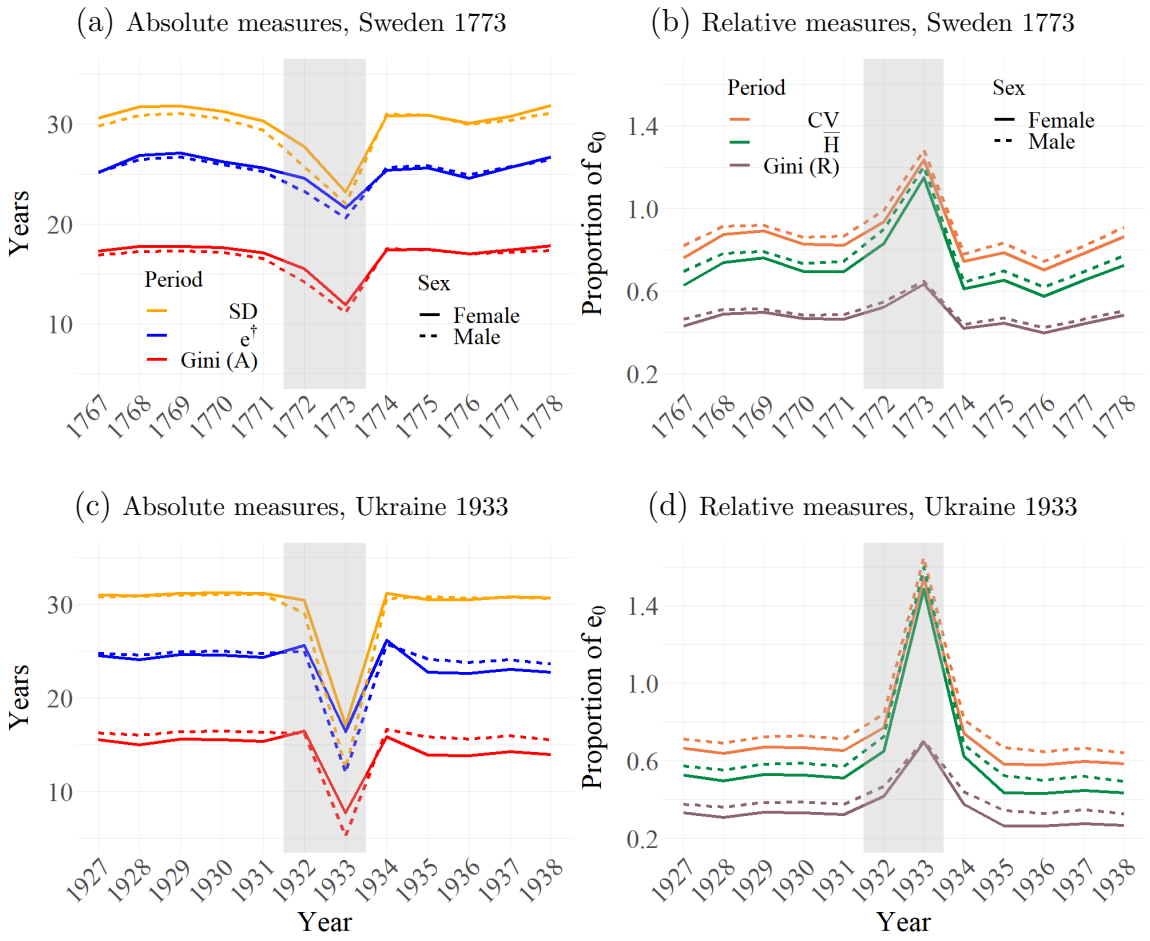
The 1882 epidemic again affected females more than males. More striking, however, is the complete convergence after age 40, for individuals who were immunised during the earlier epidemic and, thus, remained unscathed by this one. There is also a greater increase in infant and childhood rates compared to 1846 and a clear spike for young adults. It is unclear why these latter individuals should have disproportionally suffered from a disease which typically affects children and which showed no such pattern just 40 years before. Still, 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.

