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Mortality Shocks and the Human Rate of Aging

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Mortality shocks and the human rate of aging

Virginia Zarulli*

Abstract

Investigating the effect of mortality shocks on humans is difficult in the absence of laboratory experiments. However, some events in human history serve as natural experiments. Using data for Australian prisoners during WWII and for the Ukrainian Famine in 1933, I analyzed the effect of sudden changes in external conditions on the rate of aging. This may help to decide whether the rate of aging is sensitive to the environment or is stable. The mortality of the prisoners of war was higher during the imprisonment but the slope of the curve did not change. During the Ukrainian Famine, the curves in the years of crisis converged at old ages. By adopting a cohort perspective I found evidence of selection that could be the cause of the convergence. The analysis suggests that sudden and transitory exposure to severe conditions shifts the mortality curve upward proportionally at all ages, leaving the rate of aging unchanged.

Keywords rate of aging, shock, mortality, famine, prisoners of war

1 Introduction

It is well known that environmental conditions affect mortality of populations. Lifespan can be extended or contracted either by lowering or raising the mortality curve proportionally or by a change in the slope of the curve, also called the rate of aging. The debate is open on whether changes in external conditions produce either one or the other mechanism.

Several experiments conducted on diverse organisms analyze the effect of environmental changes and genetic modifications on survival. Some of these

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studies find that the intervention changes the slope of the mortality trajectory significantly (De Magalhães et al. 2005; Johnson 1990). Others find no effect on the rate of aging (Flurkey et al. 2001; Mair et al. 2003; Magwere et al. 2004).

Brief and intense stress, like sudden dietary restriction or exposure to desiccating air flows applied to Drosophila, cause mortality to switch rapidly between the case and the control group (Aziz et al. 1995) and seem to have no impact on the rate of aging. Single shocks are also likely to influence initial mortality only, while repeated mild shocks throughout life seem to affect the rate of mortality increase as well (Wu et al. 2009). Whether genetic interventions affect the rate of accumulation of damage is more controversial (Partridge et al. 2005).

Investigating how death rates of human populations react to sudden changes in external conditions and exposure to extremely harsh situations is more difficult because, fortunately, it is not possible to set up laboratory experiments with humans. However, recent human history provides some documented situations of "mortality shock", like famines, deportations, internments, that can be used to address these questions.

I investigate the question whether these shocks have an impact on the rate of aging or mainly shift the mortality curve proportionally at all ages. Studying the effect of catastrophic events on human mortality helps to decide whether the rate of aging is biologically determined and stable or is sensitive to sharp environmental changes.

These shocks, also referred to as "natural mortality experiments", serve as a proper tool of analysis because they resemble the experimental condition called a "natural experiment". They affect the whole population not selectively and this helps reducing the possibility of bias due to confounding factors in the evaluation of the effect of the treatment.

Famines are good examples of mortality shocks and the literature about them is wide. Depending on the availability of data they have been more or less extensively analyzed. Examples of sadly famous famines are the great Finnish Famine in the 1866-68 (Pitkänen and Mielke 1993; Pitkänen 1992, 1993), the Dutch Famine in 1944-45 (Lumey and Van Poppel 1994), the Ukrainian Famine in 1932-33 (Meslé and Vallin 2003), the Potato Irish Famine in 1840 (Guinnane et al. 2002) and the devastating famine that accompanied the Chinese great Leap Forward in 1958-61 which caused around 30 millions excess deaths

(Ashton et al. 1984; Peng 1987; Song 2009).

A substantial number of studies exist about imprisonment in war camps or in cities under siege, that are other good examples of mortality shocks.

However, the great majority of research conducted so far has not focused specifically on what kind of effect such events have on the rate of aging. Several studies are concerned with assessing whether the cohorts born during a famine suffer from a higher late life mortality than the cohorts born right before and right after (Doblhammer-Reiter et al. 2011; Kannisto et al. 1997; Myrskylä 2010; Song 2009) but the results are contradictory. Others look into the effect of starvation and malnutrition early in life on specific health outcomes, like blood pressure and diabetes, in adulthood (Sparn et al. 2004; Stanner et al. 1997; Lumey et al. 2007; Painter et al. 2005; Chen and Zhou 2007).

Australian veterans surviving Japanese WWII camps showed a higher overall mortality rate than non-prisoners in the years after release (Dent et al. 1989), although the difference was more pronounced in the years immediately following the release and diminished after a longer time. The same pattern has been found also for Holocaust survivors (Williams et al. 1993), and this draws the attention on the role of selection processes. On the other hand (Costa 2011) finds that, for the survivors to the military camp of Andersonville during the American Civil War, what matters in determining better or worse conditions compared to the non internees is age at imprisonment.

Looking for cases of natural experiments in human mortality and data to use I collected some cases, although I am aware that the list is obviously not exhaustive. This includes Australian prisoners of war surviving to the Japanese camp of Tjimahi in Java during WWII, the Ukrainian famine in 1933, the case of freed American slaves emigrated to Liberia in the nineteenth century and the Finnish famine in 1866-68.

The next section of the paper illustrates these cases and describes the data available for each of them. Sections 3 and 4 illustrate the methods and the results for the cases selected for the analysis (Japanese prisoners of war and Ukrainian Famine). The last section discusses the results along with concluding remarks.

