Long-term Effects of Famine on Life Expectancy.
A Re-analysis of the Great Finnish Famine of 1866-1868.

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The effects of early life conditions on late-life health and mortality have been repeatedly demonstrated in a long series of studies, starting with the pioneering works of Fridlizius (1993) and Barker (1994). Results from historical populations in the 18th and 19th centuries (Bengtsson & Lindström 2003, Doblhammer 2004, van den Berg, Doblhammer & Christensen 2009), as well as from contemporary populations of the 20th century (for a review see Gluckman, Hanson, Spencer, & Bateson 2005, Leon 2004), have demonstrated the long-term effects of early life conditions on health. Historical studies have stressed the adverse long-term consequences of infectious disease in the first year(s) of life (Bengtsson & Lindström 2003, Fridlizius 1994), particularly epidemics in childhood diseases. Other studies have explored the impact of exogenous stressors, such as economic cycles at the time of birth (van den Berg et al. 2009) or the month of birth (Doblhammer & Vaupel 2001, Doblhammer 2004). Meanwhile, research into contemporary populations has focused on the effects of unbalanced nutrition on the human fetus (Barker 1994) and the development of the thrifty phenotype (Hales & Barker 2001). The underlying hypothesis is that impaired nutrition in utero leads to the development of a thrifty metabolism which is better adapted to survive in a nutritionally stressed post-natal environment. If this metabolic adaptation is in unsuited to the environment after birth, it will result in negative long-term consequences for health (Leon 2004).

Famines are extreme cases of nutritional stress, and have been used by a series of studies to explore the long-term consequences of the fetal or childhood environments. In this context, the most widely studied famines are the Dutch famine of 1944-1945, the Chinese famine of 1959-1961 and the famine of the Siege of Leningrad in 1944. Various indicators of long-term health have been used and various associations have been found. A review of the Dutch Hunger Winter Families Study (Lumey, Stein, Kahn, van der Pal-de Bruin, Blauw, Zybert & Susser 2007) reported a clear association between anthropometric measures and prenatal exposure to famine, and a modest association with current blood pressure. Famine has been

shown to have long-term effects on antisocial personality disorder (Neugebauer, Hoek & Susser 1999), early onset of coronary artery disease (Painter, de Rooij, Bossuyt, Simmers, Osmond, Barker, Bleker & Roseboom 2006) and coronary heart disease (Roseboom, van der Meulen, Osmond, Barker, Ravelli, Schroeder-Tanka, van Montfrans, Michels & Bleker 2000). The results of some studies have also indicated that famine can have lasting epigenetic effects, including less DNA methylation of the imprinted IGF2 gene (Heijmans, Tobia, Stein, Putter, Blauw, Susser, Slagboom & Lumey 2008) and fingertip ridge counts (Kahn, Graff, Stein & Lumey 2009). Exposure to famine during any stage of gestation has been shown to be associated with glucose intolerance, while exposure during the early stages of gestation has been found to be related to coronary heart disease, a more atherogenic lipid profile, disturbed blood coagulation, increased stress responsiveness and higher obesity rates. Women exposed to famine in early gestation have also been shown to have an increased risk of breast cancer. People exposed to famine in mid-gestation appear to have higher rates of microalbuminuria and obstructive airways disease (all Roseboom, Rooij & Painter 2006). Chinese famine studies have reported the effects of famine during gestational age and early childhood on a range of socioeconomic outcomes (Almond, Edlund, Li, & Zhang 2007, Chen & Zhou 2007, Meng & Quian 2006), such as literacy, labor market status, wealth and marriage market outcomes; as well as on height (Chen & Zhou 2007, Gørgens, Meng, & Vaithianathan 2005, Meng & Quian 2006), upper arm circumference (Meng & Quian 2006), the prevalence of linear enamel hypoplasia (Zhou & Corruccini 1998) and schizophrenia (St Clair, Xu, Wang, Yu, Fang, Zhang, Zheng, Gu, Feng, Sham & He 2005). Unlike in the Netherlands (Stein, Zybert & Lumey 2004), the Chinese famine was found to have reduced the sex ratio (males to females) in two generations (Almond et al. 2007). Reports from men who experienced the Siege of Leningrad around the age of puberty point towards a greater incidence of high blood pressure, and higher mortality from ischemic heart disease and stroke, including hemorrhagic stroke (Sparén, Vågerö, Shestov, Plavinskaja, Parfenova, Hoptiar, Paturot & Galanti 2005).

Conflicting evidence exists concerning the effects of famine on mortality and life expectancy. While an analysis of the Dutch potato famine found higher late-life mortality for cohorts born during the famine (van den Berg, Lindeboom & Portrait 2007), the pioneering cohort study by Kannisto, Christensen & Vaupel (1997) for the Great Finnish Famine of 1866-1868, and the study by Painter, Roseboom, Bossuyt, Osmond, Barker & Bleker (2005) for the Dutch Hunger Winter, did not find differences in mortality at older age for cohorts born during famine.

This study re–analyzes the Finnish cohort data used by Kannisto et al. (1997), which are available in the Human Mortality Database. The Kannisto study used four indicators to explore the survival of the cohorts born before the famine (1861-1865), during the famine (1866-1868) and after the famine (1869-1873): survival up to age 17, survival from ages 17 to 40, survival from ages 40 to 60, and survival from ages 60 and above. For the various birth cohorts, they found large differences in survival up to age 17, and none thereafter. Cohorts born during the famine were found to have lower survival rates up to age 17, but no long-term effect on survival at older ages was demonstrated.