In conclusion, the increase in age-specific mortality rates seems to have been proportional with a convergence at older ages, with different trends usually explained by the social or epidemiological context. Because of the variations in the latter, we have found three main patterns. The two famines, together with the 1809 epidemic (also nutrition-related) saw an increase in mortality which was attenuated for infants and for older ages. In 1773, there is also a slightly greater increase for children, while the 1809 epidemic sees the greatest increase for young adult males. In 1846 mortality increased especially for children and older adutls, notably for males. In 1882, finally, the increase is circumscribed to ages smaller than 40, with a clear, although surprising, peak for young adults. These were not necessarily the trends we had expected, however their consequences in terms of lifespan variation does not change. As an equal increase in rates holds more weght at younger ages than at older ones, we expect an increase in lifespan variation for all case studies, driven by increased child mortality. However, this increase could ld be attenuated by the peak in adult mortality for 1882 and, to a lesser extent, for males in 1809.

5.2 Trends of lifespan variation

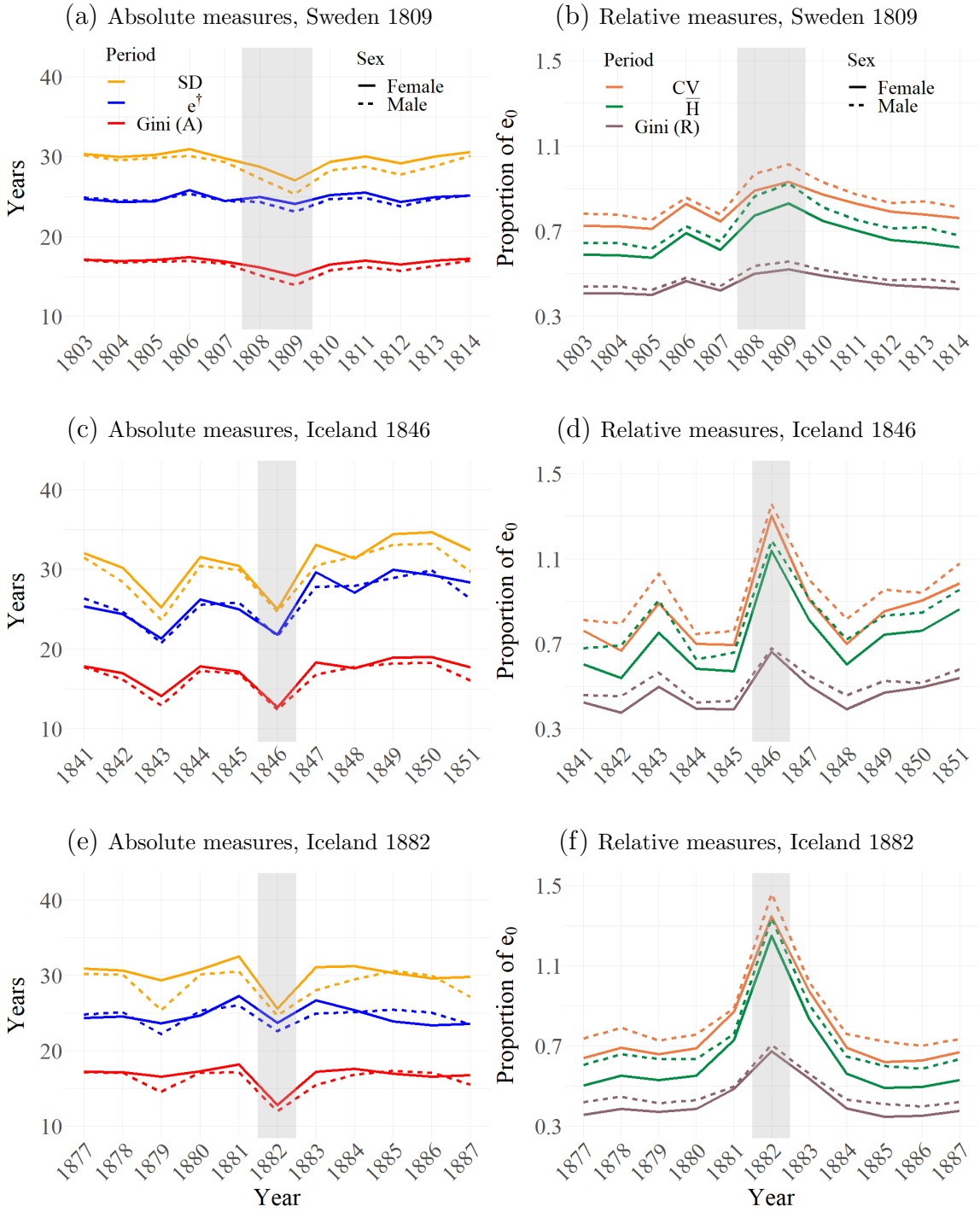
In figures 3 and 4, we examine how absolute and relative lifespan variation measures behave in our five cases (mark that in this case, each non-crisis year is handled separately and not averaged like in the previous section). The first striking feature, which reoccurs in all the graphs, is that absolute and relative variations present opposite dynamics: the former decreases during the crisis, while the latter goes up. 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 life expectancy absolute variation

