Mortality shocks and the human rate of aging

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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 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, as well as of genetic modifications, on survival. Some of these 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.

Using some of these documented events I investigate the question whether these shocks have an impact on the rate of aging or mainly shift the mortality curve proportionally. 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

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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 1866-1868 (Pitknen and Suomen vesttieteen 1993; Pitkänen and Mielke 1993; Pitkänen 1992), the Dutch Famine in 1944-1945 (Lumey and Van Poppel 1994), the Ukrainian Famine in 1932-1933 (Meslé and Vallin 2003), the Potato Irish Famine in 1840 (Guinnane and Gráda 2002) and the devastating famine that accompanied the Chinese Great Leap Forward in 1958-1961 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 over-all 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 survivors to holocaust (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 19th century and the Finnish famine in 1866-1868.

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 1944-1945 in the camp of Tjimahi

During WWII thousands of people experienced periods of confinement all around the world. Internment camps were of both types, military and civilian. The great majority of these cases was not properly documented but for some of them, fortunately, the documentation was more accurate.

Right after the end of WWII Bergman (1948) wrote about the Japanese concentration camp of Tjimahi, in Java, where he had been interned and had practised general medicine. He reports 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:

> The conditions of life were virtually the same for all the prisoners. [...] Very few has a camp stretcher. The majority had bare stones. It was very crowded. [...] A few hundred prisoners, working in the kitchen or in the field outside the camp, could sometimes supplement their meager rations. [...] The rest had a ration between 1000 and 1250 calories a day.

He compared the death rates in the camp with the death rates of the Australian 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 (Fig. 1 a).

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. This mid term information is very important because it allows one to count the exposure more precisely. From the paper, in fact, we know that the camp was not completely closed and small movements of prisoners happened throughout the period. Monthly exposures are obtained by subtracting the number of deaths month by month from the number of survivors to the previous month.

2.2 The Great Ukrainian Famine in 1933

In the 20th 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. With perestroika and access to the archives it become clear that the authorities had hidden 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 (Fig. 1 b).

2.3 Freed American Slaves emigrating to Liberia in the 19th 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 19th century (33 years for men and 34 for women (Eblen 1974; Preston et al. 1991)), 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 (Fig. 1 c).

2.4 The Great Finnish Famine in 1866-1868

During the 18th 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 1808-1809, 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. If 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 1861-1865, the years immediately before the crisis, and for 1866, 1867 and 1868, when the crisis reached its peak (Fig. 1 d).

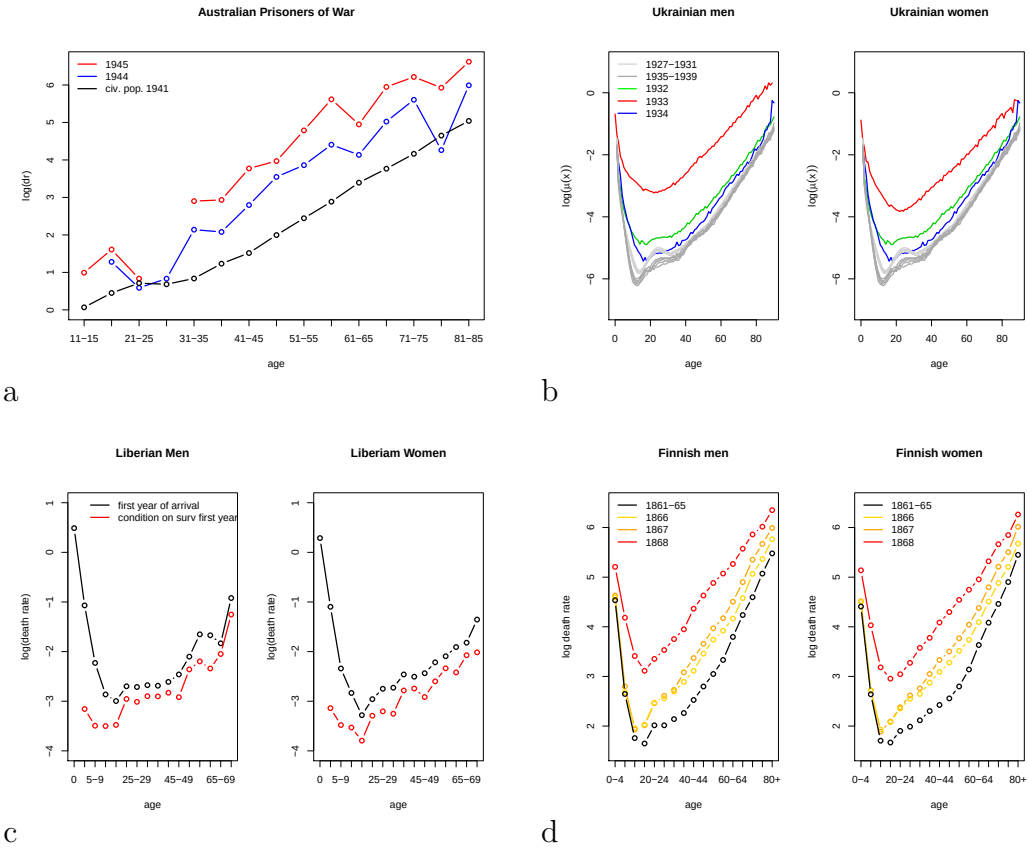

Figure 1: Four Natural Mortality Experiments: y axis reports log mortality. a and d plots: log rates x thousands; b and c: log rates.

