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Research Article

Divergent trends in lifespan variation during mortality crises

Serena Vigezzi

José-Manuel Aburto

Iñaki Permanyer

Virginia Zarulli

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Divergent trends in lifespan variation during mortality crises

Serena Vigezzi[1]

José-Manuel Aburto[2]

Iñaki Permanyer[3]

Virginia Zarulli[4]

Abstract

BACKGROUND
Lifespan variation has been attracting attention as a measure of population health and mortality. Several studies have highlighted its strong inverse relationship with life expectancy during periods of steady mortality decline, but this association weakens, and even reverses, when mortality does not improve equally over age. To date no study has comprehensively explored the behaviour of lifespan variation when mortality increases significantly.

OBJECTIVE
We investigate lifespan variation trends around various mortality crises, focusing on age-specific contributions and sex differences.

METHODS
Drawing data from the Human Mortality Database and Meslé and Vallin's Ukrainian lifetables, we analyse five European epidemics and famines across three centuries. We use six measures of lifespan variation and adopt the linear integral method of decomposition.

RESULTS
During these crises, relative lifespan variation increases, while absolute variation declines, and subsequently both quickly revert to pre-crisis levels. We show that mortality at older ages leads to a temporary increase in absolute – but not relative – variation. The

[1] Interdisciplinary Centre on Population Dynamics, University of Southern Denmark, Odense, Denmark. Email: sevi@sam.sdu.dk.
[2] Leverhulme Centre on Demographic Science and Department of Sociology, Nuffield College, University of Oxford, Oxford, United Kingdom.
Interdisciplinary Centre on Population Dynamics, University of Southern Denmark, Odense, Denmark.
[3] Centre d'Estudis Demogràfics, Universitat Autònoma de Barcelona, Barcelona, Spain.
Catalan Institution for Research and Advanced Studies (ICREA)
[4] Interdisciplinary Centre on Population Dynamics, University of Southern Denmark, Odense, Denmark.

lifespan variation of females is less affected than that of males, because of differences in the impact of infant and child mortality.

CONCLUSIONS
Even when infant mortality is high, mortality at older ages can influence lifespan variation. Our results also underscore the sex differences in the vulnerability of young individuals in periods of extreme mortality.

CONTRIBUTION
By underlining different trends of lifespan variation by sex and indicator, we offer new insight into the consequences of mortality crises. Contrary to what is often asserted, we show that the choice of lifespan variation indicator is not always inconsequential.

1. Introduction

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

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

By focusing on populations that experience mortality crises such as famines and epidemics, we contribute to filling this gap in two ways. First, by analysing lifespan

variation we uncover the effect of massive mortality shocks on heterogeneity, which would be overlooked when using life expectancy (Zarulli et al. 2018). Second, by comparing the patterns of a diverse set of measures of lifespan variation, we shed light on their behaviour during periods of substantial mortality increase and show that the choice of indicator is not always inconsequential.

2. Background

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

Mortality crises have also been examined. What constitutes a mortality crisis has been debated, from Goubert's initial suggestion of a doubling of the number of deaths (Goubert 1958) to more complex indices (for an introduction to the issue, see Charbonneau and Larose 1970). Nevertheless, mortality crises can be classified in four groups in relation to their causes (Sogner, in Charbonneau and Larose 1970; Goubert 1958): (1) subsistence crises (or famines) are brought about by lack or unavailability of food for a considerable part of the population; (2) epidemics are caused by diseases; (3) some crises combine both components, such as when a famine weakens the population, leaving it vulnerable to pathogens; (4) crises can have other causes, such as wars or natural phenomena. This latter class of crises presents specific dynamics, making it difficult to compare with the others. Therefore, we focus on the first three types. We adopt a straightforward definition of a mortality crisis: a period when life expectancy at birth e_0 decreased drastically and quite suddenly. For the purposes of this article we only consider relatively short crises, spanning up to two years.

No single mortality pattern can be expected during crises, because age- and gender-specific death rates vary. Males and older individuals seem to be at higher risk of dying from COVID-19 (Sharma, Volgman, and Michos 2020; Verity et al. 2020), while other crises – such as natural disasters – have a greater effect on women, children, and the

elderly (Bern et al. 1993; Frankenberg et al. 2011; Neumayer and Plümper 2007). Bongaarts and Cain (1982, in Kane 1987) hypothesise that in the case of short famines, mortality will reach its peak at the end of the famine period and then gradually decline. After about a year it could even drop below pre-famine levels (as was found by Goubert (1958) for Northern France in the 17th century), as only the most robust individuals will have survived. However, age- and gender-specific mortality patterns vary depending on the cultural and social environment. Bengtsson, Campbell, and Lee (2009) find that infants in 19th century Sweden were generally less affected by food availability, as they depended on breastfeeding, while older children were more sensitive to it. The elderly were also affected, but showed little difference in terms of gender and socio-economic status, possibly as a result of selection into old age. Younger women in the same populations might have been more vulnerable to food deprivation than males because of breastfeeding and pregnancy, in addition to potential discrimination in household food distribution, which has been highlighted in 19th century populations in Germany (Klasen 1998). Notwithstanding, females experience lower mortality at almost all ages, even in populations that experience extremely high mortality – possibly because of biological advantages, as shown by Zarulli et al. (2018) in diverse populations across space and time. However, the authors also find that this gap can reverse due to social preferences and the incidence of gynaecological diseases or childbirth complications.

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

3. Context

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

Dribe et al. (2015) describe the 1772–1773 famine and the 1808–1809 epidemic. Crop failures in large regions of Sweden caused by unusual weather in 1772 exacerbated

high food prices and led to a famine which peaked the following year. In 1773, death rates were 86% higher in the most affected counties than in the others and crude death rates doubled in central Sweden. Although all age groups were affected, children between ages 1 and 14 suffered the most in proportional terms, while infants witnessed a relatively small proportional increase in mortality. Mortality was mostly driven by nutrition-related diseases, specifically typhus and dysentery (which alone accounted for 50% of the excess mortality that year). Typhus and dysentery were also the diseases involved in the 1808–1809 epidemic, which followed troop movements in the Finnish War. Despite the impact of the war, the increase in mortality is thought to have resulted mainly from the epidemic (Glei et al. 2019). As a consequence, mortality followed the same age pattern as in 1772–1773.

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

The context of the Ukrainian famine of 1932–1933 is more difficult to describe, as data was not publicly accessible until the dissolution of the Soviet Union. Naumenko (2021) reports three main competing explanations. First, bad weather would have lowered harvest yields in 1932, leading to famine in the following months (Tauger 1991). Others consider that the crop output declined because of the collectivisation policies that had been implemented since the late 1920s. A poor harvest in 1931 and the effort by the government to maintain food distribution in the cities caused hunger in the countryside, which evolved into widespread famine in the following years (as Naumenko herself maintains). Finally, a third strand of research argues that the famine was a result of the government's decision to curtail food distribution in order to quell anti-government sentiment in Ukraine, which had been sparked by collectivisation policies (Graziosi 2015). However, it is agreed that during these years Ukraine experienced a harsh famine, which led to the death of between 2.1 and 3.9 million people (Meslé and Vallin 2012; Rudnytskyi et al. 2015).

4. Research hypotheses

Our first research question looks at trends in mortality increase and lifespan variation. We anticipate three main patterns of mortality change, partly following the arguments of Zarulli (2013). For the two Swedish and the Ukrainian crises we expect to see a proportional increase in mortality at adult ages, together with some convergence at older ages due to selection of the most robust individuals, and a disproportionate increase in child mortality (given the vulnerability of children), at least in Sweden (Dribe, Olsson, and Svensson 2015). In Iceland we anticipate two different patterns: for the first epidemic, a proportionately greater increase of mortality in children and the elderly (one-year crises may not be long enough for a selection effect to appear); for the second, a greater increase in children only, as the elderly had already acquired immunity.

While increases in mortality at any age lower life expectancy, increases at different ages may have divergent effects on lifespan variation. Increases in mortality at young ages typically contribute to increasing lifespan variation. However, increases at older ages compress the mortality distribution, so that these ages contribute to a decline in lifespan variation. Each mortality distribution presents a unique and specific age at which contributions go from positive to negative (and vice versa when mortality declines). This is called the threshold age and it is usually close to life expectancy at birth (Aburto et al. 2019; Gillespie, Trotter, and Tuljapurkar 2014; Zhang and Vaupel 2009). Thus, the expected mortality increases for children and the elderly would contribute to the change in lifespan variation in opposite directions, as each age group sits on a different side of the threshold age. We expect both of these age groups to suffer the most during a crisis, as they are physiologically weaker than younger adults. Moreover, since very young children and the elderly contributed little to the survival of a household, their wellbeing may have been more neglected during such times (Bengtsson, Campbell, and Lee 2009). However, only the most robust individuals reached old age, so that we expect children to be more strongly affected by the crises than older individuals. Therefore, in all five crises we expect lifespan variation to initially increase and then decline – either gradually, if the consequences of the crisis continue to affect weakened individuals, or sharply, if there is selection at the vulnerable ages during the crisis, as has been found for Ukraine (Zarulli 2013).

The second research question analyses gender differences. Colchero et al. (2016) show that the gender gap in lifespan variation, which generally favours females (van Raalte 2011), does not change in high mortality situations. We expect similar results for our analyses. We also expect the trends to differ between sexes according to the gender specificity of the crisis: for example, the measles epidemics and possibly famines might have exacerbated existing inequalities in household food distribution, resulting in starker changes for (young) females.