Figure 3: Lifespan variation, famines



Source: HMD (Sweden) and Meslé & Vallin

Figure 4: Lifespan variation, epidemics



Source: HMD (Sweden and Iceland)

starts decreasing as well (as shown in figure A1 and examined further in IÑAKI'S PAPER). When life expectancy decreases under a certain limit, there are fewer years to be lived by everyone, so that we actually witness a compression of mortality. On the contrary, relative variation keeps rising even at such extreme levels of life expectancy. Indeed, while absolute variation is independent, at least by definition, from e_0, relative variation is instrinsically linked to it. These diverging patterns, then, show that e_0 drops proportionally more than absolute 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).

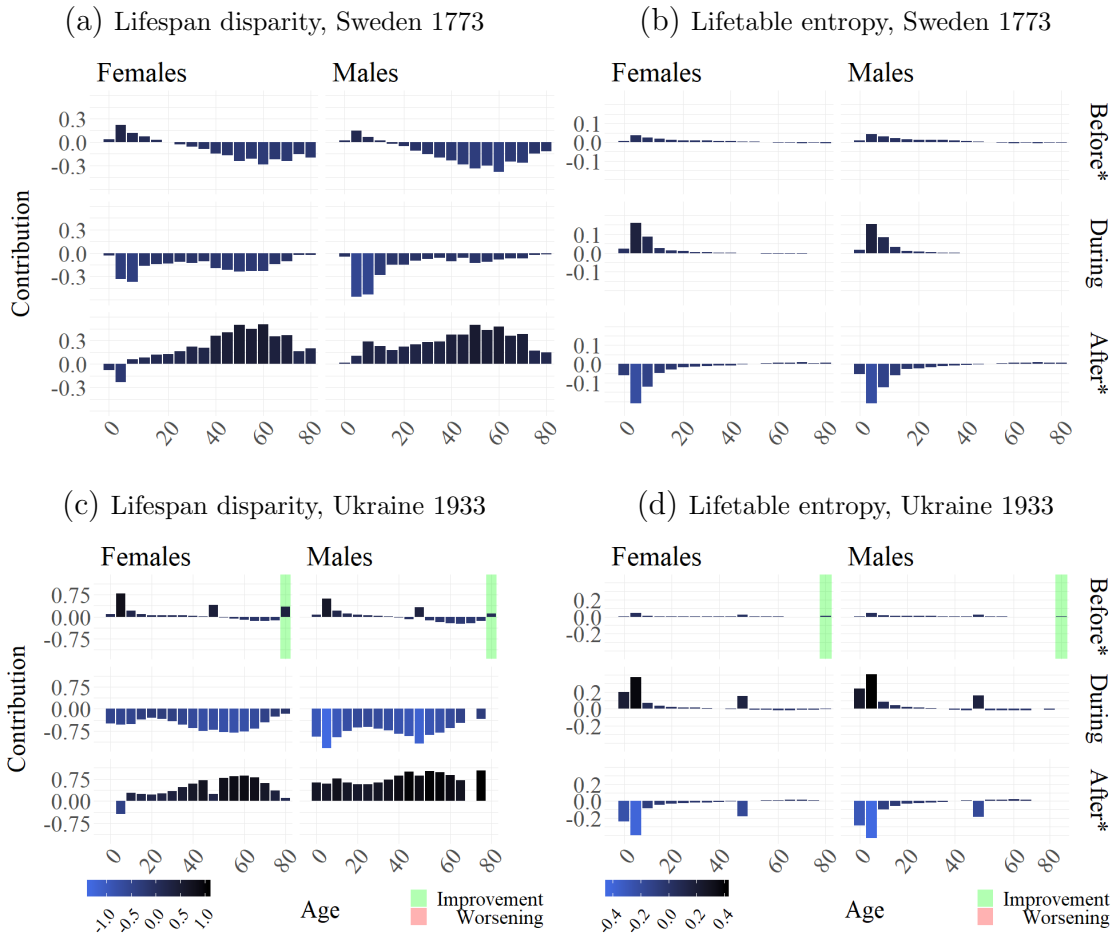
We can also find some differences within the two groups of measures. The two Gini coefficients present the flatter curves, with smaller absolute changes. However, the lesser proportional increase in its relative counterpart G_0 shows that G_0^{abs} is more sensitive in proportional terms to the changes in mortality. At the opposite end we have $e^\dagger$, as shown by the greater proportional increase of $\bar{H}$ in all five cases. Comparing sex-patterns shows that male absolute variability decreases more during a crisis. However, relative variation trends show that this is likely due to a greater drop in e_0.