The log mortality rates for the 4 cases (Fig. 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.

3 Parallel lines in the Australian Prisoners in WWII

The empirical curves plotted on a log scale (Fig. 1 a) give, at first glance, the impression of fairly parallel log-mortality curves between mortality in the normal regime and mortality in the camp.

As a first step, to investigate whether there are significant differences, I fitted a mortality model to the empirical data. The most widely used mortality model is Gompertz (Gompertz 1825). According to this model, at any age x, the force of mortality $\mu(x)$ is expressed by an initial morality level, a, and by an exponential increase of the mortality rate by age, the parameter b, which is also defined as the rate of aging (Shock 1967; Finch 1994):

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

Using non linear least squares regression I fitted the gompertz model from age 30 onwards to the death rates of the Australian male civilian population in 1941 and to the death rates of the camp in 1944 and 1945.

The estimation indeed, confirms the first impression. The confidence intervals for the estimate of the parameter b in the 3 years overlap indicating that the difference between them is not statistically significant. The difference in the initial level of mortality between the normal regime and the imprisonment regime, a, instead, is significant (More details about the coefficients are given in appendix A, Table 1).

I then looked more deeply into the monthly information. There is a dominant monthly pattern in all age groups (Fig. 4 in appendix B) that reflects the seasonal pattern typical of tropical areas, subjected to the alternating cycle of dry months/humid months of 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 the spread of dysentery raised the death rate in the camp but the Japanese authorities did not allow anybody to die in the camp and sent the sick to nearby hospital camps. Consequently the death rate of the camp fell (Bergman 1948).

For the analysis I used a generalized linear model with Poisson log-link function. The Poisson distribution in fact is the most popular distribution for modelling rates, 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 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:

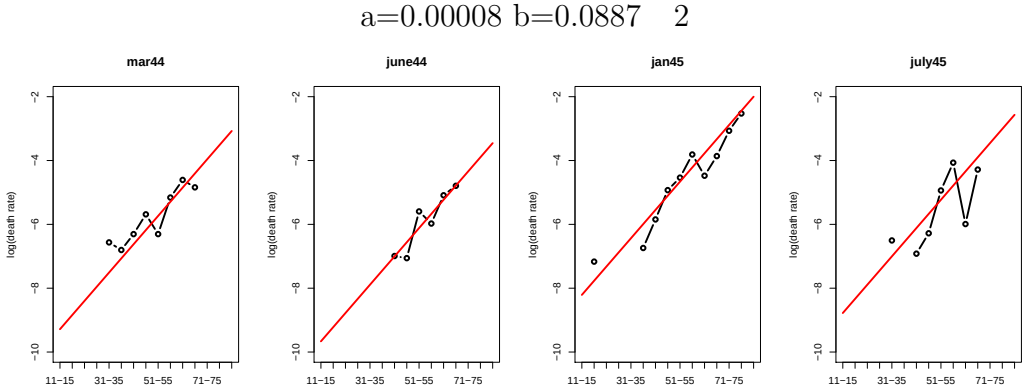

Figure 2: Log mortality rates in different months.

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

An anova test reveals that the second model does not improve the fit significantly, that is, there is no significant interaction between age and month. The regression coefficients are reported in appendix B, table 2.

In virtue of the Poisson regression link function, $\exp(\beta_0)$ 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 (fig. 2).

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 Ukraine after 1940 are likely to create discontinuities in the series of deaths.

Plotted on a log scale (Fig. 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 the hazard $h(x)$ with the formula:

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

I fitted a gamma-gompertz model (Vaupel and Yashin 1983) on the pre-crisis cohort data (years 1927 to 1931) and then I extrapolated mortality for the years after

the crisis (years 1935-1939) in order to compare the observed after-crisis mortality with the predicted one, based on the pre-crisis data. 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 model. The gamma-gompertz model, in addition to 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 (the first group comprises cohorts whose initial age was from 50 to 53 and its mean age is 51.5, the second group consists of 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[1], where $i = 1, ..., 7$:

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

Results. 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 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 ages and when we 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 pre-

[1]The model estimates one b. The log-mortality curves of the different analyzed cohorts are parallel, showing the same rate of aging. Therefore, considering also that the model already contains many parameters, I decided to estimate a single b.

dicted by the theory. Tables 3 and 4 in appendix C report the estimated parameters for men and women.

