Implications of mortality crises in age at death

Compression or more lifespan variation?

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Introduction

Life expectancy is often used as a summary measure to describe the state of a population in terms of mortality, as well as in terms of health (van Raalte, Sasson, and Martikainen 2018). In this sense, life expectancy is indeed very convenient, as it expresses the average remaining years of life for a certain cohort, be it real or synthetic (Preston, Heuveline, and Guillot 2001). However, precisely because of this synthesis, 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 the modal age at death have increased (Colchero et al. 2016; Kannisto 2001; Smits and Monden 2009; Vaupel, Zhang, and Raalte 2011). 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. Greater lifespan variation has concrete implications on lifecycle investments and consumption, as individuals asses their chances of benefiting from such decisions in the future (van Raalte et al. 2018; Tuljapurkar 2011). In fact, although individuals are rarely aware 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 (Hurd and McGarry 1995).

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 and Scholl 2019; van Raalte 2011). 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. However, 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 fill this gap by focusing specifically on populations that have experienced a mortality crisis. Although such mortality crises happened in the past and are not a current event in contemporary Europe, recent evidence suggests that they will 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). By analysing and comparing the evolution of different populations, we aim to understand whether a regular pattern emerges which could precede or be a consequence of mortality crises. In this way, studying the patterns in lifespan variation of past populations could help us better understand the impact of mortality crises today. Moreover, we study the patterns in lifespan variation across age and gender. These results could be particularly helpful for organising the response to mortality crises in the future. Indeed, if some specific age groups are revealed to be especially vulnerable in such cases, the organisations responsible for the response will be able to better prepare and organise it.

Mortality patterns during times of crises have already been the object of research. Although these studies do not tackle the issue of lifespan variation, they do shed light on what we can expect from a broader point of view. First of all, no single pattern can be expected, as age and gender specific mortality rates vary, depending on the nature of the crisis itself. For example, male adults are more at risk during wars or some epidemics such as those caused by HIV/AIDS (Gaylin and Kates 1997; Hosegood, Vanneste, and Timæus 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). In this paper, we focus on two specific types of mortality crises: famines and epidemics. More specifically, we consider four 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.

Bongaarts and Cain (1982, in Kane 1987) hypothesised that 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. Age and gender specific mortality patterns vary depending on the cultural and social environment. Some information about Scandinavian trends can be found in Bengtsson, Campbell and Lee (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 indications 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 (Australian Institute of Health and Welfare 2010), while malaria, an infectious disease, affects predominantly young children (World Health Organization 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. The last crises we analyse was a typhus and dysentery epidemic, diseases which kill especially weakened individuals, such as children and the elderly (Castenbrandt 2014).

Context

Dribe, Olsson and Svensson (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 that crude death rate 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 than from war itself (Glei et al. 2019). 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.

In Iceland, we consider two measles epidemics, in 1846 and 1882. In both years, particularly cold spring and summer forced fishermen to concentrate in shore 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 that 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, Haggett, and Graham 1983; Shanks et al. 2015).

Research questions

In this work, we will answer three main research questions.

First, we want to study whether lifespan variation changes before, during and after a mortality crisis. In order to provide an answer, we will compute a lifetable using the average age-specific mortality rates for the five years prior to the beginning of the crisis and compare the lifespan variation thus obtained to the one measured during the crisis year(s) and in the following years. One could expect that a severe enough crisis would cross social lines, affecting the whole population equally, as happened for the European Black Plague (Livi Bacci 2012), so that lifespan variation would decrease in such situations. However, more recent episodes have shown clear inequalities in the mortality during extreme events,

famously hurricane Katrina (Zoraster 2010). In fact, most of our data is connected to famine episodes, from which the wealthy are protected to a certain degree. Even if a crisis were to cross social lines, it would likely affect individuals differently depending on their age, disproportionately increasing the mortality of the extreme and more vulnerable ages and thus variation. Finally, a decrease in life expectancy, which is inevitable during a mortality crisis, gives mechanically more space for variation in age at death, as the modal age at death shifts to the left. All of these considerations lead us to believe that variation will increase rather than decrease during a mortality crisis. After the end of the episode, we expect that variation will continue to be higher than pre-crisis level, but that it will gradually decrease as the parts of the population most affected by the crisis recover. This trend could be balanced by a reduction in lifespan variation due to a selection, during the crisis, of the more robust individuals, who would die later on average.

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, we expect that mortality crises will affect both subpopulations similarly, so that the gender gap in lifespan variation, which generally favours females (van Raalte 2011), will not change in high mortality situations.