~~Finally, some differences between cases merit a short commentary. First of all,~~ absolute variation starts decreasing right away in only two of the three cases where the crisis spans more than one year. 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 how the inclusion of e_0 modifies the behaviour 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 smaller 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 the three age-patterns of mrotality we identified in the previous section: during these mortality crises absolute lifespan variation declines, while relative lifespan variation increases.

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. For the crises which lasted two years, we analyse three changes: between the average of the 5 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.

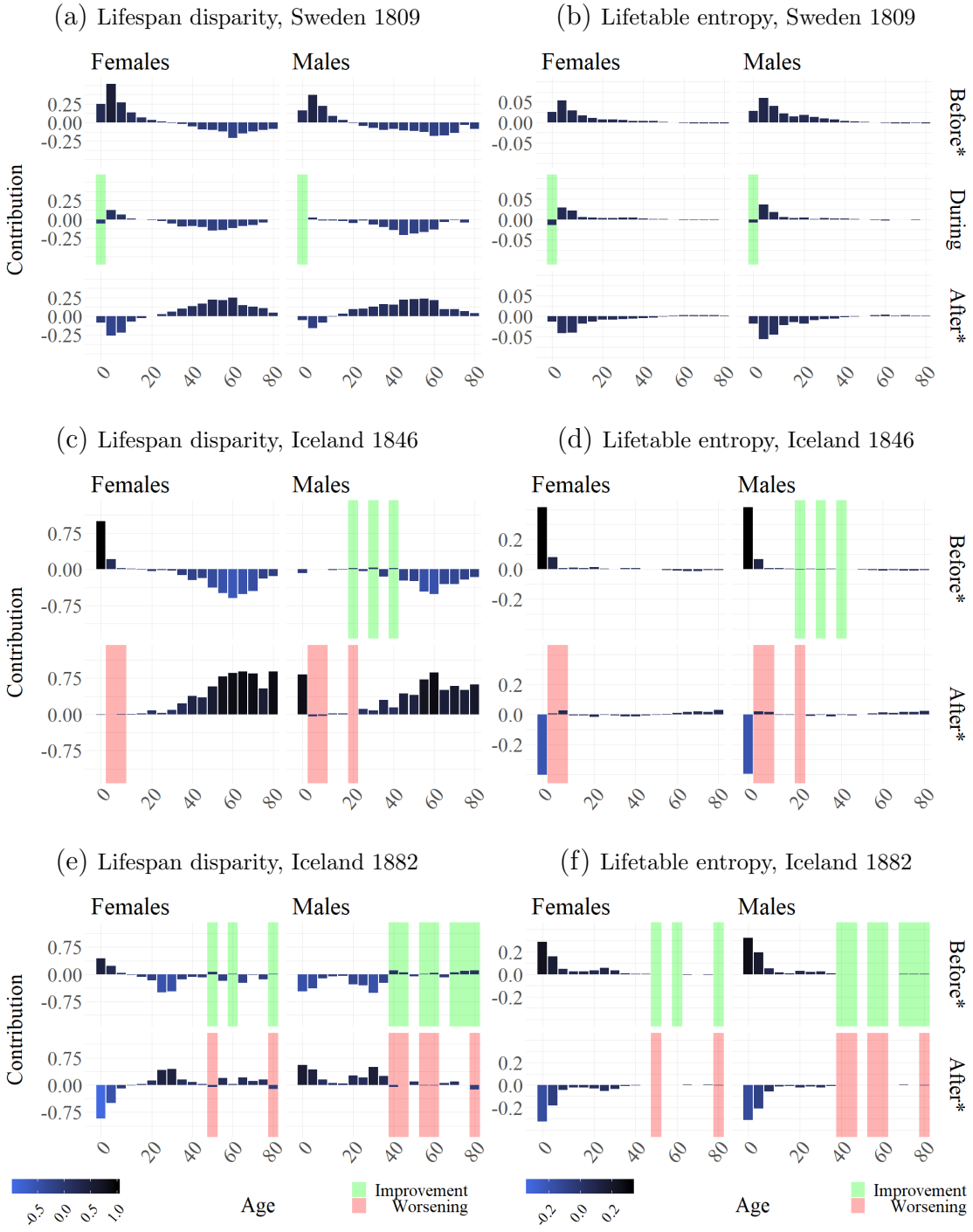
Figure 5: Lifespan variation decomposition, famines