2 A selection of natural mortality experiments

2.1 Australian prisoners of war 1944-45 in the camp of Tjimahi

After the end of WWII, Bergman (1948) wrote about the Japanese concentration camp of Tjimahi, in Java, where he had been interned and had practiced general medicine. He reported that at the end of February 1944 more than 10000 Australian male civilians between age 10 and 85 were interned in the camp until August 1945. Dietary and living conditions were extremely harsh.

He compared the death rates in the camp [1] with the death rates of the Australian male civilian population in 1941, that can be considered as reference mortality level in normal regime, and noticed that they were extremely higher at all ages (Figure 1a). He compiled tables of monthly death counts by 5 year age groups from age 11-15 to 81-85, from March 1944 to August 1945. He also compiled a table on the number of individuals by age group that were interned at the beginning, March 1944, and how many individuals by age groups were alive in January 1945.

These data have been cited over the last decades as an example of death rates that increased proportionally for all ages during the exposure to harshness, suggesting that adverse environments throughout life do not strongly influence mortality rates acceleration. Jones (1959, 1961) included them in his discourse on the relation of human health and mortality to age, place and time, and about the mechanism of aging in altered death risks. Finch (1994) plots the age specific death rates during the internment on a logarithmic scale and adds the log mortality curve for the US population in 1980 to show that the lines have the same slopes. More recently, they have been used by Rozing and Westendorp (2008) in their article where they not only criticize the classic approach of looking at the parallelism of the log-mortality lines (via linear regression on log transformed data), but firmly question the validity of the slope of the log mortality curves as an estimate for the rate of aging.

Whatever was the point of view of the analysis that have used these data so far, none went beyond the study of the yearly death rate in the camp, neglecting the additional available information represented by the monthly

[1] Please not the Bergman calculated probabilities of death in the camp, although he called them death rates.

death counts. Such additional information provides the possibility to extend the analysis and gain a deeper insight on the question whether the internment period affected or not the rate of aging.

2.2 The great Ukrainian famine in 1933

In the twentieth century Ukraine experienced particularly turbulent demographic trends that mirror a history of major crises. Meslé and Vallin (2003) made an interesting and fascinating historical and demographic reconstruction of these events.

Among the republics of the Soviet Union, Ukraine was one of the most strongly affected. Several famines, wars, invasions, deportations and waves of repression followed one another and for many years they were kept secret. Among these was also the great famine in 1933 that followed the collectivization of agriculture. When perestroika made possible to access the archives, it become clear that the authorities had tried to hide these disastrous events.

From 1931 to 1954 no statistics on population were published, the 1939 census was falsified and the deaths counts were affected by underregistration. Although imperfections and gaps still occur in data, the newly opened archives give researchers the possibility to improve the estimates, relying on the 1926, 1939 and 1959 censuses, and combining different sources.

The estimates on the great famine in 1933 report about 2.9 million individuals missing because of excess mortality, 1.1 million due to lower fertility and 0.9 million who migrated or were forcibly deported or exiled. They also show an incredibly low period life expectancy during the crisis. In 1931 life expectancy at birth was 43.5 for men and 47.9 for women, while in 1933 it fell to 7.3 and 10.9 respectively and went back to normal levels in 1935.

The authors reconstructed and made available several data series. Among these, period life table probabilities of death for men and women, by 1 year age group from age 0 to 89 for the years 1927-2002 are particularly relevant to my research question, because they allow to investigate the impact of the great famine in 1933 compared to previous and following years (Figure 1b).

2.3 Freed American Slaves emigrating to Liberia in the nineteenth century

Between 1820 and 1843 freed slaves from the USA were encouraged to migrate back to Africa. Many of them decided to undertake this risky trip and migrated to Liberia. All of them were born and grew up in the USA, a disease environment certainly very different from the one they were going to face in Liberia.

Using data collected by the American Colonization Society from 1820 to 1843 McDaniel (1992) estimated life tables reflecting the mortality experience faced by these slaves in their first year of arrival and in the subsequent years, showing one of the highest mortality ever registered.

The arrival in Liberia was a shocking event concerning external environmental conditions. In fact, about 43% of them died on the first year, when life expectancy at birth has been estimated to be 1.68 years for men and 2.23 for women. However, those who survived the shock of the first year saw their mortality probability drastically reduced. The estimated life expectancy, conditioned on survival through the calendar year of arrival, jumped to 23 and 25 years for men and women respectively.

The recovery after the shock is significant but, if compared to the most robust life expectancy estimates of black slaves in the USA in the nineteenth century (33 years for men and 34 for women (Eblen 1974)) it shows how penalizing the migration to a completely different and more hostile environment was. Two sex specific life tables by five years age groups from age 0 to 70 are available, with and without conditioning on survival to the first year of arrival, making it possible to compare the death rates during the shocking transition and after it (Figure 1c).

2.4 The great Finnish Famine in 1866-68

During the eighteenth century Finland experienced intermittent crop failures followed by epidemic diseases but their impact was usually negligible. In the 19th century, however, two major demographic crises hit the country. The War of Finland in 1808-09, connected with the Napoleonic wars all around Europe, seems to have catalyzed epidemics that raised mortality to very high levels. The second crisis arrived at the end of the 1860s, when a series of poor harvest

started in 1862, continued in 1865 and, after the total crop failure in 1867, caused a devastating famine.