Our final question asks whether some ages especially contributed to the change in variation witnessed 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.

Data and Methods

We use data from the Human Mortality Database (HMD, www.mortality.org). The HMD supplies data covering multiple populations with nearly complete information, assuring a high level of quality. For this reason, only few countries, mostly European, are included in the database and fewer yet contain data series from the XVIII or XIX centuries, where mortality crises were more common in Europe. We have chosen to focus on Scandinavian countries, for which data were collected by parishes at a reasonably precise level during this time. More precisely, we use these data to study four mortality crises, two in Sweden and two in Iceland.

Lifespan variation indicators

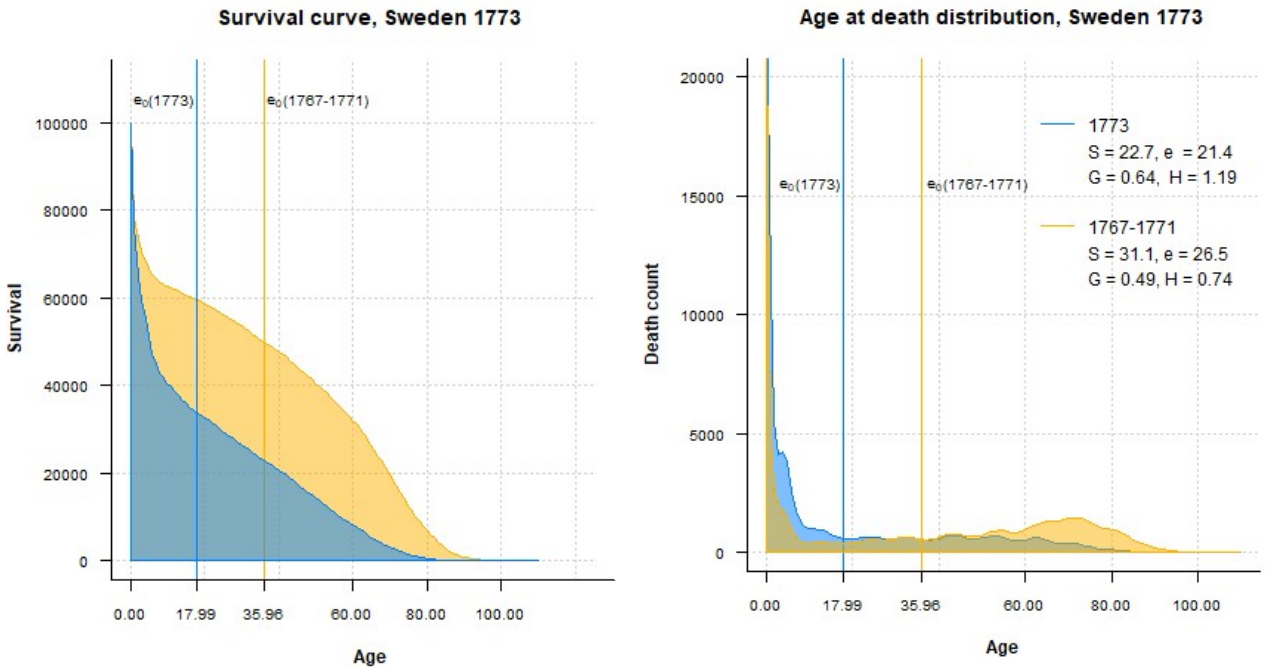
Lifespan variation can be measured using a wide range of techniques and all of them have been found to be highly correlated in empirical datasets when measured from young ages (Wilmoth and Horiuchi 1999). They are not, however, completely interchangeable. As van Raalte and Caswell (2013) point out, they differ in their formal properties and in the underlying concept they gauge. Following the authors' analysis, we have decided to include four measures of lifespan variation in this work: the standard deviation S, life disparity $e^{\dagger}$ (Vaupel and Canudas-Romo 2003), the Gini coefficient G (Shkolnikov, Andreev, and Begun 2003) and the lifetable entropy $\overline{H}$ (Keyfitz 1977; Leser 1955). These represent a mix between absolute and relative measures of variation. It is useful to consider relative variation for comparisons, especially when considering dramatic changes in life expectancy, which could mask significant differences. At the same time, absolute variation is a more directly understandable measure and can better inform us on the concrete changes experienced by our populations. Moreover, using various methods with different sensitivities to changes in age-specific mortality rates will allow us to come to more robust conclusions. We also prefer these to other measures, because they are easily interpretable. S is the square root of the variance, which is itself the average squared distance from the mean age at death, meaning that S is measured in years. Life disparity $e^{\dagger}$ is the remaining life expectancy at death and can be interpreted as the average life years lost at death. Considering measures of relative variation, G is the average distance between each individual's age at death, expressed as a proportion of life expectancy. Finally, $\overline{H}$ represents the elasticity of life expectancy to a proportional change in mortality, or the variation in the length of life as a proportion of life expectancy (Aburto et al. 2019; Wrycza, Missov, and Baudisch 2015). While the measures of absolute variation we have chosen are expressed in years, this is not the case for G and $\overline{H}$, which are both expressed as a proportion of life expectancy.