* 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)

~~A striking difference~~ between results for $e^\dagger$ and $\bar{H}$ is that older ages contribute very little to the change in the latter, even when they contribute significantly to the change in the former. In fact, 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 stable. Thus, younger ages lead the change in $\bar{H}$, whereas the change in $e^\dagger$ between non-crisis and crisis periods (see previous section) runs opposite to the contribution of younger ages in almost all cases. Moreover, after the crisis most ages contribute positively to the increase in variation. Under usual circumstances mortality improvements at young ages contribute negatively to changes in lifespan variation, since they are below the threshold age (Aburto et al. 2020; Gillespie et al. 2014). However, during mortality crises this age decreases significantly, so that even young ages remain above it. In fact, Zhang and Vaupel (2009) have shown that when $\bar{H}$ increases over 1 the threshold age for $e^\dagger$ ($a^\dagger$) becomes 0. These results explain the unforeseen decrease of absolute variation during mortality crises. While we had expected older ages to contribute little to this change, their mortality actually leads change in absolute variation, even with very high infant mortality. Moreover, this shows how the concept of the threshold age may be important in understanding the trends of lifespan variation during crises (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).

~~Let us now examine our cases more in detail.~~ Looking at changes in log-mortality rates, we had found three patterns of mortality change. The three nutrition-related crises saw a smaller change for infants and for older ages. Indeed, infants contribute little to changes in both $e^\dagger$ and $\bar{H}$, especially when compared to older children. Similarly, contributions to $e^\dagger$ decline after about age 60, although it is difficult to judge whether this is due to the lesser mortality increase or to the dwindling survival function. 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. While the improvement for males between the period before 1846 and the epidemic is likely due to age heaping, the other cases reflect patterns of mortality, which were already underlined: potential deleterious consequences of the epidemic for young ages after 1846 and acquired immunity for older adults during the 1882 epidemic, so that mortality patterns for this population is not directly tied to the epidemic itself. The decomposition results mirror the 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 results 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.

6 Conclusion

In these analyses, we have found three main patterns of mortality change. While they are divided between nutrition-related crises and measles epidemics, they also reflect other distinctions, such as between larger and smaller populations or between two- or one-year crises. Still, all of our cases show the same pattern of lifespan variation: a drop in absolute variation, together with an increase in relative variation. Finally, by decomposing these changes by year, we have found that relative variation change is clearly led by infant and child mortality, while older ages play a role for absolute variation change, opposing contributions which underline the role of the threshold age in such situations.