Our third question is to determine whether some ages contributed more than others to the change in variation during and after the crisis. We anticipate that a mortality crisis will particularly affect children (with the exception of infants, protected by breastfeeding) and the elderly. These sub-groups are less physically equipped to deal with extreme conditions and children might receive less care in situations where resources are scarce, although the latter phenomenon is context-specific (Lanau and Fifita 2020). Gender discrimination can also exacerbate the uneven allocation of resources for girls. Moreover, deaths at the extremes of a distribution will have a greater effect on variation, with lifespan variation indices typically very sensitive to changes at young ages (van Raalte and Caswell 2013). Therefore, we expect that these age groups will increase lifespan variation. However, because of the reasons outlined earlier, the contribution of older age groups might be smaller than that of young people.

5. Methods and data

5.1 Methods

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

$$e^\dagger = \int_0^\omega d(x)e(x)dx$$

while its relative counterpart lifetable entropy is

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

where ω is the highest age in the population, $d(x)$ is the death distribution, and $e(x) = \frac{1}{l(x)}\int_x^\omega l(a)da$ *is* life expectancy at age x. e_0 represents life expectancy at birth, which equals $\int_0^\omega l(a)da$, for $l(0) = 1$.

In a similar way, the standard deviation is

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

and the coefficient of variation

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

Finally, the absolute Gini coefficient is

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

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

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

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

While absolute and relative measures both aim to capture the existing differences in lifespans between all individuals within a population, they define inequality in different ways. Absolute indicators measure these differences in number of years. For example, the absolute Gini coefficient is the average distance, in years, between any pair of individuals' ages at death (Shkolnikov, Andreev, and Begun 2003), while the standard deviation is the square root of the average squared distance from the mean age at death. Finally, lifespan disparity can be interpreted as the average number of years lost at death by the individuals in the population. Because of their definitions, absolute indicators remain constant if the absolute distance between individuals does not change: if all lifespans increase (or decrease) by the same number of years, these indicators remain

unaffected. This does not mean, however, that absolute indicators are independent of life expectancy, which is still present in their formulas. In fact, life expectancy levels bind the realistic range of values absolute variation can take: a population where life expectancy is measured in days (e.g., flies) cannot realistically have a variation of years (as a human population does).

Relative indicators are composed of absolute indicators divided by a measure of longevity, in our case life expectancy, to enable comparing populations with very different life expectancy levels. These indicators are the basis of the pace and shape framework proposed by Baudisch (2011), which is used to make comparisons across species (Jones et al. 2014). Relative indicators, then, are dimensionless, and in this paper express variation as a proportion of life expectancy.[5] Thus, relative indicators remain unaffected by a proportional change across all lifespans in a population, even if the absolute difference in years changes.

We use multiple measures for three reasons. First, by comparing outcomes we can make more robust conclusions. Second, relative and absolute measures of variation each have their own advantages. Relative measures facilitate comparisons across populations, but absolute measures of lifespan variation are easier to interpret. Moreover, some research has suggested that a mix of relative and absolute measures of variation can better describe the idea of equality in some disciplines (Asada 2010), so that both kinds of measure should be considered in research. Finally, by using both sets of measures we can observe the differences in their behaviours under such extreme mortality circumstances.

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

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

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

[5] $\overline{H}$ can also be interpreted as a measure of the elasticity of life expectancy to a proportional change in mortality (Keyfitz and Golini 1975; Leser 1955).

continuously, which is particularly appropriate when studying changes across time. The analyses were performed in R version 4.1.2.

5.2 Data

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

We use data for Iceland and Sweden, which parishes have collected since the 18th century (Andreeva, Borges, and Dukhovnov 2020; Glei et al. 2019). The raw data was corrected but a number of issues remain, such as age heaping and age exaggeration. Moreover, in Sweden out-migration was not recorded, which could bias our analyses if the crises encouraged migration. This should not be an issue for Iceland, because of its isolation and the rapidity of the epidemics. At the same time the Icelandic population is so small (a little under 60,000 in the 1840s (Tomasson 1977)) that it is likely to show a fair deal of statistical noise. Death rates at older ages were smoothed to exclude excessive fluctuations and to disaggregate open age intervals. These procedures caused some implausible patterns for these categories. Therefore, we reduce the number of age categories by creating an open-ended class at age 80, instead of 110. We also use abridged period lifetables to lessen the effect of digit preference. The other issues are more difficult to tackle and should be kept in mind when interpreting results.

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

6. Results

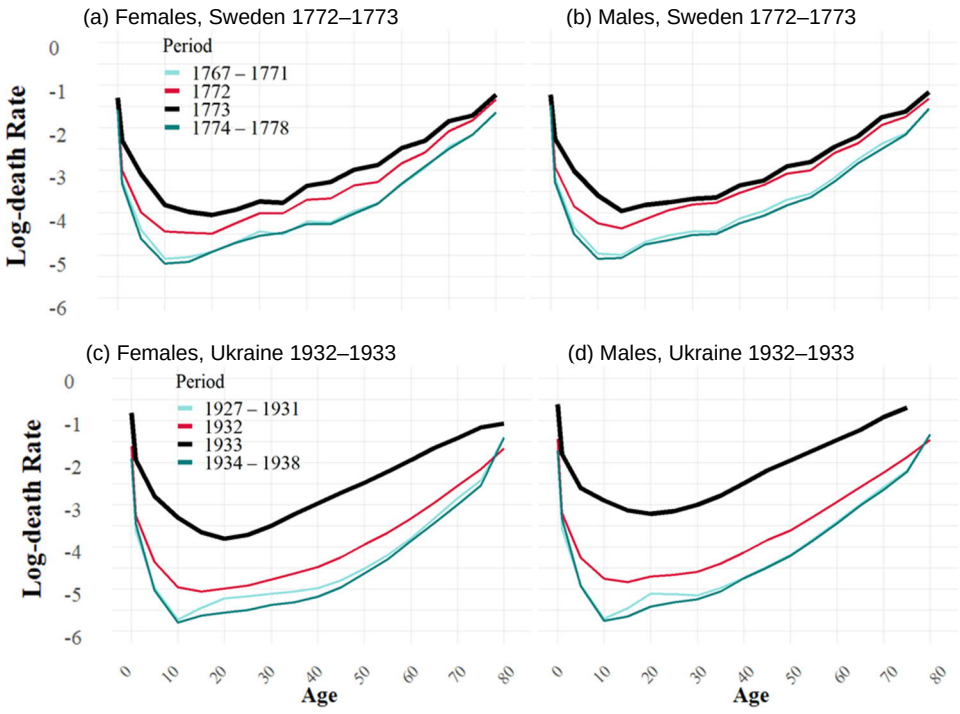
6.1 Age patterns of mortality

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

Despite some differences in magnitude and in the temporal dynamics of famines (Figure 1), there are many parallels. In both cases the increase in mortality is smaller at age 0 and there is evidence of convergence at the oldest ages for both sexes in Sweden and for females in Ukraine (as the survival function for Ukrainian males in 1933 reaches 0 at age 75, the rates stop short of age 80). Sex differentials also behave similarly: not only are male rates always higher, but they also increase more, reflecting other findings of greater male vulnerability to mortality crises (Zarulli et al. 2018).

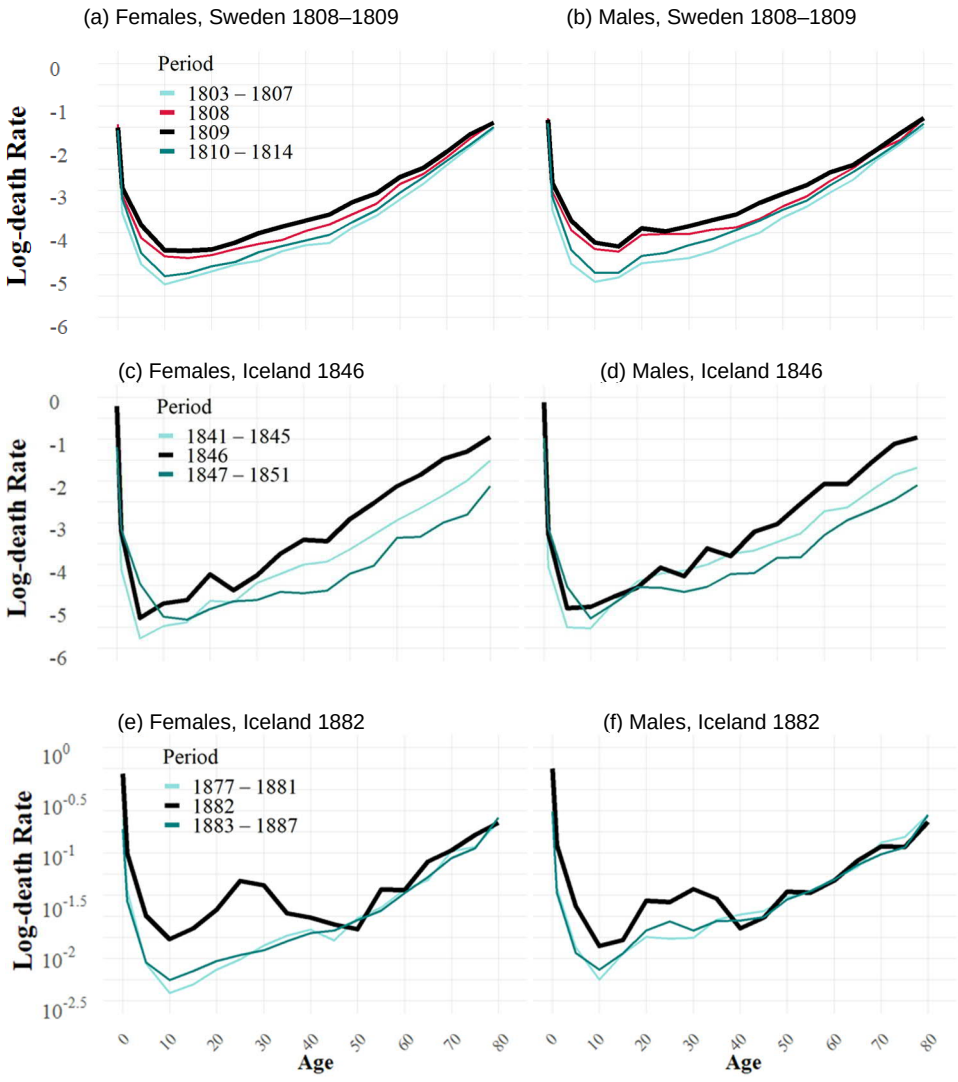
Figure 2 shows log-death rates for the three epidemics. Contrary to what happened for famines, each epidemic presents a distinct mortality pattern. During the 1809 typhus and dysentery epidemic, infant mortality was not affected. Male mortality is higher for all periods, but even more so in 1808–1809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