Figure 3 shows the observed (black) and predicted (red) mortality values after the crisis by sex and age. The predicted values as if there had been no crisis are higher than the observed ones. This confirms my hypothesis and may indicate the presence of selection mechanisms during the crisis that lower 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 1b. The male pattern is quite surprising, showing a weaker and more immediate harvest effect than for women.

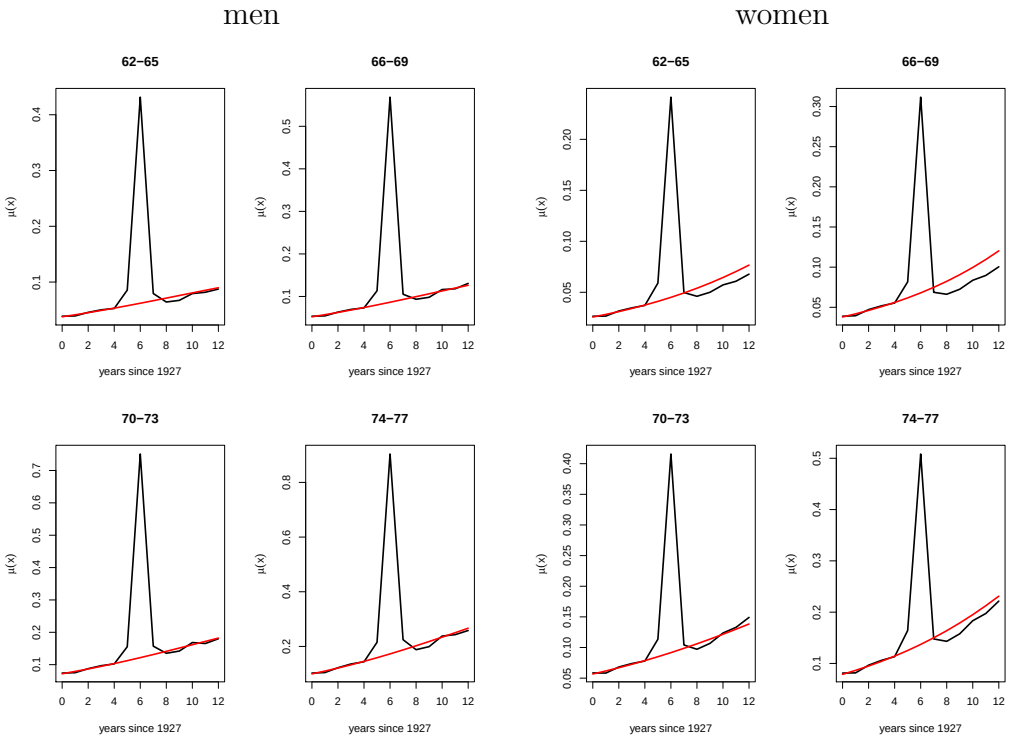

Figure 3: Force of mortality - observed (black line) and predicted (red line) values if there had been no crisis.

5 Conclusion

Investigating the effect of mortality shocks on human populations is difficult because fortunately 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.

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 (Mesle vallin 2004), 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 (Rozing and Westendorp 2008). 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.

In the case of the Australian civilians imprisoned in the Japanese camp of Tjimahni from March 1944 to August 1945, the log-mortality curves of the prisoners appeared to be shifted upward in a parallel fashion compared to the civilian Australian population in time of peace. The analysis with Poisson regression confirmed that the rate of aging was not affected by physical hardship.

In tropical areas the seasonal pattern is dominated by the alternation of dry months (lower mortality period) and humid months (higher mortality period). The analysis shows that the initial level of mortality was clearly affected by the seasonal pattern and the length of stay in the camp: mortality during the dry months in 1945 was, in fact, lower than mortality during the humid months between 1944 and 1945 but higher than mortality during the dry months in 1944, when the imprisonment was just at the beginning (possibly because the longer time spent in the camp weakened the individuals). During this period the mortality curve shifted up and down according to this pattern but its slope was not affected.

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, 1983).

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 mortality experience of human populations 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 human mortality shocks that resemble the conditions of natural experiments.

The two cases analyzed in this paper suggest that sudden and transitory exposure to extremely severe conditions shifts the mortality curve upward in a parallel fashion, leaving the rate of aging unchanged. However, to fully answer the question more cases of human shocks need to be analyzed and more evidence needs to be collected.