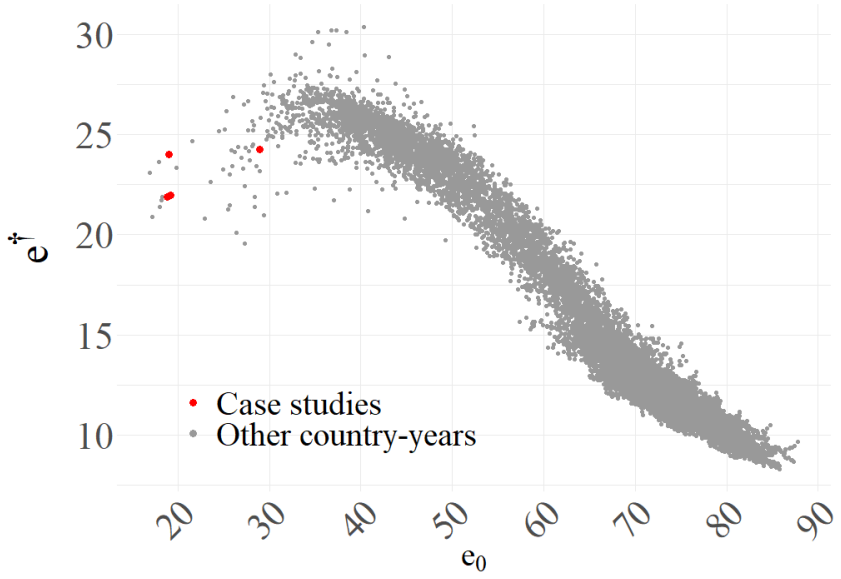
This text aims to contribute to the understand of lifespan variation and mortality crises in two ways. First of all, it shows a common trend of lifespan variation beyond the nature of the crisis itself. This can advance the understanding of human mortality under extreme conditions. Analyses into "other cause" crises could show whether this pattern is more widely true. Secondly, by comparing different kinds of measures, we hope to have given new insight into their characteristics and into the nature of their behaviours, providing information to help the choice of their use.

Naturally, this study also presents some limitaitons. The discrete implementation of the decomposition analysis leads to some imprecisions, which can be exacerbated by extreme mortality change. However, to our knkowedge more appropriate methods are not available and the interpretation of results should rely on the researcher's good sense. The focus on historical populations inhibits our investigation in three main ways. First of all, data quality is not necessarily assured, especially in the case of Ukraine here. Even when of good quality, the data do not provide information on social status of geographic location, for example, which could help in refining these results. Finally, although our results provide general patterns for such historic populations, they are not necessarily extendable to modern populations, characterised by older mortality, nor to the modern world, where migration and international aid could modify the mechanisms at play.