Although at the time of the famine Finland was a rural and poor country, the source of materials and documentation of this event is quite rich, thanks to the long tradition of accurate historical records in the Scandinavian countries. In fact the Finnish famine has been extensively analyzed.

In the most affected regions, mainly the northern and central ones, the death rates reached levels of 200 per 1000. Finland, which in 1865 had a population of 1.8 million, lost more than 100,000 individuals over the famine. The economic and social turmoil caused by the famine left marks for many years (Pitkänen and Mielke 1993; Pitkänen 1992).

Age and sex specific death rates for the Finnish population by 5 years age groups, from age 0 to 80+ are available for the years 1861-65, the years immediately before the crisis, and for 1866, 1867 and 1868, when the crisis reached its peak (Figure 1d).

The log mortality rates for the 4 cases (Figure 1) clearly present a similar pattern: during the shock the mortality curves shift up or down in a parallel fashion. In the case of the Ukrainian and Finnish Famines the curves tend to converge at old ages. However this convergence is visibly more pronounced in the second or third year of the crisis and almost imperceptible before.

Parallel curves may indicate that the shock acts proportionally on the overall level of mortality. On the other hand, curves with different slopes might denote an impact on the rate of aging. However, in the case of multiple years of crisis, convergence at old ages could result from a selection process of the more robust individuals year by year (Vaupel et al. 1979; Vaupel and Yashin 1985). This convergence, moreover, is likely to be more pronounced in the second or third year of the crisis, when selection has had some time to take place.

To analyze whether the rate of aging is affected by the shock and to investigate the presence of selection I will focus on the Australian prisoners of war and on the Ukraine famine because, among the data at my disposal, they are the richest.

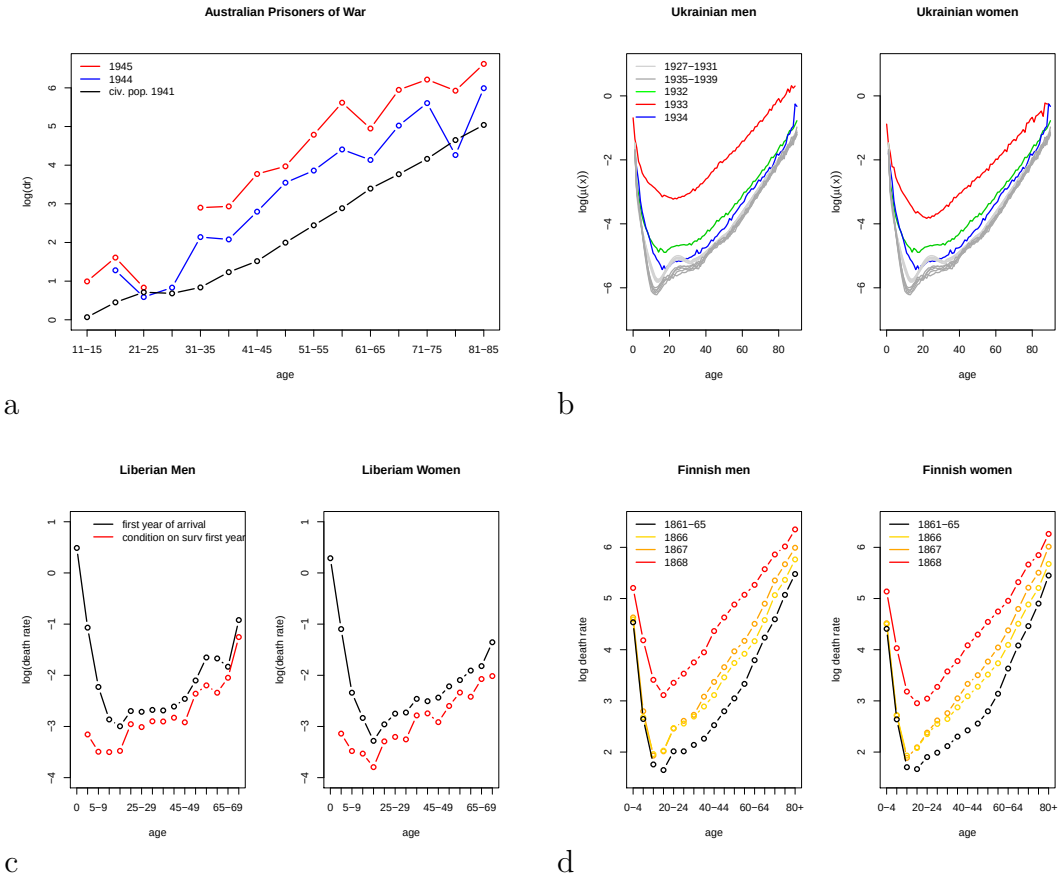

Figure 1: Four Natural Mortality Experiments: Australian Prisoners in WWII (a), Ukrainian famine in 1933 (b), US freed slaves migrated to Liberia in the nineteenth century (c), Finnish famine in 1866-68 (d). Y axis reports log mortality, a and d plots report log death rates per thousands, plots b and c report log death rates.