Figure 1: Log-death rates, famines



Source: HMD (Sweden) and Meslé and Vallin.

Figure 2: Log-death rates, epidemics



Source: HMD (Sweden and Iceland).

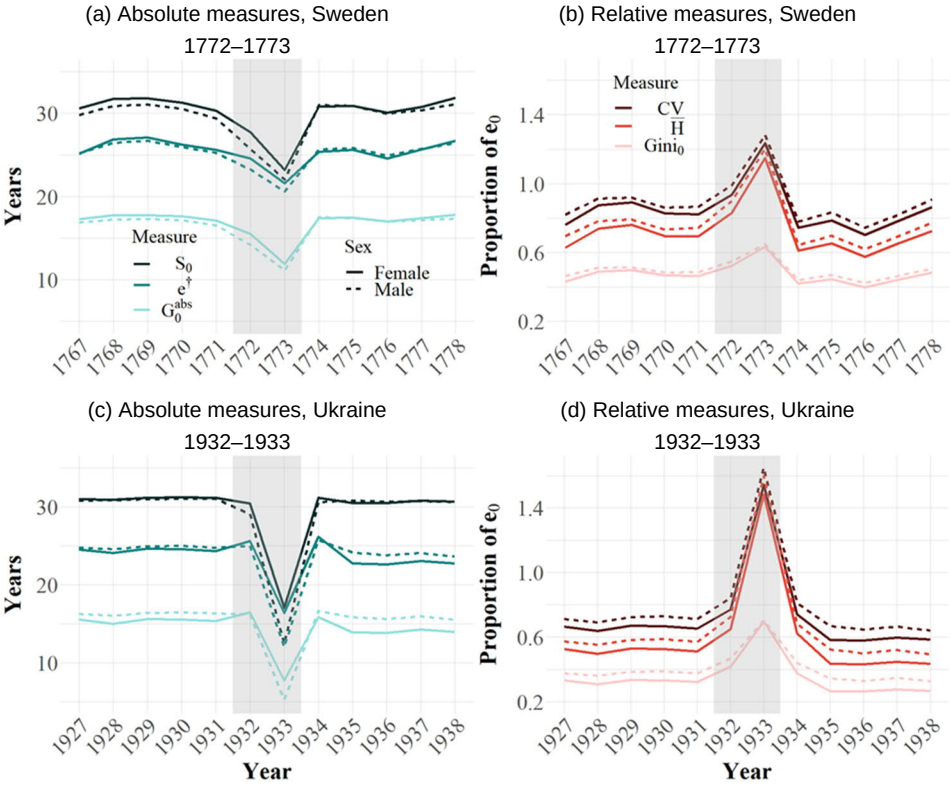
6.2 Trends of lifespan variation

In Figures 3 and 4 we examine how absolute and relative lifespan variation measures behave (note that we use single years rather than 5-year averages for pre-crisis and post-crisis periods). A striking finding is that absolute and relative variations present contrasting dynamics: the former decreases during the crisis, while the latter increases. While the increase in relative indicators can be attributed to the stark drop in its denominator (e_0), the decrease in absolute variation is more surprising. This can be explained by the substantial impact of mortality crises on young ages, so that a high proportion of deaths are concentrated in early ages, compressing the distribution, lowering life expectancy, and thus diminishing the impact of mortality at young ages towards increasing variation.

Another difference is that absolute variation is usually higher for females than for males, whereas the opposite is true for relative measures. Male absolute variation also changes more during a crisis, whereas relative variation does not. However, both kinds of measure show that typically the change in lifespan variation only lasts during the crisis itself, with little effect during the following years, as in previous findings by Colchero et al. (2016). This mirrors the trend of life expectancy, which also returns to pre-crisis levels right away; two notable exceptions being the 1809 Swedish epidemic, where e_0 increased slowly after the crisis, and the 1846 Iceland epidemic, where e_0 returned to its pre-crisis level for one year, but declined again soon after (see Appendix, Figure C-1).

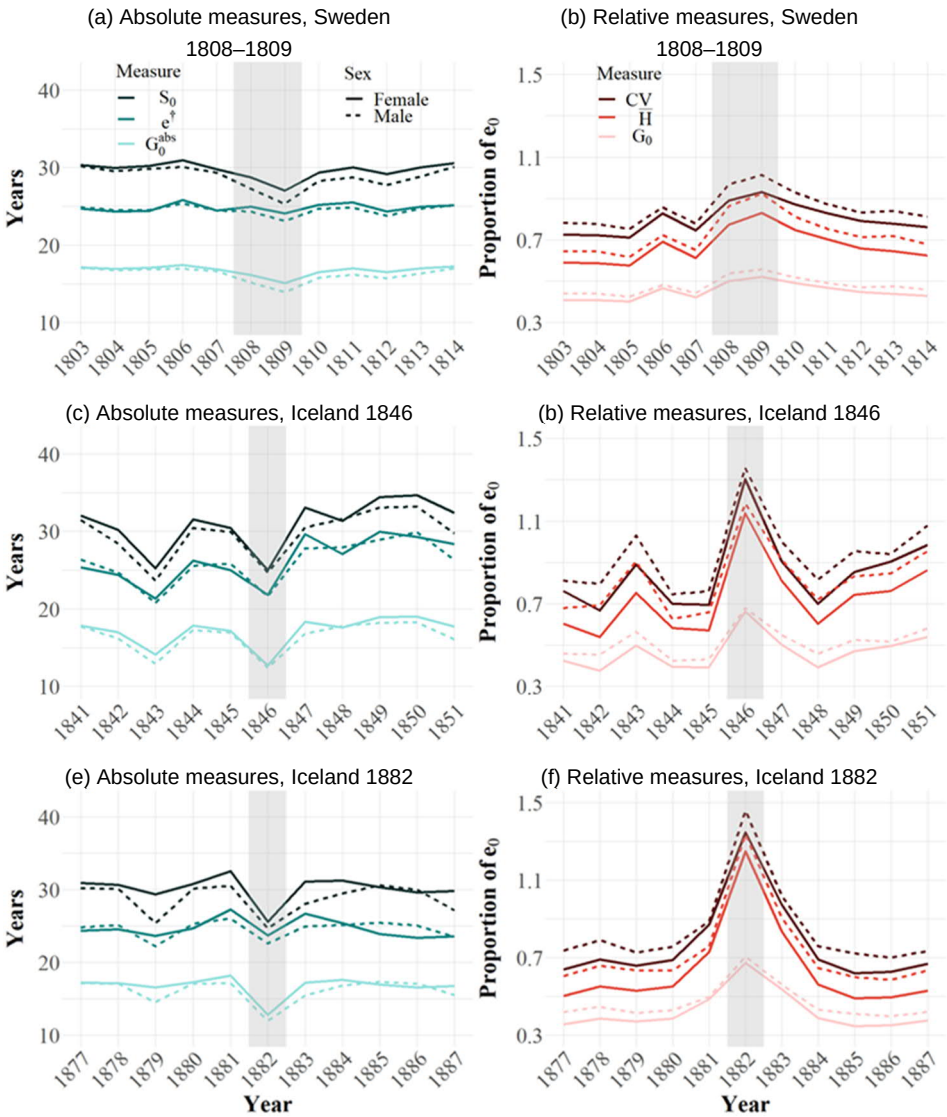
We also find differences within each group of measures. The two Gini coefficients present the flatter curves, with smaller absolute changes. However, the lower proportional increase in its relative counterpart G_0 shows that G_0^{abs} is more sensitive in proportional terms to the changes in mortality.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

