Abstract

1 Introduction

Life expectancy is often used as a summary measure to describe the state of a population in terms of mortality, as well as in terms of health (van Raalte et al. 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 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 the modal age at death have increased (Colchero et al. 2016; Kannisto 2001; Smits and Monden 2009; Vaupel et al. 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. 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 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 et al. 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. 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. Although such 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). The vulnerability of countries across the world to such events was made evident by the ongoing pendemic of COVID-19 and this research has become all the more relevant in order to understand the mechanisms and consequences of this crisis. 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 in assessing a response in the wake of the COVID-19 pandemic and for organising the response to mortality crises in the future. Indeed, our results can shed light on the potential consequences of mortality crises for the inequality in age at death, and on the distribution of mortality in such occasions.

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, following the order of presentation of our research questions. In the conclusion, we take 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 Finland (Brønnum-Hansen 2017, van Raalte et al. 2014). 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. For this reason, we adopt the framework of ageing proposed by Baudisch (2011), which distiguishes two separate dimensions. On the one side, the pace of ageing identifies the scale on which mortality progresses and can be quantified by a variety of measures, life expectancy being one of the most common. On the other, the shape of ageing expresses how rates change with age. Measures for this second dimensions should be "time-independent estimators of the degree of change in pace-standardised mortality over age" (Wrycza et al. (2015, p. 9)), further highlighting the independence (at least from a theoretical perspective) of pace and shape. Although we wish to integrate this framework, we will also go beyond it, by including some measures of lifespan variation, which cannot be considered measures of shape, as will be delineated in the methods section.

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), brought about by a lack of food, or by its unavailability to a considerable part of the populatio; epidemics, caused by diseases;

crises which combine famine and epidemic, most often in this order as the famine weakens the population leaving it vulnerable to pathogens (as indeed happens most of the time, as individuals rarely die of hunger alone); crises with 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.

In the next part of this section, we present some known trends tied with famines and epidemics. 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, 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 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. 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 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 (noa 2010), while malaria, an infectious disease, affects predominantly young children (noa 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. As anticipated, we focus on two specific types of mortality crises: famines and epidemics, together with their combination. More specifically, 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 ~~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 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 uring 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

Our analysis is structured around 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 compare the values of 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. We expect to see a clear change in lifespan variation during crisis years, as diseases and lack of food would likely affect individuals differently depending on their age, disproportionately increasing the mortality of the extreme and more vulnerable ages. Thus, we predict to see an increase in lifespan variation during the crisis. Moreover, 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. 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.

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

Various measures have been suggested for this second dimension. Although a number of desirable properties have been identified by Wrycza et al. (2015), to our knowledge one that can be proven to respect them all has yet to be found. Still, a number respect

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 ~~or δ~~ 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 $G_0^a bs$ 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

$$e^\dagger = \int_0^{\inf} \mu(x) l(x) e_\theta(x) dx \tag{1}$$

while the lifetable entropy is

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

In a similar way, the standard deviation is

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

and the coefficient of variation

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

Finally, the absolute Gini coefficient is

$$G_0^abs = e_0 - \frac{1}{[l_0]^2}\int_0^{\inf}[l_0]^2 dx \tag{5}$$

and its relative counterpart becomes

$$G_0 = \frac{G_0^abs}{e_0} = \frac{e_0 - \frac{1}{[l_0]^2} * \int_0^{\inf}[l_0]^2 dx}{e_0} = 1 - \frac{1}{e_0[l_0]^2}\int_0^{\inf}[l_0]^2 dx \tag{6}$$

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 allow comparisons between different populations. Although this characteristic is often used within the pace and shape framework to compare results across species (see for example Jones et al. 2014), it can be also convenient when comparing human populations in starkly different circumanstances, as is the case when a population experiences drastic changes in mortality.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. However, 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 inequality could be a better fit for the conception of inequality, 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. hor (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 $c_i = \int_{x_i 1}^{x_i 2} \frac{\partial y}{\partial x_i} dx_i$ 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. These tests should also address concerns about age heaping and age-class splitting. 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.

5 Results and discussion

6 Conclusion

Appendix A

References

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