3 Parallel lines in the Australian Prisoners in WWII

As a first step, to investigate whether there are significant differences in the rate of aging between the normal mortality regime and the mortality in the internment camp, I fitted the Gompertz mortality model (Gompertz 1825), from age 30 onwards, to the death rates of the Australian male civilian population in 1941 from the Human Mortality database and to the death rates in the camp in 1944 and in 1945. To obtain the camp death rates I calculated the exact exposure time starting from the population alive at the beginning of the internment and subtracting the number of deaths month by month. From the paper we know that the camp was not completely closed and small move-

ments of prisoners happened throughout the period. Fortunately, a mid term information about how many individuals were alive in January 1945 allows to cope with this problem and to count the exposure more precisely.

According to the Gompertz model, at any age x, the force of mortality $\mu(x)$ is expressed by an initial mortality level, a, and by an exponential mortality increase by age, the parameter b, which is also defined as the rate of aging (Shock 1967; Finch 1994) and represents the slope of mortality curve on logarithmic scale:

$$\mu(x) = ae^{bx} \tag{1}$$

Previous analysis of these data used linear fitting on the empirical log-death rates. However, the logarithmic transformation tends to flatten and level out possible differences. Using a different approach I performed weighted non linear least squares regression on the empirical age death rates. Using not weighted regression yielded to high standard errors that resulted in non significant a parameter in 1944 that hindered the assessment of the differences between the curves in the three years.

Figure 2 shows empirical and fitted values. The curves on arithmetic scale show how much higher mortality in the camp was, diverging more and more from the normal mortality regime at older ages. However, on log scale the picture appears different. The three curves have very similar slopes, suggesting that the rate of mortality increase by age was not accelerated but internment period. This is confirmed by the confidence intervals for the estimates of the parameter b in the three years, which overlap and indicate not statistically significant difference. The difference in the initial level of mortality between the normal regime and the internment regime a, instead, is significant. Therefore, it can be said that mortality during the period of interment was significantly higher, proportionally at all ages (More details about the coefficients are reported in Appendix A, Table 1).

I then looked more deeply into the monthly information. There is a dominant monthly pattern in all age groups that reflects the seasonal pattern typical of tropical areas, subjected to the alternating cycle of dry months/humid months driven by the monsoon season. Mortality in dry months (from May to September) is typically lower than in the humid months (Shek and Lee 2003).

The monthly mortality counts, however, also reflect the camp dynamics. One change is most important above all: in March dysentery spread in the

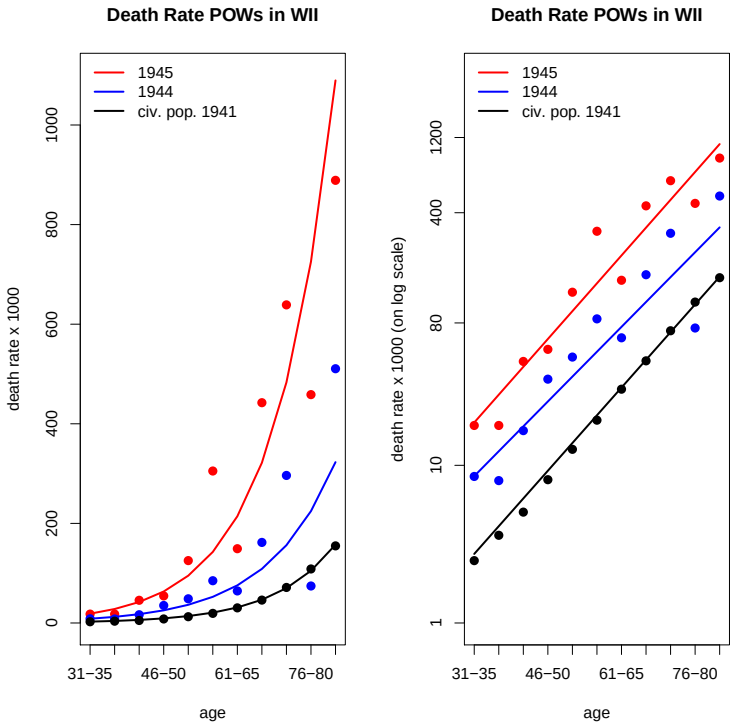

Figure 2: Australian male civilian prisoners of war in WWII. Empirical death rates and Gompertz fit in interment regime (1944-45) in the camp of Tjimahi (Java) and Australian male civilian population in 1941 (considered as normal mortality regime).

camp but the Japanese authorities, in order to prevent a rise in mortality, sent the sick ones to nearby hospital camps and did not allow anybody to die in the camp. Consequently the death rate of the camp fell (Bergman 1948).

For the analysis, I used a generalized linear model with Poisson log-link function. According to the literature, the Poisson distribution is the most appropriate distribution for modeling rates (King 1988; Cameron and Trivedi 1998), that are based on the count of events occurring in a certain amount of time.

In the first model I used an age variable and one month variable for each of the 18 months spent in the camp. In a second model I introduced the interaction between age and month. For every $month_i$, with $i = 1, ..., 18$, the logarithm of the death rate in that month is:

$$model1 : \log(\mu_i) = \beta_0 + \beta_1 age + \beta_{2,i} month_i$$
$$model2 : \log(\mu_i) = \beta_0 + \beta_1 age + \beta_{2,i} month_i + \beta_{3,i} age * month_i$$

An anova test reveals that the second model does not improve the fit signif-

icantly, that is, there is no significant interaction between age and month. The anova test, the regression coefficients of model 1 and the comparison between observed and predicted values are reported in appendix A, Table 2 and 3 and Figure 5.