Figure 3: Lifespan variation, famines



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

Figure 4: Lifespan variation, epidemics



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

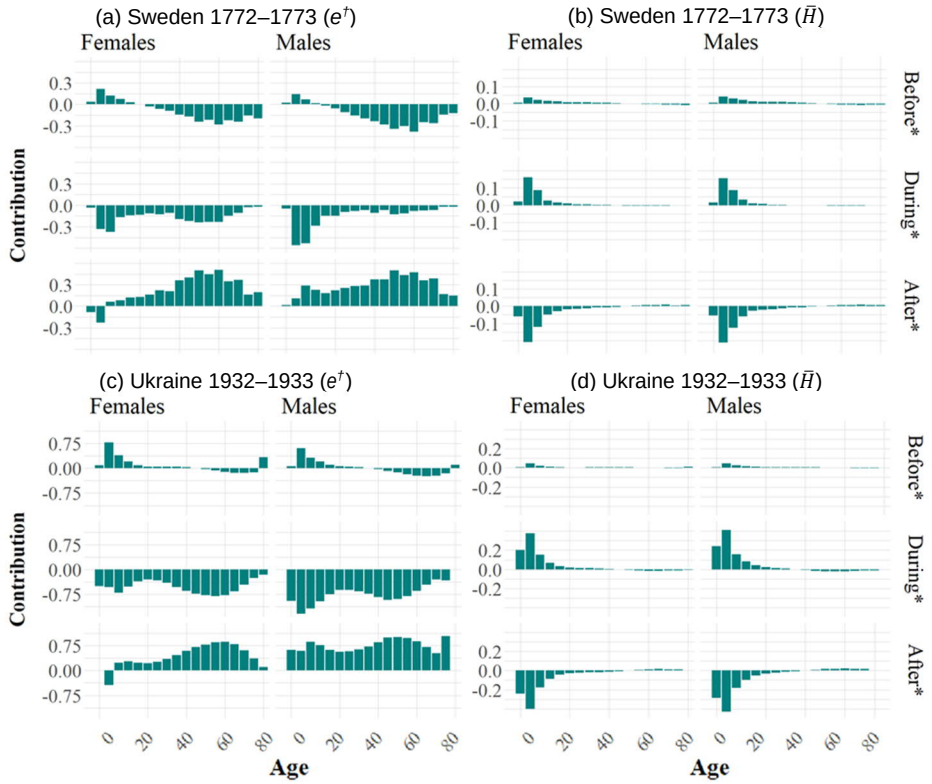
Absolute variation starts decreasing right away in two of the three crises spanning two years. In the Ukraine there is a noticeable drop only in 1933, with the small exception of male S_0. However, relative variation already begins to rise (albeit slightly) in 1932, further underscoring the different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

6.3 Decomposition of lifespan variation

Figures 5 and 6 show the results of the decompositions for males and females. We report results for two measures, $e^\dagger$ and $\bar{H}$, but the same main trends appear for the others, as shown in the Appendix, Figures E-1 to E-4. These figures analyse how the change in lifespan variation between the periods before, during, and after each crisis can be attributed to the mortality change in each age group for the corresponding period. Each panel is divided into two (for one-year crises) or three (for two-year crises) rows, which analyse the change in lifespan variation between the period before the crisis and the crisis ('Before' row), during the crisis itself (when it lasted two years) ('During' row), and between the crisis and the period afterwards ('After' row). The contribution of each age group is indicated by a blue column and can be positive (i.e., contributes to increase lifespan variation) or negative (i.e., contributes to decrease lifespan variation) depending on the direction of the mortality change experienced by the age group and its position below or above the threshold age. The overall change in lifespan variation is calculated by summing all contributions. For example, the change in Swedish female infant mortality between the period 1767–1771 and the beginning of the famine in 1772 contributed +0.034 years to the change in $e^\dagger$, which decreased by 1.58 years. To ensure readability, the graphs have different scales.

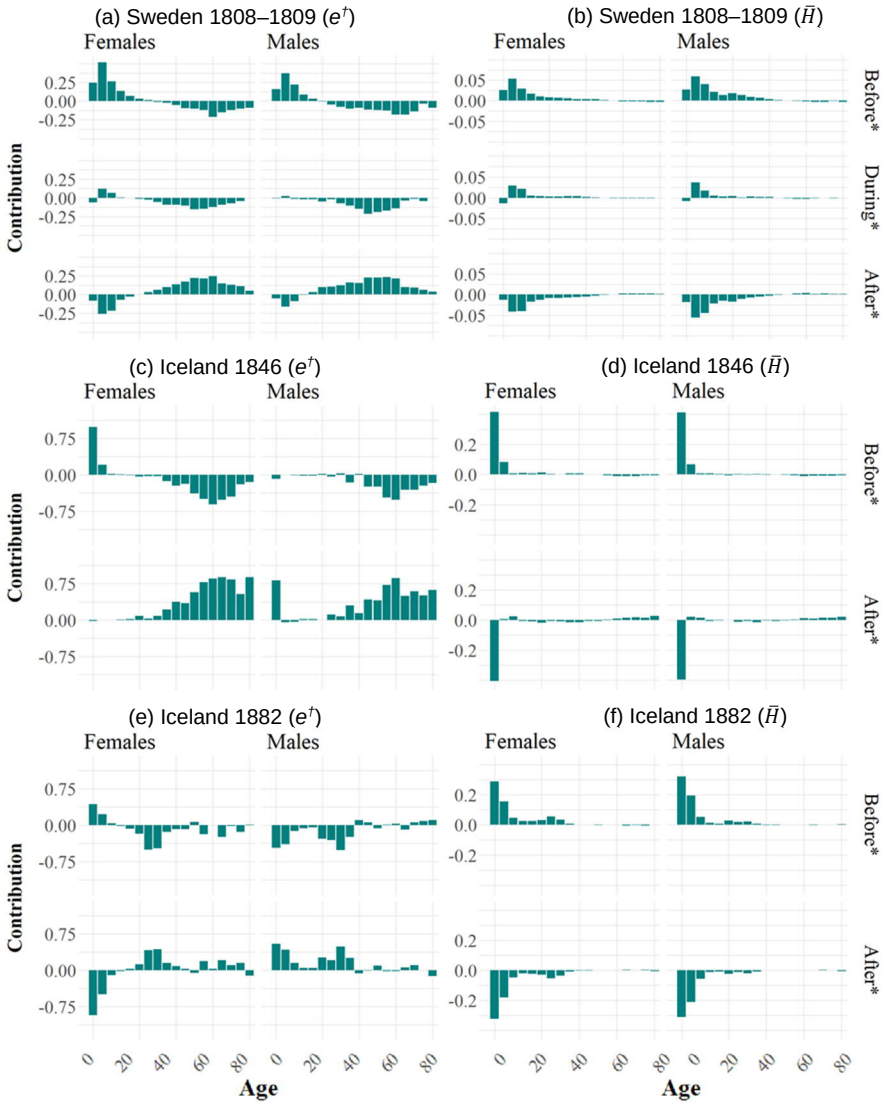
Across crises, the small columns on the right hand of the graphs related to $\bar{H}$ show that older ages have very little influence on the change in relative lifespan variation, even when they contribute significantly to the change in $e^\dagger$. Differences in the contribution of mortality changes at older ages, then, seem to be behind the diverging trends of absolute and relative variation during these mortality crises.

Figure 5: Lifespan variation decomposition, famines



Source: HMD (Sweden) and Meslé and Vallin.
Note: * Before = change in indicator between the average of the previous 5 years and the (first) crisis year, During = change in indicator between the two crisis years, After = change in indicator between the (last) crisis year and the average of the following 5 years. $e^{\dagger}$ indicates lifespan disparity, $\bar{H}$ indicates lifetable entropy.

Figure 6: Lifespan variation decomposition, epidemics



Source: HMD (Sweden and Iceland)
Note: * Before = change in indicator between the average of the previous 5 years and the (first) crisis year, During = change in indicator between the two crisis years, After = change in indicator between the (last) crisis year and the average of the following 5 years. $e^{\dagger}$ indicates lifespan disparity, $\bar{H}$ indicates lifetable entropy.

Sex differences are more accentuated for $e^{\dagger}$ compared to $\overline{H}$, especially at young ages. Across all crises, young males have smaller contributions before and during the crisis and a greater contribution afterwards, suggesting the existence of a common underlying mechanism. The sex-specific effect of measles can also help explain the exacerbated sex differences for Iceland, although it cannot do so fully.

Beyond these differences, however, there are some trends common to both indicators. The three nutrition-related crises (the two famines and the 1809 epidemic) saw small contributions of infants to the change in both $e^{\dagger}$ and $\overline{H}$, especially when compared to older children. Contributions to the change in $e^{\dagger}$ also decline after about age 60. The decomposition results for the Icelandic epidemics mirror age-specific patterns: significant contributions of infants (and older adults) in 1846, and of infants (and younger adults) in 1882.

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

7. Limitations

The data quality is not necessarily guaranteed. The sources of the HMD's death and population estimates do not record migrants. However, migrants are likely to have been healthier than those who stayed behind (Helgesson et al. 2019), so this recording issue probably does not influence our results to a significant extent. The Ukrainian data poses some specific issues. Namely, deaths are likely to have been underreported. This is especially true for infant deaths, which may in fact have been underreported in both Sweden and Iceland (Zarulli et al. 2018). Higher infant mortality would concentrate the deaths, thus accentuating the drop in absolute variation, but would also decrease e_0, so that relative variation might still increase. We addressed these issues through sensitivity analyses, changing the death rate by ±10% for every age and for infants only. We also changed the open-ended category in the lifetables between ages 70 and 90. Finally, we ran further sensitivity checks on a different dataset related to the Ukrainian famine (graciously provided by professor Wolowyna (Rudnytskyi et al. 2015)). All of these confirmed our main results. We also tested sensitivity to the length of the non-crisis periods, comparing crisis years with 10, 15, and 20-year periods before and after each crisis.[6] These tests did not change our main results either.