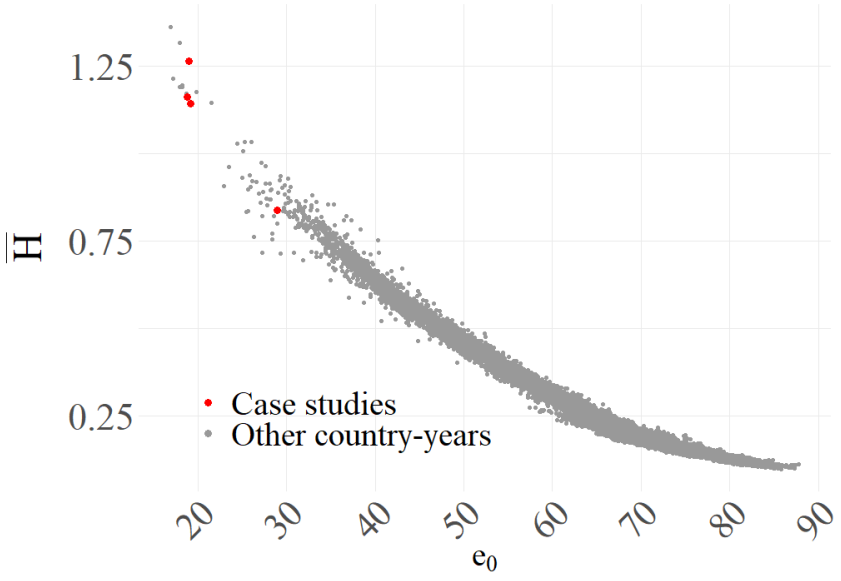
Appendix A

Figure A1: e_0 and lifespan variation

(a) e_0 and $e^\dagger$

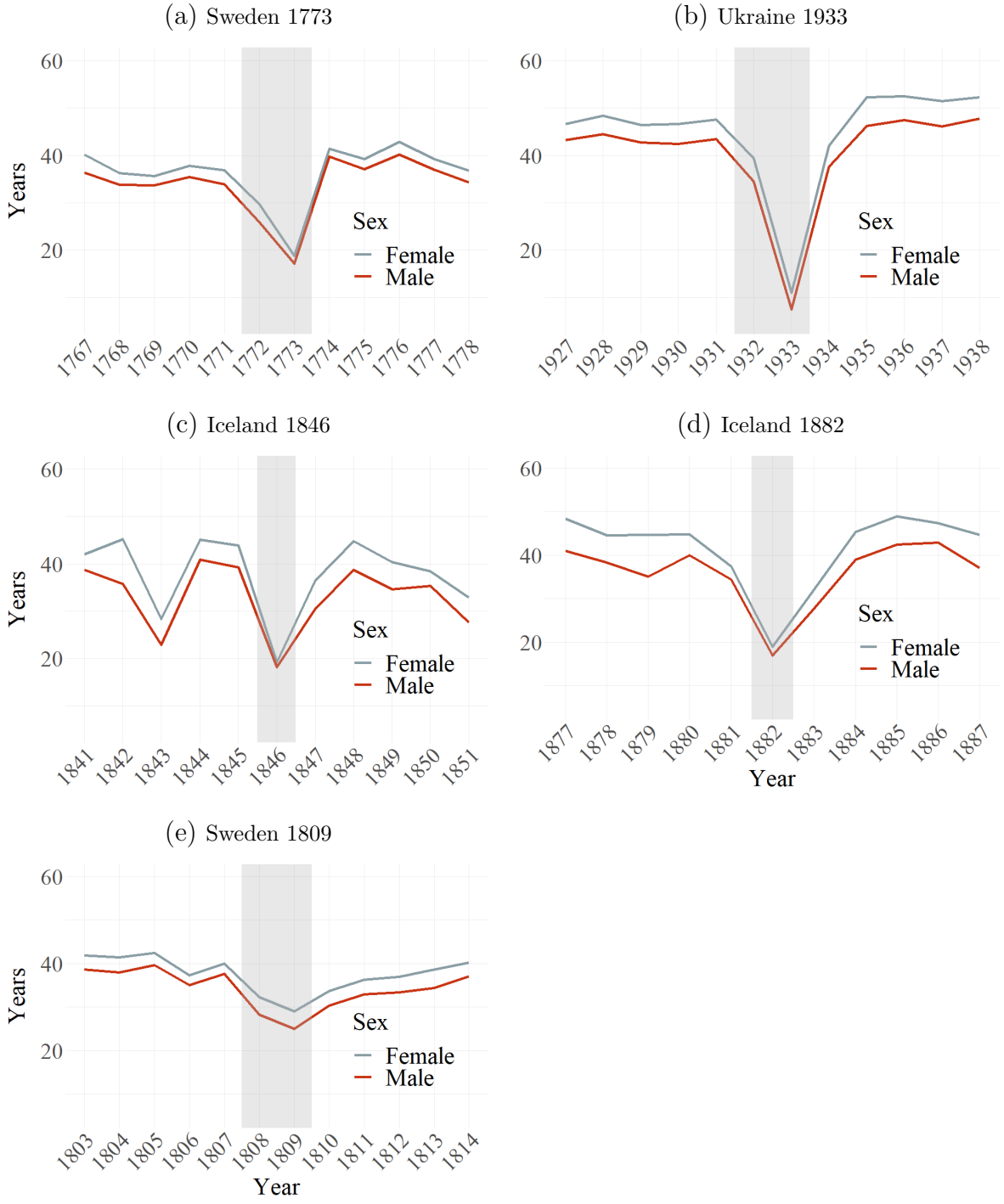


(b) e_0 and $\bar{H}$