In this regression setting, $\exp(\beta_0)$ can be interpreted as the a term of the Gompertz equation, β_1 as the b term and the coefficients $\exp(\beta_{2,i})$ as multiplicative terms with proportional effects on the baseline hazard. Since there is no significant interaction between the month and the age term, we conclude that the months shift the mortality curve up and down proportionally and have no effect on the rate of aging (Figure 3).

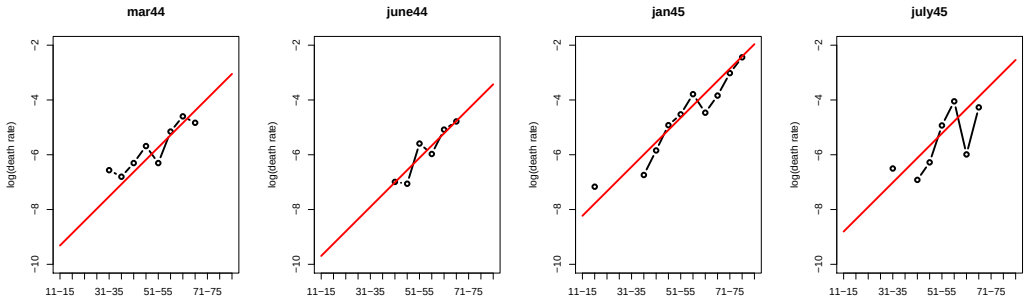

Figure 3: Australian male civilian prisoners of war in WWII. Log mortality rates in different months in the internment camp of Tjimahi (Java) in 1944-45.

4 Selection in the Ukrainian Famine

Reconstructed period life table probability of death from age 0 to age 89 are available from 1927 to 2002 (Meslé and Vallin 2003). I used data from 1927 to 1939 because WWII and different geographical configuration of the Ukrainian territory after 1940 are likely to create discontinuities in the series of deaths.

Plotted on a log scale (Figure 1 b) the curves tend to converge at older ages, especially among women. To investigate whether this could be due to selection effects I decided to use a cohort perspective (cohort data, although more difficult to find, are known to be better than period data in selection analysis). Partial cohort observations have been reconstructed taking the diagonals of the 1 year age groups year by year from the period data matrix. Therefore I have portions of cohort mortality observed for 13 years. After transforming the

probability of death $q(x)$ into hazard $\mu(x)$ with the formula

$$\mu(x) = -\log(1 - q(x))$$

I fitted a Gamma-Gompertz model (Vaupel and Yashin 1985) on the cohort data before the crisis (years 1927 to 1931). Then I extrapolated mortality for the years after the crisis (years 1935-39) and compared the observed after-crisis mortality with the predicted one, based on the data before the crisis. Because the distortion between individual and population mortality due to selection and individual unobserved heterogeneity is supposed to become significant at old ages, I analyzed the cohorts aged between 50 and 77 in 1927. The 77 year old individuals in 1927 are the last ones that can be observed longitudinally for the entire 13 years observation time (given that period data range up to age 89).

My hypothesis is that, because of selection mechanisms reducing the number of weaker individuals during the famine, the after-crisis mortality should be lower then the mortality the cohorts would have experienced if there had been no crisis. Results from simulations of heterogeneity of frailty in human populations show that selection in the presence of a mortality shock leads to such mortality patterns (Vaupel et al. 1988).

The Gamma-Gompertz model, in addition the Gompertz parameters a and b, introduces individual unobserved heterogeneity with an additional parameter for the variance of *frailty* in the population at the initial age, σ^2.

The initial age of the fitted cohorts is different. In fact the mortality surface is slanted because each cohort is observed from a different age (one at age 50, one at age 51 and so on). When performing the estimation this needs to be taken into account both for the initial level of mortality and for the variance of frailty at initial age.

Each cohort is followed for five years (from 1927 to 1931) corresponding to 5 subsequent ages, that I model with ages from 0 to 4. However these are fictitious ages and the model has to take into account that the real initial age at which the cohorts are observed is different and, precisely, older and older. Therefore each cohort has its own a and σ^2 parameters.

I grouped the cohorts in 7 groups of 4 (first group comprises cohorts whose initial age was from 50 to 53 and its mean age is 51.5, second group comprises ages from 54-57 and its mean age is 55.5 and so on), created dummy variables

for each of them and estimated the following Gamma-Gompertz model[2], where $i = 1, ..., 7$:

$$\mu(x) = \frac{(\sum_{i=1}^{7} a_i * cohort_i)e^{bx}}{1 + (\sum_{i=1}^{7} \sigma_i^2 * cohort_i)\frac{\sum_{i=1}^{7} a_i * cohort_i}{b}(e^{bx} - 1)}$$

As expected, the a parameters increase from group 1 to group 7. The cohort-group's real starting age is older and older and, consequently, the respective initial levels of mortality is higher and higher.