[6] We could not run these tests for the first Iceland epidemic or the Ukrainian famine, as our data does not span sufficient years.

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

8. Discussion

We analysed five mortality crises – two famines and three epidemics – that occurred in three countries and span 160 years. Each case presented a pattern of mortality change through the crisis, which can be linked to its specific context. Despite these differences, each crisis was accompanied by a drop in absolute lifespan variation and a spike in relative variation. Decomposing by age showed sex differences, especially at younger ages, and a different effect of mortality change at young and older ages between absolute and relative measures.

8.1 Mortality patterns

The five mortality crises we study were characterised by extreme and sudden drops in life expectancy at birth. In similar contexts, Zarulli (2013) found that mortality increases proportionally at adult ages, with convergence for older individuals when a crisis lasts more than one year. We observe comparable trends in our five cases. Even for the short Icelandic epidemic of 1846 there is evidence of a potential mortality displacement at adult ages, translating into a lower-than-pre-crisis mortality following the crisis itself. Some trends deviate from this model, but they are mostly attributable to the context in which the crisis happened, and to its characteristics. The lower increase in infant mortality for

the two Swedish and the Ukrainian crises is consistent with the protective effect of breastfeeding against nutrition deficiency and nutrition-related diseases (Livi-Bacci 1990). Bengtsson, Campbell, and Lee (2009) argue that in the past, children – as net consumers of resources – might have become expendable in times of crisis. As this attitude changes across time and space, it could clarify the disproportionate increase in child mortality in the Swedish famine, which did not happen in the Ukrainian famine. However, the Ukrainian data has been extensively manipulated and smoothed, and might be masking a similar increase for children. In the Ukrainian case, death rates also increase proportionally less for young adults than for older ones, especially for females. This could be due to a greater resilience of these ages in the circumstances of the Ukrainian famine or, again, to the manipulation of the data.

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

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

8.2 Lifespan variation trends

Lifespan variation gives us insight into the distribution of ages at death in a given year. The rise in relative measures indicates that mortality crises negatively affected both e_0 and lifespan variation, but the drop in absolute measures nuances this interpretation. Mortality increases led to a drop in life expectancy and in the threshold age, thus limiting

the influence of a worsening of mortality at younger ages on increasing variation. At the same time, fewer individuals reached advanced ages under current conditions of mortality. Thus, the crises we have studied compressed the mortality distribution in absolute terms. However, deaths increased substantially at the extremes of this compressed distribution, especially at young ages where individuals are more vulnerable. These differential impacts of mortality changes across the age span led to the divergence between absolute and relative lifespan variation. Our results add to the literature on lifespan variation that shows that the relationship between life expectancy and lifespan variation – which is typically found to be negative (Colchero et al. 2016; Sasson 2016; Smits and Monden 2009; van Raalte et al. 2011) – is not so straightforward (Edwards and Tuljapurkar 2005; Permanyer et al. 2018; van Raalte, Martikainen, and Myrskylä 2014). What this also suggests is that even a generalised increase in mortality affects individuals differently. This could be due to biological differences, as Zarulli (2018) suggests in relation to the sex gap, to societal structure, or to some other factor.

Despite the massive effect that our five crises had on mortality, we cannot see noticeable mid-term consequences. Not only does e_0 go back to pre-crises levels right away, but so does lifespan variation. In this sense, Figures 3 and 4 are a testament to the resilience of populations. However, as Goldstein and Lee (2020) point out, period measures may overstate the impact of a short-lived mortality crisis, because they "implicitly [assume] that the epidemic is experienced each year over and over again" (Goldstein and Lee 2020: 22037). Despite this limitation, because lifetable measures are standardised for the structure of a population and express the underlying risks of mortality, they make it possible to meaningfully compare the experiences of different populations in this area.

8.3 Sex differences

Figures 3 and 4 show that absolute variation tends to decrease less for females than for males. Thus, male mortality is more concentrated. However, since e_0 also decreases more for males than for females, the increase in relative variation is more similar across sexes. We also find that the sex difference in absolute variation trends is mostly due to differences in the weight of young ages: before the crisis, young males contribute less than young females to the change in $e^{\dagger}$, thus compensating less for the influence of older ages, while they contribute more in the other periods. The fact that a similar pattern is present for all crises suggests that a common mechanism may underlie these trends. Previous research has shown that males are physiologically more vulnerable during infancy and early childhood (Drevenstedt et al. 2008; Pongou 2012; United Nations, Department of Economic and Social Affairs, Population Division 2011), but the greater

absolute contribution of young males to lifespan variation is only present during and after each crisis. Increased discrimination against young females could counterbalance this male physiological vulnerability when mortality has not yet increased too much. Moreover, the male threshold age decreases more than that of females, thus decreasing the weight of young male mortality and possibly dampening the effect of biological differences.

Although the contribution of child mortality to the change in $\overline{H}$ is not identical across the sexes, the differences are less pronounced. An exception to these trends is represented by the two Icelandic epidemics, where lifespan variation changed either equally, or more for females. These differences again seem to be mostly explained by differences in the contribution of young ages, only this time with greater female mortality because of measles (Garenne 2015). Thus, young ages determine existing sex differences in lifespan variation change.

8.4 Methodological implications

Our analyses also show that relative and absolute measures are not interchangeable. While at mid-range levels of e_0 the relationship with absolute variation is indeed negative, when e_0 drops to very low levels, absolute variation also starts to decrease (as shown in panel b of Figure A-1 in the Appendix). Permanyer and Shi (unpublished) find a similar plateau in absolute lifespan variation for very high levels of e_0. They link this plateau to a recent slowdown in e_0 gains compared with gains in longevity; however, it is more difficult to adopt this framework in our case, where longevity is to a great extent determined by previous and drastically different mortality structures. Decomposition results give us some clarifying insight. As expected, the greatest single contributions to the change in $e^\dagger$ come from very young ages. However, it is actually older ages that lead the direction of this change. Since the threshold age for $e^\dagger$ ($a^\dagger$) trails e_0 closely (Aburto et al. 2019), it was quite low in the periods we considered (see Appendix, Table D-1). Thus, even at relatively low ages, mortality increases contribute to decreasing $e^\dagger$ and their sum can offset the positive contribution of younger ages. A similar mechanism explains the sudden increase of $e^\dagger$ after the crisis. Zhang and Vaupel (2009) show that when $\overline{H}$ surpasses 1, which consistently happens during our crisis years, $a^\dagger$ becomes 0. Because of this and because, after the crisis, mortality largely improves, all ages contribute to an increase in $e^\dagger$, bringing it back to pre-crisis levels.

On the other hand, relative variation meets our expectations. Panel a of Figure A-1 in the Appendix shows a monotonic and negative relationship between $\overline{H}$ and e_0 throughout the age distribution. The definition of relative measures is key to understanding this trend. e_0 is more sensitive to the crisis than absolute variation

(Appendix, Figure C-1). Because the denominator decreases more than the numerator, relative variation increases, even as its absolute counterpart drops. Decomposition results show a clear predominance of the contribution of very young ages over all the others. Decomposing the change in e_0 (Appendix, Figure B-2) shows that the contributions of young ages are greater than for the change in $e^\dagger$, while older ages contribute about the same to both measures. Here again, a disproportionate change in the denominator is bound to accentuate the contribution of young ages, while at older ages the ratio remains constant. Moreover, the threshold age for $\overline{H}$ (a^h) is much more stable than $a^\dagger$ (Aburto et al. 2019), so that it rarely drops below age 30, even during these crises. Therefore, the proportion of the age distribution above and below the threshold is more balanced.

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

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

8.5 Policy implications

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

the public debate should be accompanied by a conscious reflection on the meaning of the tools we use to measure it, so as to mirror the preferred conception of equality. A debate already exists about measures of broader health inequality (Asada 2010; Atkinson 2013). While it has not, to our knowledge, indicated any one measure (or kind of measure) as optimal,[7] it shows how contributions from different disciplines, such as philosophy and economics, are crucial in pushing the reflection forwards. Population scientists should also contribute to this reflection, but in this paper we can only offer an example of why this discussion matters for lifespan variation, and an exploration of the different properties of each measure.

9. Conclusion

Lifespan variation can illuminate some of the mechanisms of mortality crises. In our analyses we have found a common trend for the crises examined: by increasing relative lifespan variation and decreasing absolute variation, mortality crises shorten all (period) lifespans, but to unequal extents. The effect of crises on lifespan variation also differs across sexes, which is mostly due to discrepancies in the contribution of young ages. These results reflect the literature that has questioned the straightforward inverse relationship between e_0 and lifespan variation. Moreover, they underline the importance of older age mortality even in contexts with very high infant mortality. The reoccurrence of this pattern throughout our five cases suggests that it might exemplify the behaviour of human mortality under extreme conditions. The greater mortality of older individuals compared to children (Bhopal et al. 2021) suggests that our results could hold during the current pandemic, as shown by Aburto et al. (2021) for England and Wales in 2020. At the same time, the crises we have studied differ in a number of ways from the current pandemic (e.g., mortality at young ages, vaccinations, international ties) as well as from other disasters, and more analyses should be carried out on different kinds of crises. Longer and milder crises could present an interesting mid-way case study and could better show long-term effects of adverse conditions, as could analyses of the cohort dimension. Studying modern crises would show whether demographic, social, and geo-political changes have affected lifespan variation trends and could provide data for more detailed analyses. By comparing different kinds of measures we also hope to have contributed to the understanding of their characteristics and behaviours, so as to add to the debate around the use of absolute or relative measures of variation – although here more research is also needed to inform a debate that we hope will cross disciplinary boundaries.