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A Gompertz Fit

Year	Estimate	2.5%	97.5%	t value	Pr(>|t|)
			a		
1941	0.00186	0.00163	0.00212	17.10831	0.00000
1944	0.00748	0.00435	0.01136	4.54573	0.00139
1945	0.01651	0.00866	0.02730	3.79284	0.00426
			b		
1941	0.08513	0.08247	0.08788	70.80429	0.00000
1944	0.07146	0.05062	0.08944	7.89819	0.00002
1945	0.07851	0.05468	0.09980	7.47909	0.00004

Table 1: Parameters estimates of the Gompertz fit

B Poisson Regression

	Model 1			
	Estimate	Std.Error	z value	Pr(>|z|)
Intercept	-9.45746	0.23427	-40.37052	0.00000
age	0.08872	0.00356	24.91966	0.00000
mar44	-0.98084	0.23936	-4.09780	0.00004
apr44				
may44	-0.70774	0.21882	-3.23429	0.00122
june44	-1.36146	0.27951	-4.87091	0.00000
july44	-1.23739	0.26680	-4.63793	0.00000
aug44	-1.63994	0.31458	-5.21315	0.00000
sept44	-2.32949	0.42695	-5.45606	0.00000
oct44	-1.07687	0.25148	-4.28206	0.00002
nov44	-0.56686	0.21023	-2.69639	0.00701
dec44	-0.39942	0.20004	-1.99670	0.04586
jan45	0.09381	0.17896	0.52422	0.60013
feb45	0.10814	0.17897	0.60423	0.54569
mar45	0.24674	0.17358	1.42143	0.15519
apr45	0.41850	0.16728	2.50171	0.01236
may45	0.63217	0.16077	3.93203	0.00008
jun45	-0.28371	0.20485	-1.38495	0.16607
july45	-0.47408	0.21889	-2.16581	0.03033
aug45	-0.26366	0.20487	-1.28695	0.19811

Table 2: Model 1. Poisson regression coefficients.

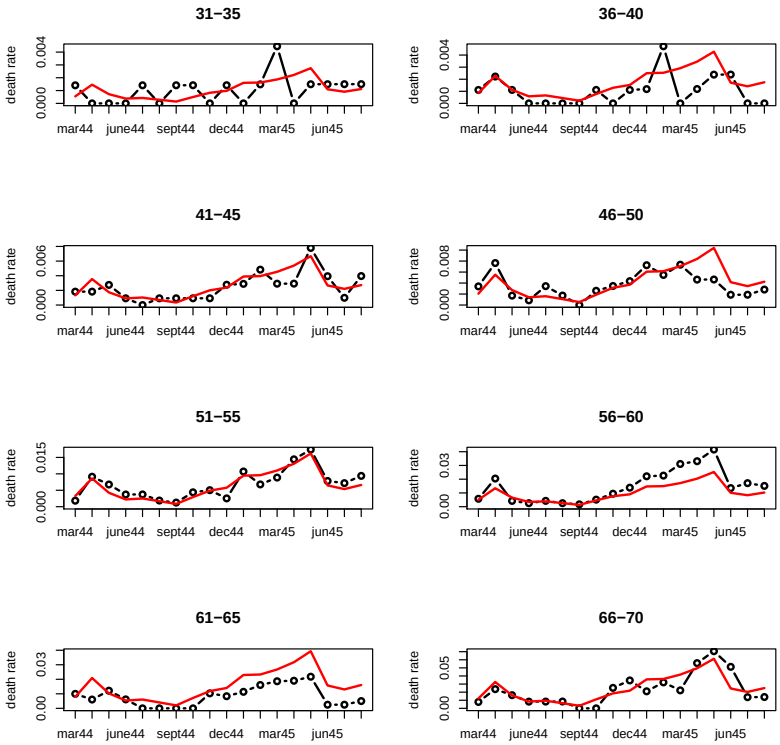

Figure 4: Model 1:$\log(\mu_i) = \beta_0 + \beta_1 age + \beta_{2,i} month_i$ - Observed (black) and Predicted (red) values.

C Gamma-Gompertz Model

	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
G1	2.4620	7.6665	0.32	0.7486
G2	1.7847	5.2449	0.34	0.7342
G3	1.2167	3.6605	0.33	0.7402
G4	0.7946	2.5772	0.31	0.7584
G5	0.5524	1.8396	0.30	0.7645
G6	0.3612	1.3167	0.27	0.7843
G7	0.2271	0.9396	0.24	0.8094

Table 3: Men- estimates of the gamma-gompertz model

	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
G1	3.8356	12.5166	0.31	0.7598
G2	2.0402	8.9053	0.23	0.8192
G3	0.8483	6.1923	0.14	0.8913
G4	0.1857	4.2012	0.04	0.9648
G5	0.0501	2.7883	0.02	0.9857
G6	0.2721	1.9219	0.14	0.8876
G7	0.0698	1.3451	0.05	0.9587

Table 4: Women - estimates of the gamma-gompertz model