Also the σ^2 parameters behave accordingly to what predicted by the theory and decrease from group 1 to group 7. These are, in fact, parameters for the variance of frailty of different cohorts that are observed at older and older initial age. When we start to observe them, they are supposed to appear more and more selected, that is, more homogeneous. Although the estimates for σ^2 are not significant, the model captures the trend predicted by the theory. Tables 4 and 5 and in Appendix B report the estimated parameters for men and women.

Figure 4 shows the observed (black) and predicted (red) mortality curves after the crisis by sex and age. The predicted curves in the absence of crisis are higher than the observed ones. This confirms the hypothesis and may indicate the presence of selection mechanisms during the crisis that lowers the mortality immediately after the shock. The phenomenon is stronger among women than among men, and this is consistent with the more pronounced convergence of the female curves in Figure 1 b. The effect on the men, instead, appears to be weaker and more immediate than women, visible only immediately after the peak caused by the famine.

5 Conclusion

Investigating the effect of mortality shocks on human populations is difficult because it is impossible to set up randomized experiments with humans. However, some events serve as natural experiments. Such events in human history are not rare but only a few of them are documented well enough to be analyzed. Such mortality shocks, also referred to as "natural mortality experiments",

[2]The model estimates one single b. This is justified by the fact that the log-mortality curves of the different cohorts have the same slope. This helps parsimony in the estimation procedure, given that the model estimates already several parameters.

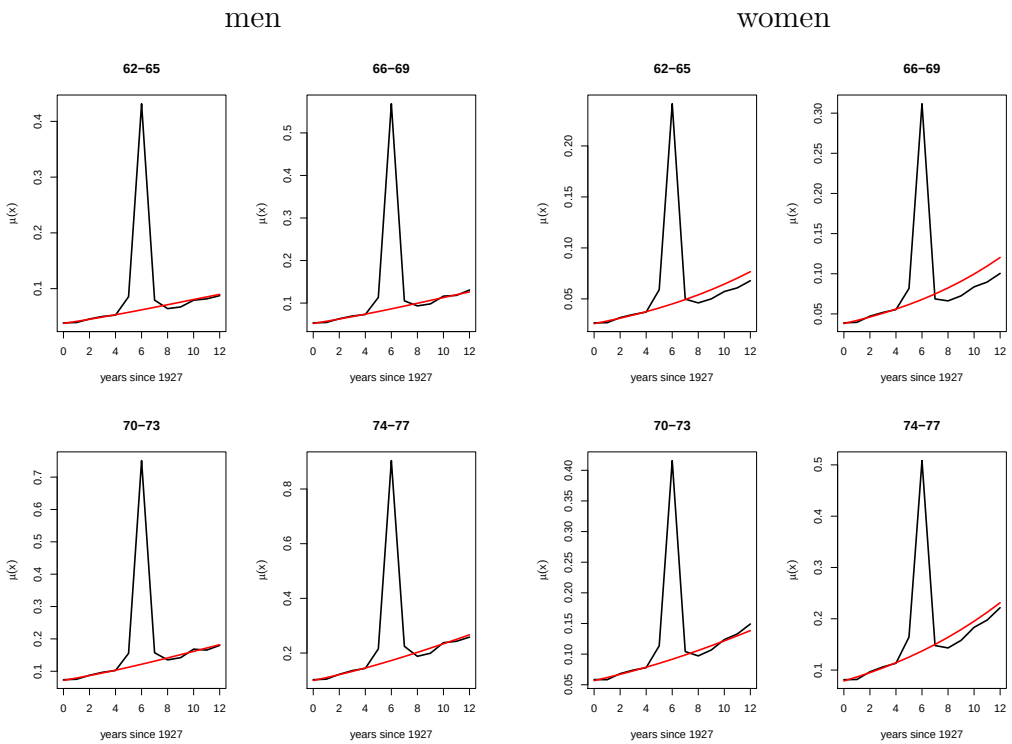

Figure 4: The effect of the Ukrainian famine in 1932-34. Observed force of mortality (black line) for cohorts of Ukrainian men and women aged 62-77 in 1927, followed up for 13 years (1927-39) that include the 3 years of the crisis, showing the mortality peak in 1933. Predicted mortality (red line) shows the mortality regime as if there had been no crisis. The predicted values are the results of a Gamma-Gompertz fit for the years before the crisis and of an extrapolation, from this fit, for the years after the crisis.

serve as a proper tool of analysis because they resemble the experimental condition called "natural experiment". They affect the whole population not selectively, helping to reduce the possibility of bias due to confounding factors. In this paper, using data for approximately 10000 Australian civilian prisoners in a Japanese camp during WWII (Bergman 1948) and for the Ukrainian Famine in 1933 (Meslé and Vallin 2003), I analyzed the effect on the mortality curve of sudden and temporary exposure to extremely harsh conditions.

I investigated whether such change in conditions have an impact on the rate of aging or act mainly on the initial level of mortality, addressing a question that still lights up a lively debate. Moreover, studying the effect of catastrophic events on human mortality may help to decide whether the rate of aging is sensitive to sudden environmental modifications or is biologically determined

and stable.

Parallel mortality curves in the presence of a shock may indicate that the shock acts only on the initial level of mortality, raising or lowering it. In contrast, curves with different slopes might denote an impact on the rate of aging, that can be slowed down or accelerated.