[7] In fact, it can be questioned whether a single indicator captures 'true' inequality, as each indicator is based on different, and often contrasting, normative concepts of what constitutes inequality.

10. Contributions

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

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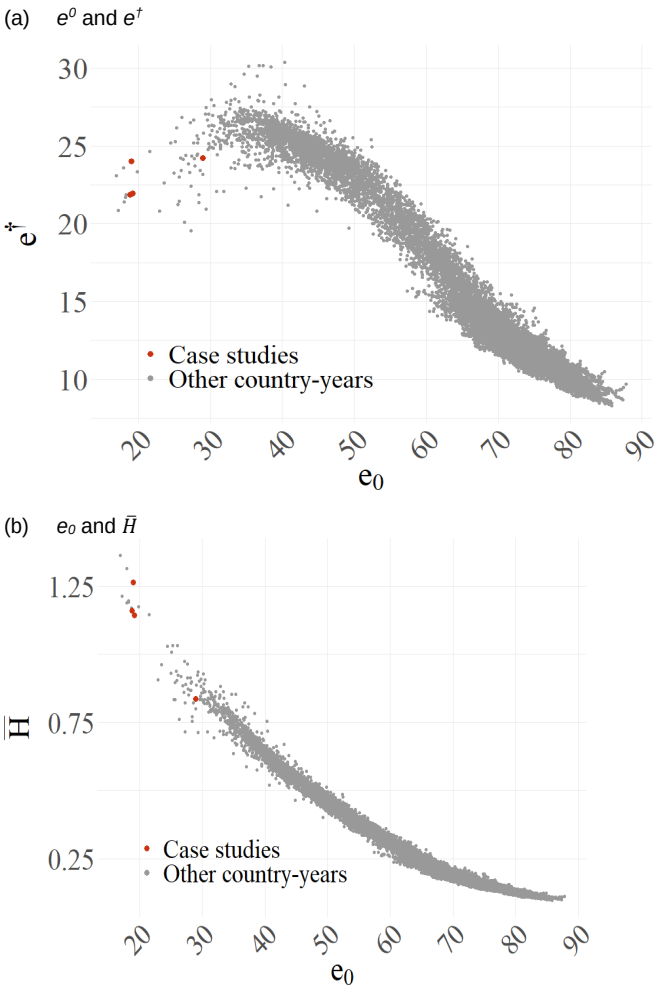
Appendix A

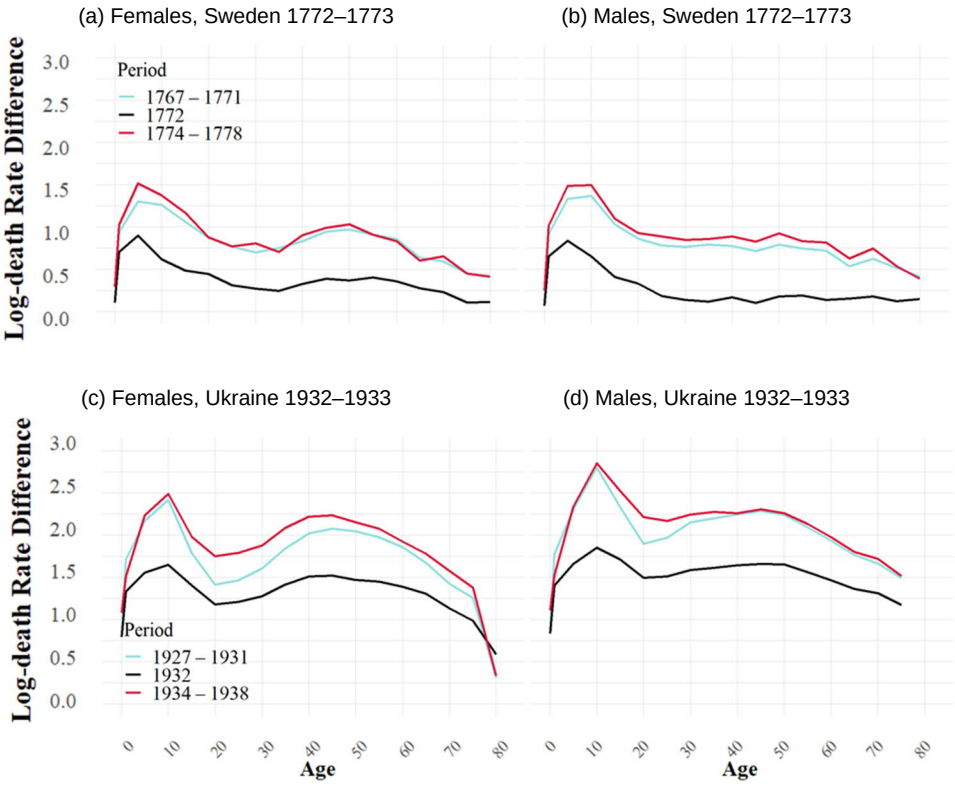
Figure A-1: Life expectancy and lifespan variation



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

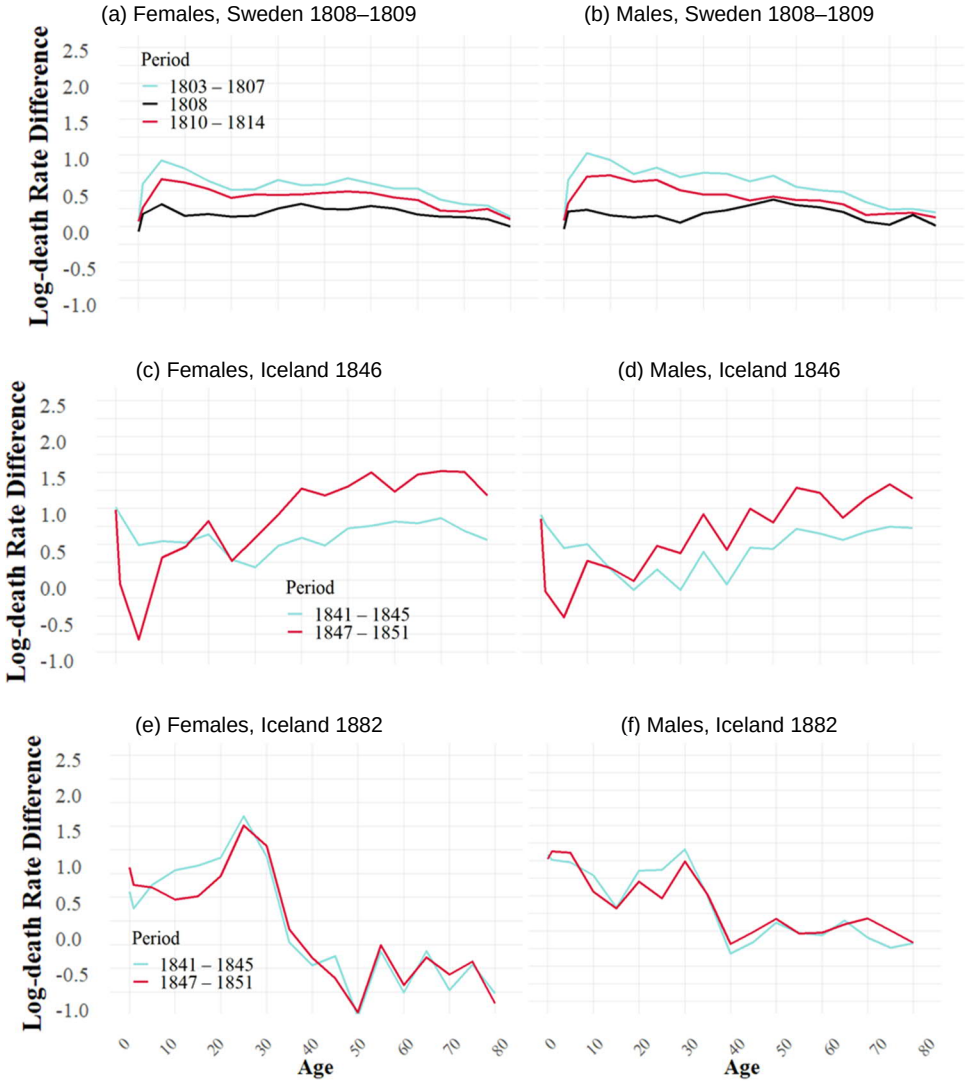
Appendix B

Figure B-1: Absolute differences in log death rates with main crisis year, famines



Source: HMD (Sweden) and Meslé and Vallin.