Decomposition methods

Although studying the variation in the mortality distribution will already inform us on its general trend, we also plan to analyse these changes more in detail, as the information included in the HMD makes it possible to identify gender and age at death. We will first compare lifespan variation differences between males and females, to study the behaviour of the gender gap under crisis conditions. Then, we will decompose the change in variation by age, to understand whether certain age groups contributed more to the increase or decrease in

lifespan variation. We will do so by using the life table response experiments (LTRE) method, which expresses the observed change in the value of a function, in our case one of the measures of lifespan variation, as a combination of the sensitivity of this function to its parameters and of the changes in the parameters themselves, in our case, age-specific mortality rates (Caswell 2010). Although other methods of decomposition by age have been developed (see for example Appendix B of Wilmoth and Horiuchi 1999), LTRE has been shown to be applicable to the measures we consider through relatively straightforward calculations (van Raalte 2011).

Preliminary Results



In blue, survival curve, age at death distribution and life expectancy for the total population of Sweden in 1773. In yellow, survival curve, age at death distribution and life expectancy for a synthetic population, derived from the average mortality rates for the total population of Sweden between 1667 and 1771. Th vertical lines indicate life expectancy. In the legend, S is the standard deviation of the age at death, e indicates life disparity $e^{\dagger}$, G is the Gini coefficient and H the lifetable entropy $\overline{H}$.

Figure 1 – Survival curves and age at death distributions for Sweden, in 1767-1771 and 1773

Figure 1 shows the Swedish survival function and age at death distributions for 1773 and for a synthetic lifetable computed from the average death rates for the period from 1667 to 1771 (1772 was not included in this period, as the famine had already started by then, but sensitivity checks will be conducted). We see that the survival curve for 1773 is much steeper at young ages than for the previous years. Moreover, while the yellow curve shows some signs of rectangularisation, the blue one is much hollower, with a relatively constant steepness

from around 10 to 75 years. On the contrary, the pre-crisis survival distribution decreases more slowly between 10 and 50 years circa, to drop afterwards. The corresponding age at death distribution tells us the same story. During the crisis year, more people died between birth and a bit under 30 years of age than in the previous period. However, from 18 to 60 years, the death count remained constant in 1773, while the previous years saw the usual increase tied to old age, so that after 50 deaths in the pre-crisis period actually overcame those during the famine.

When looking at the values for life expectancy and lifespan variation, we see some surprising results. Unsurprisingly, life expectancy at birth dropped from 36 to 18 years, mirroring the dramatic worsening of living conditions. The two measures of relative lifespan variation, G and $\overline{H}$, increased, following the known inverse relationship between life expectancy and lifespan variation. The measures of absolute variation S and $e^{\dagger}$, however, decreased sensibly in 1773, indicating that absolute lifespan variation was lower during the famine than in the previous years.

Discussion

These preliminary analyses yielded two main results. The first concerns the relationship between life expectancy and lifespan variation. While an inverse association has been shown to prevail in the long term (Zarulli et al. 2018), it seems that this trend can be reversed under extreme conditions. These exploratory results suggest that, contrary to what we had expected, the 1773 Swedish famine reaped victims throughout the population. The decrease of absolute variation in 1773 suggests that the increased burden of mortality might have been more largely shared than anticipated. Our second result relates to the choice of lifespan variation measure. Indeed, our analyses show that, under the same conditions, indicators of absolute and relative variation can behave in opposite ways. Especially at times when life expectancy changes drastically, the preference of one underlying construct over the other should be an object of careful consideration.

Naturally, the results that we have presented here only relate to one of the four cases we plan to analyse and do not examine differences across ages and gender. Our future analyses aim at understanding whether such trends of lifespan variation are generalizable to other periods of crisis and from which population subgroups they arise. Finally, by considering patterns in variation during the years following the crises we will understand what the long-term effects of such events can be.

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