Data on male Australian prisoners in Java during WWII published in 1948 (Bergman 1948) have been often cited (Jones 1959, 1961; Finch 1994; Rozing and Westendorp 2008). The previous analysis, however, focused on the annual death rate in the 2 years of interment (1944 and 1945), using linear fitting on log-transformed data that tend to flatten existing differences. Moreover, they have neglected the available monthly information about the death counts.

In this paper I re-analyzed the data using non linear least squares regression and extend the analysis to the monthly death counts. They allowed me to compute exact exposure times and, therefore, more precise death rates, and to analyze the monthly mortality pattern.

I found that years of imprisonment did not accelerate the rate of aging but raised the mortality curve proportionally at all ages. This finding is strengthened by the analysis of the monthly counts. Mortality during the imprisonment followed the seasonal fluctuations typical of tropical areas but the oscillations left the rate of aging unchanged.

In the case of the Ukrainian famine the mortality curves during the years of the crisis, contrary to the case of the Australian prisoners, showed convergence at old ages. The sound demographic research conducted so far gives a solid basis to consider such convergent patterns as a possible artifact of selection effects of the most robust individuals, which causes a mortality level off at the population level (Vaupel et al. 1979; Vaupel and Yashin 1985).

By applying a Gamma-Gompertz model that controls for unobserved heterogeneity of frailty among individuals, the analysis has detected and showed that mechanisms of selection of the most robust individuals might actually have taken place in the Ukrainian case. Therefore the observed pattern does not necessarily indicate that the rate of aging was affected by the shock, but that the convergence could be an artifact of selection.

How sudden changes in environmental conditions affect the human mortality and, above all, whether they have an impact on the rate of mortality increase by age are crucial questions for understanding aging processes. Contrary to what is possible with laboratory organisms, in the case of humans it is

impossible to investigate these questions with controlled and randomized experiments. The only way that we have so far is to rely on documented mortality shocks.

These findings contribute to the idea that sudden and transitory exposure to extremely severe conditions shifts the mortality curve upward in a parallel fashion, leaving the rate of aging unchanged. The also highlight the possible presence of selection processes acting during the shocks. However, more cases need to be analyzed and more evidence needs to be collected.

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A Australian Prisoners of War

	a			b		
Year	Estimate	2.5%	97.5%	Estimate	2.5%	97.5%
1941	0.00233	0.00200	0.00270	0.08098	0.07793	0.08415
1944	0.00738	0.00433	0.01117	0.07266	0.05198	0.09059
1945	0.01588	0.00828	0.02640	0.08132	0.05715	0.10320

Table 1: Australian male civilian prisoners of war in WWII. Parameter estimates of a Gompertz fit for the death rates in interment regime (1944-45) in the camp of Tjimahi (Java) and for the death rates of the Australian male civilian population in 1941.

	Resid. Df	Resid. Dev	Df	Deviance	P(> | Chi |)
model 1	251	296.86			
model 2	234	272.35	17	24.51	0.1061

Table 2: Australian male civilian prisoners of war in WWII. Anova test between two Poisson regression models for the monthly death rates in the internment camp of Tjimahi (Java), from March 1944 to August 1945. Model 1 regresses death rates on age and months, while model 2 also includes an interaction term between age and months.

	Estimate	Std. Error	z value	Pr(> | z |)
Intercept	-9.4883	0.2347	-40.42	0.0000
Age	0.0895	0.0036	25.08	0.0000
mar44	-0.9900	0.2394	-4.14	0.0000
apr44	reference	-	-	-
may44	-0.7145	0.2188	-3.27	0.0011
june44	-1.3699	0.2795	-4.90	0.0000
july44	-1.2481	0.2668	-4.68	0.0000
aug44	-1.6508	0.3146	-5.25	0.0000
sept44	-2.3422	0.4270	-5.49	0.0000
oct44	-1.0877	0.2515	-4.33	0.0000
nov44	-0.5706	0.2102	-2.71	0.0066
dec44	-0.4020	0.2000	-2.01	0.0445
jan45	0.0954	0.1790	0.53	0.5940
feb45	0.1105	0.1790	0.62	0.5370
mar45	0.2492	0.1736	1.44	0.1511
apr45	0.4292	0.1673	2.57	0.0103
may45	0.6441	0.1608	4.01	0.0001
jun45	-0.2851	0.2049	-1.39	0.1641
july45	-0.4794	0.2189	-2.19	0.0285
aug45	-0.2681	0.2049	-1.31	0.1906

Table 3: Australian male civilian prisoners of war in WWII. Estimated coefficients from Poisson regression of the monthly death rates in the camp of Tjimahi (Java), from March 1944 to August 1945.