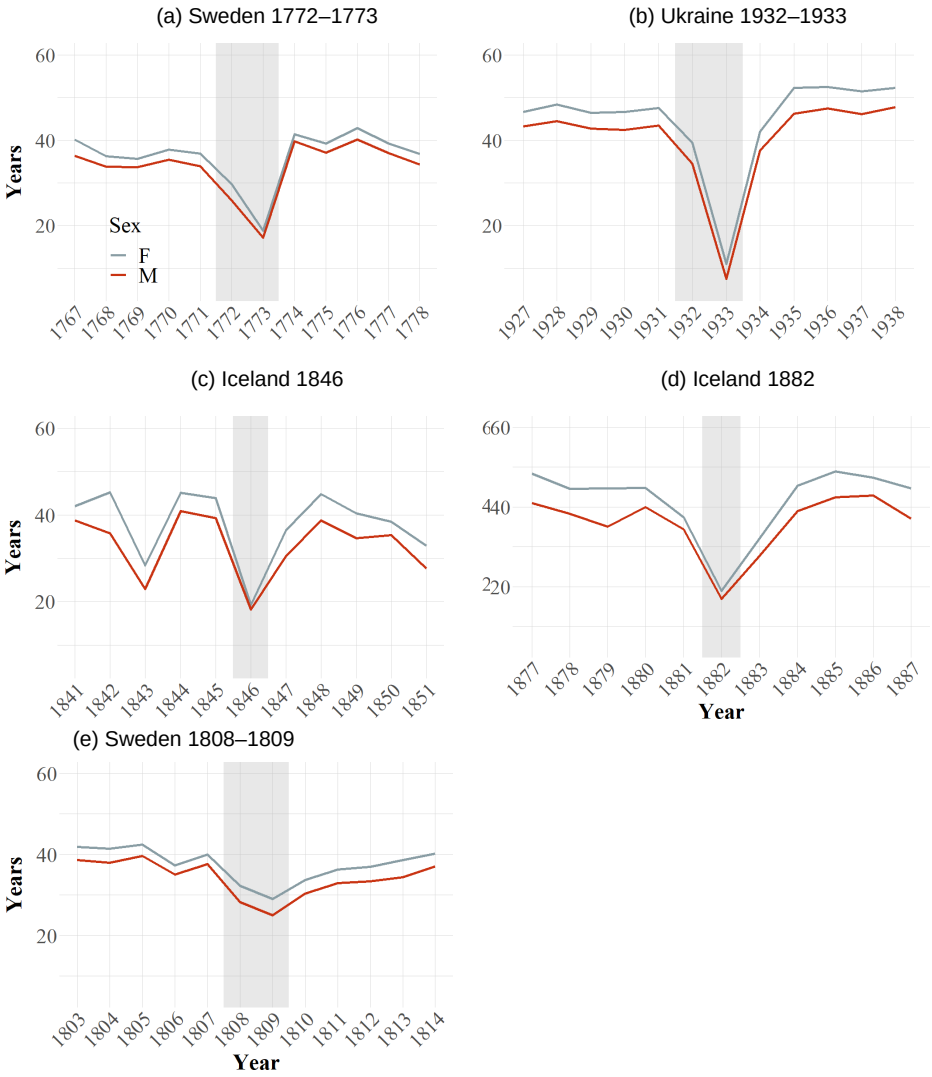
Figure B-2: Absolute differences in log death rates with main crisis year, epidemics



Source: HMD (Sweden and Iceland).

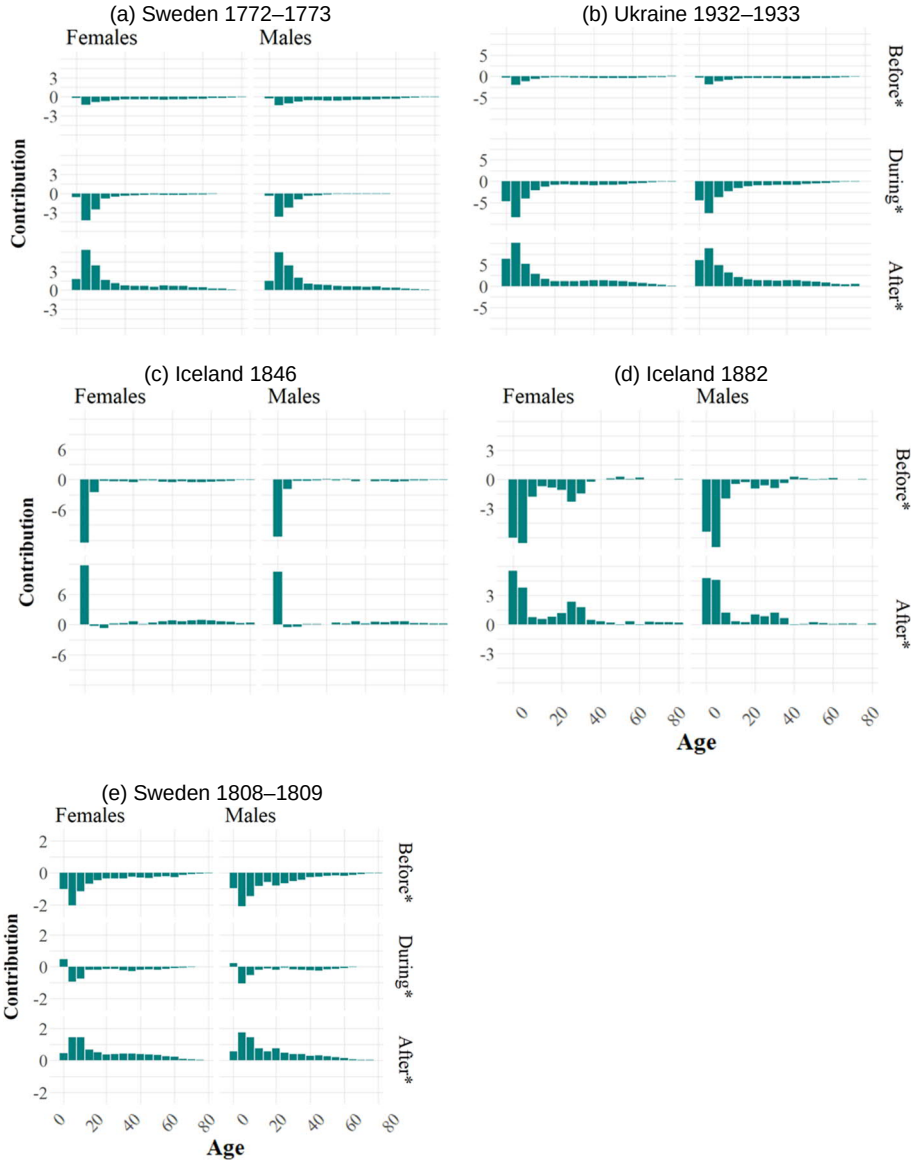
Appendix C

Figure C-1: Life expectancy trends



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

Figure C-2: Decomposition of e_0



Source: HMD (Sweden and Iceland) and Meslé and Vallin.
Note: * Before = change in indicator between the average of the previous 5 years and the (first) crisis year, During = change in indicator between the two crisis years, After = change in indicator between the (last) crisis year and the average of the following 5 years

Appendix D

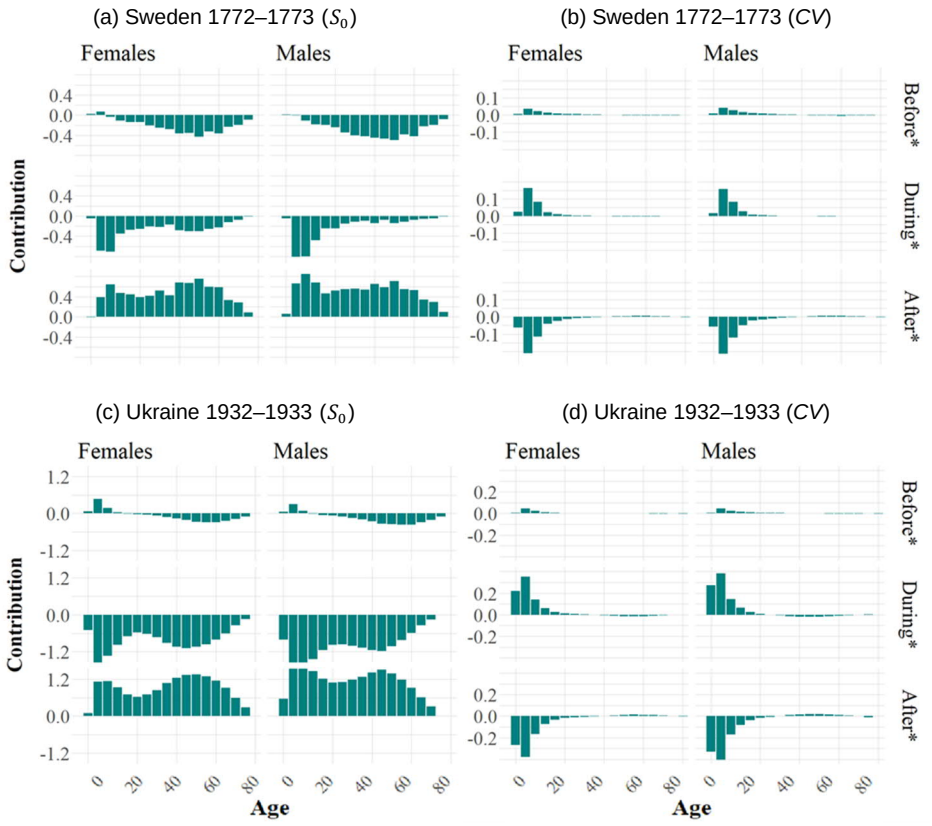
Table D-1: e_0 and threshold ages for $e^\dagger$ and $\bar{H}$