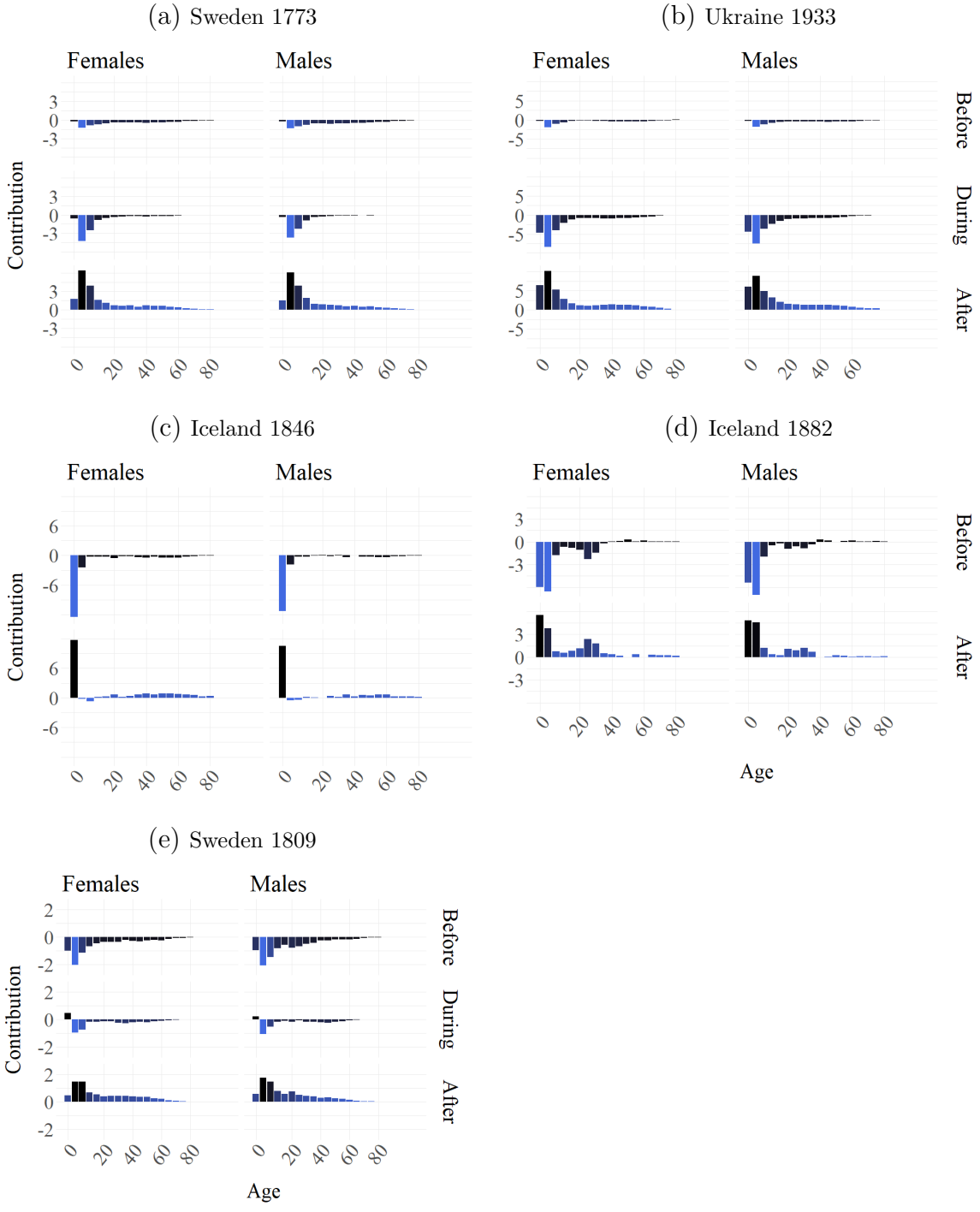
Source: HMD, all countries

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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