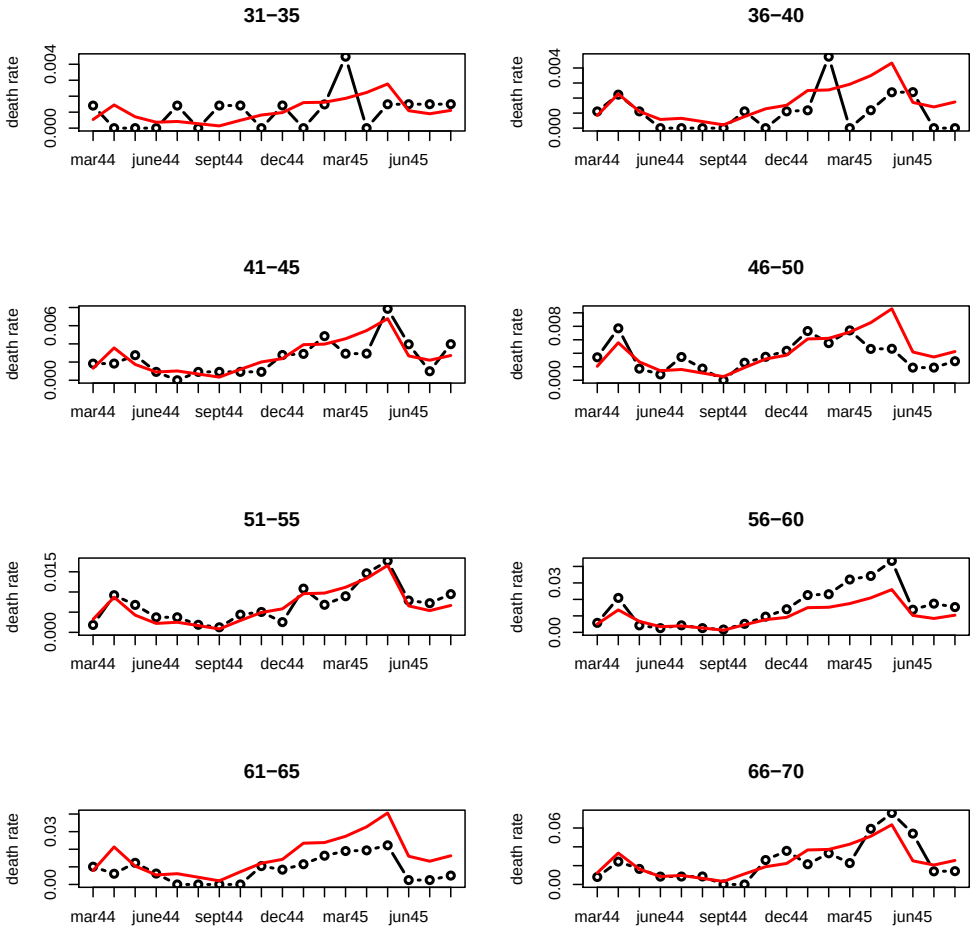

Figure 5: Australian male civilian prisoners of war in WWII. Observed death rates (in black) for five year age groups (between age 31 and 70), during the months of internment (March 1944 - August 1945) and predicted death rates by Poisson regression (in red).

B Ukrainian Famine

	Estimate	Std. Error	t value	Pr(> | t |)
a1	0.0143	0.0024	5.93	0.0000
a2	0.0198	0.0024	8.17	0.0000
a3	0.0273	0.0024	11.25	0.0000
a4	0.0377	0.0024	15.47	0.0000
a5	0.0522	0.0025	21.04	0.0000
a6	0.0720	0.0026	28.03	0.0000
a7	0.0997	0.0028	36.10	0.0000
b	0.1220	0.1156	1.06	0.2931
$\sigma^2 1$	2.4620	7.6665	0.32	0.7486
$\sigma^2 2$	1.7847	5.2449	0.34	0.7342
$\sigma^2 3$	1.2167	3.6605	0.33	0.7402
$\sigma^2 4$	0.7946	2.5772	0.31	0.7584
$\sigma^2 5$	0.5524	1.8396	0.30	0.7645
$\sigma^2 6$	0.3612	1.3167	0.27	0.7843
$\sigma^2 7$	0.2271	0.9396	0.24	0.8094

Table 4: Ukrainian men in 1927-31. Gamma-Gompertz fit for the death rates of cohorts aged 50 to 77 in 1927, divided into 7 groups of 4 year age groups. The a parameters denote the initial level of mortality, σ^2 parameters the heterogeneity level at the initial age for each group and b is the rate of aging that is the same for all the cohorts.

	Estimate	Std. Error	t value	Pr(> | t |)
a1	0.0107	0.0021	5.04	0.0000
a2	0.0138	0.0021	6.61	0.0000
a3	0.0184	0.0020	9.01	0.0000
a4	0.0257	0.0020	12.67	0.0000
a5	0.0380	0.0021	18.26	0.0000
a6	0.0568	0.0022	26.17	0.0000
a7	0.0788	0.0024	33.44	0.0000
b	0.0997	0.1300	0.77	0.4445
$\sigma^2 1$	3.8356	12.5166	0.31	0.7598
$\sigma^2 2$	2.0402	8.9053	0.23	0.8192
$\sigma^2 3$	0.8483	6.1923	0.14	0.8913
$\sigma^2 4$	0.1857	4.2012	0.04	0.9648
$\sigma^2 5$	0.0501	2.7883	0.02	0.9857
$\sigma^2 6$	0.2721	1.9219	0.14	0.8876
$\sigma^2 7$	0.0698	1.3451	0.05	0.9587

Table 5: Ukrainian women in 1927-31. Gamma-Gompertz fit for the death rates of cohorts aged 50 to 77 in 1927, divided into 7 groups of 4 year age groups. The a parameters denote the initial level of mortality, σ^2 parameters the heterogeneity level at the initial age for each group and b is the rate of aging that is the same for all the cohorts.