Measure	Sweden 1772–1773							
	Females				*Males*			
	Before	Pre-crisis	Crisis	After	Before	Pre-crisis	Crisis	After
e_0	37.31	29.65	18.83	39.85	34.61	25.91	17.18	37.59
$a^\dagger$	19.59	8.75	0	24.16	15.97	3.2	0	20.83
a^h	52.91	48	39.05	53	51.34	43.37	36.32	52.07
	Ukraine 1932–1933							
	Females				*Males*			
	Before	Pre-crisis	Crisis	After	Before	Pre-crisis	Crisis	After
e_0	47.31	39.44	10.85	50.11	43.3	34.46	7.3	44.82
$a^\dagger$	28.42	24.74	0	28.72	30.36	20.62	0	31.8
a^h	53.86	53.86	25.92	53.49	54.21	52.05	12.24	54.26
	Sweden 1808–1809							
	Females				*Males*			
	Before	Pre-crisis	Crisis	After	Before	Pre-crisis	Crisis	After
e_0	40.56	32.28	28.96	37.02	37.75	28.18	24.97	33.5
$a^\dagger$	28.69	14.62	8.54	23.96	25.96	5.61	1.74	18.68
a^h	53.45	50.67	47.19	52.7	52.38	47.42	42.84	50.13
	Iceland 1846							
	Females				*Males*			
		Before	Crisis	After	Before	Crisis	After	
e_0		39.02	19.19	38.13	33.8	18.22	32.85	
$a^\dagger$		28.62	0	8.49	19.74	0	3.57	
a^h		50.94	38.43	52.39	48	36.43	51.05	
	Iceland 1882							
	Females				*Males*			
		Before	Crisis	After	Before	Crisis	After	
e_0		43.74	19.01	46.42	37.35	16.94	37.1	
$a^\dagger$		28.28	0	30.17	22.69	0	19.01	
a^h		52.82	38.1	52.33	50.23	35.06	48.96	

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

Appendix E

Figure E-1: Decomposition of S_0 and CV, famines

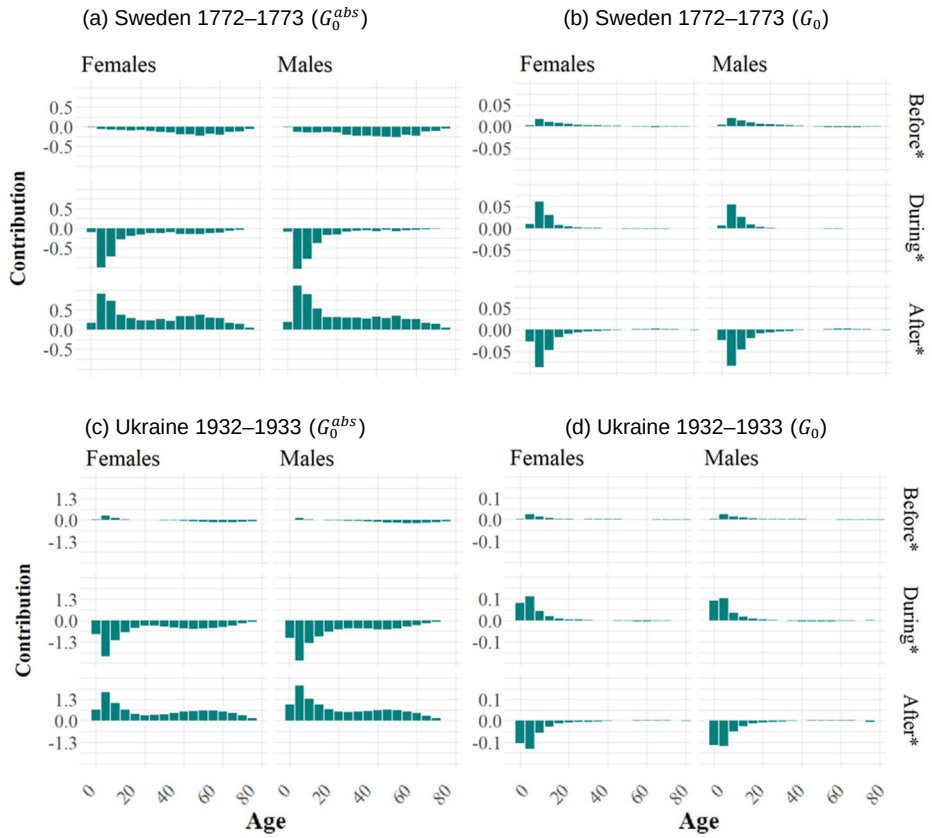


Source: HMD (Sweden) and Meslé and Vallin.
Note: * Before = change in indicator between the average of the previous 5 years and the (first) crisis year, During = change in indicator between the two crisis years, After = change in indicator between the (last) crisis year and the average of the following 5 years. S_0 indicates standard deviation, *CV* indicates the coefficient of variation.

Figure E-2: Decomposition of S_0 and CV, epidemics

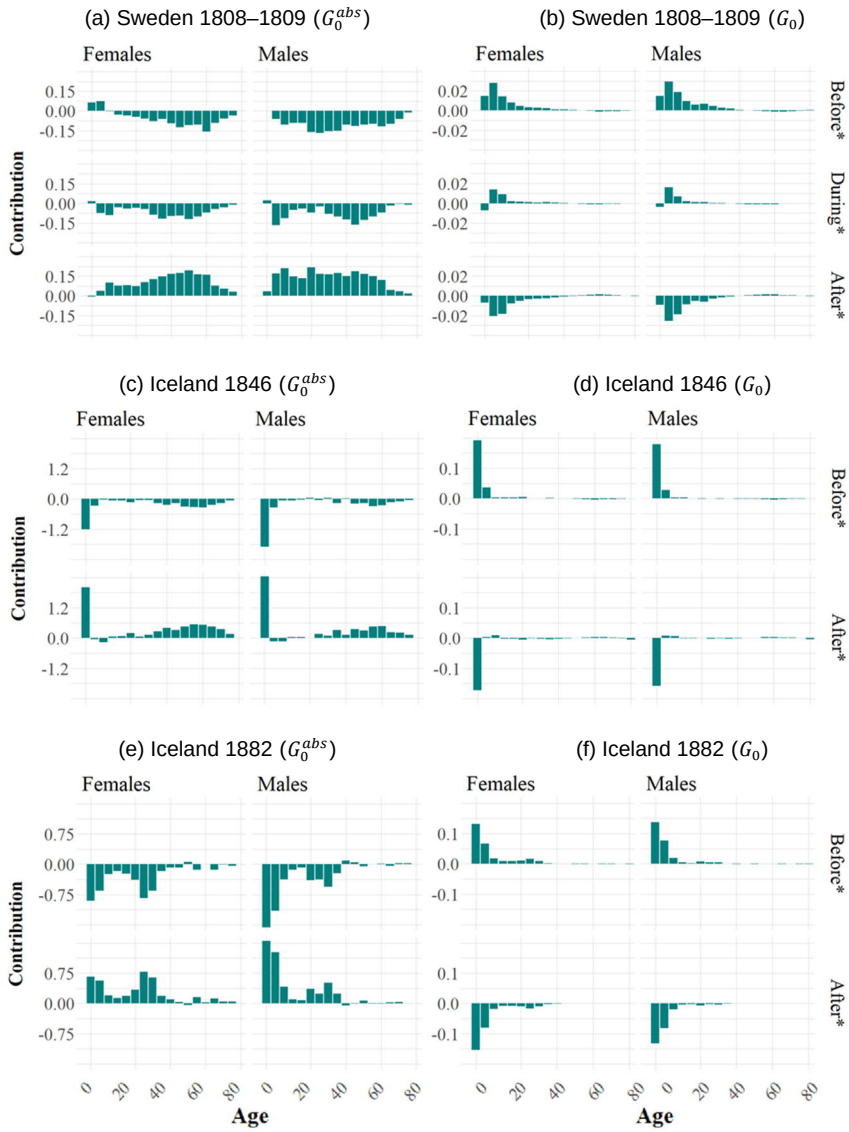
Source: HMD (Sweden) and Meslé and Vallin.
Note: * Before = change in indicator between the average of the previous 5 years and the (first) crisis year, During = change in indicator between the two crisis years, After = change in indicator between the (last) crisis year and the average of the following 5 years. S_0 indicates standard deviation, *CV* indicates the coefficient of variation

Figure E-3: Decomposition of G_0^{abs} and G_0, famines



Source: HMD (Sweden) and Meslé and Vallin.
Note: * Before = change in indicator between the average of the previous 5 years and the (first) crisis year, During = change in indicator between the two crisis years, After = change in indicator between the (last) crisis year and the average of the following 5 years. G_0^{abs} indicates the absolute Gini coefficient, G_0 indicates the relative Gini coefficient.

Figure E-4: Decomposition of G_0^{abs} and G_0, epidemics



Source: HMD (Sweden and Iceland).
Note: * Before = change in indicator between the average of the previous 5 years and the (first) crisis year, During = change in indicator between the two crisis years, After = change in indicator between the (last) crisis year and the average of the following 5 years. G_0^{abs} indicates the absolute Gini coefficient, G_0 indicates the relative Gini coefficient.