One possible explanation for the absence of any long-term effect on survival in the Kannisto study is that selection during the famine altered the frailty distribution of the population. There are three possible selection effects that need to be considered. First, famines generally affect fertility patterns in the following ways: the number of births during a famine declines, and the number of births then rises sharply shortly after the famine. The social and biological characteristics of women who conceive and give birth during famines may be different from those of women who have children in normal years. Thus, their babies may also differ in terms of the factors that influence their long-term health and survival. Second, mortality during famines is not random, resulting in the death of frailer individuals and leaving only the stronger to survive. Therefore, surviving individuals are more homogenous in terms of frailty. Third, since the surviving population is more homogenous, the offspring of the survivors should also be more homogenous. Thus, cohorts born after the famine should display less variability in theirlevels of frailty.

If we assume that the unobservable frailty of a birth cohort can be modelled by a stochastic frailty distribution, then the following predictions result from the ideas presented above:

Two forces affect the frailty distribution of the survivors of famines. On the one hand, any negative long-term effect of the famine (debilitation) should make individuals more heterogeneous in their health depending on their biological and social characteristics and their vulnerability to the famine. Therefore, the variance of the frailty distribution should increase. On the other hand, the variance should be smaller because mortality may be expected to select out frailer individuals, thus resulting in a more homogenous group of survivors. In the extreme, these two forces cancel each other out, resulting in unchanged mortality of cohorts

born during the famine. If, however, the debilitation effect is larger than the selection effect, then the variance of the frailty distribution should increase. If the opposite is the case and selection is larger than debilitation, then the variance should decrease. This should apply to all cohorts born before and during the famine to a varying extent, depending on the age-specific vulnerability to famine.

If the debilitating long-term effects of the famine are larger than the mortality selection during the famine, then the frailty distribution of the cohorts born after the famine will have a smaller variance. They are the only cohorts who are not directly affected by the famine, and they are also selected by the fertility of the survivors of the famine.

If negative long-term consequences of famines exist and the underlying cohort-specific frailty distribution is accounted for, then the cohorts born during the famine should have higher death rates, and may therefore be expected to have lower life expectancy at old age than those born after the famine. The answer to the question of whether they should have higher mortality than cohorts born before the famine is not, however, obvious. It depends on the age that is most vulnerable to famine.

In contrast to the Kannisto et al (1997) article, we explore a longer time period and use cohort data for the birth cohorts 1850 up to 1890. The reasons are twofold. First, long-term effects of the famine may exist for ages other than the first year of life, and the old-age mortality of all cohorts born before the famine may therefore be altered. Second, because selective fertility may extend well beyond the immediate years of the famine and, the frailty distributions of the cohorts born after the famine may be altered.

In addition, because it has been repeatedly shown that mortality is primarily influenced by period factors, we control for important period effects, such as the end of World War I/ the Spanish Flu Pandemic in 1918 and World War II. Finally, we compare the mortality of the Finnish and Swedish cohorts born in the 19th century. Unlike Finland, Sweden, which is the country with the best data quality at that period of time, did not suffer from any severe hunger crises in the middle of the 19th century.

Materials and Methods

This study analyzes the death rates of birth cohorts, accounting for unobserved heterogeneity in their distribution of deaths. We distinguish eight cohorts: 1850-1854, 1855-1859, 1860-1865, 1866-1868, 1869-1874, 1875-1879, 1880-1884 and 1885-1889. The birth cohorts 1860-1865 are the immediate pre-famine cohorts, those born between 1866 and 1868 are the famine cohorts, while birth cohorts 1869-1874 are the immediate post-famine cohorts. All analyses are performed separately for males and females.

For each cohort, the force of mortality $\mu(x)$ over age x follows a baseline mortality μ_0 modified by a frailty distribution Z.

$$\mu(x) = Z\mu_0 \tag{I}$$

$$\mu_0 = \alpha e^{\gamma x} e^{\sum_{i=1,..n} \beta_i Y_i} \tag{II}$$

with α and γ denoting the parameters of a Gompertz function, and Y_i denoting the n indicator variables for the single-year birth cohorts, as well the indicator variables for the period effects of WW I/Spanish Flu and WWII together, with their parameter estimates b_i. The frailty distribution Z follows a Gamma distribution with mean one and variance σ^2.

$$Z \sim \Gamma(1, \sigma^2) \tag{III}$$

In equation (II), α is independent of age and determines the general level of mortality of a cohort. Over cohorts, α is changing, which may counterbalance the cohort trend in the variance of the frailty distribution. An alternative specification is to consider the general level of mortality as part of the frailty distribution, and fix the level of mortality for the various cohorts arbitrarily at the value of one. Thus, equations I to III can be re-written into

$$\mu(x) = Z\mu_{01} \tag{IV}$$

$$\mu_{01} = \alpha_0 e^{\gamma x} e^{\sum_{i=1,..n} \beta_i Y_i} \tag{V}$$

where α_0 is fixed to one, and

$$Z \sim \Gamma\left(\alpha, \sigma^2\alpha^2\right) \qquad \text{(VI)}$$

The variance $\sigma^2\alpha^2$, which we name standardized variance, is used to test the three selection hypotheses formulated above, and the hazard rates of the Gompertz function μ_0 are used to estimate remaining life expectancy at age 60 for the standard individual of a cohort. For the sake of brevity, we refer to the latter as the standardized life expectancy.

The human mortality database http://www.mortality.org contains deaths by Lexis triangles, cross-classified by year of birth, year of death and age (Wilmoth et al. 2007). Because we are interested in the long-term effects of famine, we start our analysis at age 60. To avoid the influence of extreme longevity on the variance of the frailty distribution, we right-censor all deaths at age 95. Since migration at age 60+ is negligible and all cohorts are extinct, we use the death counts in the Lexis triangles to estimate the age-specific force of mortality for single-year age intervals.

For Finland, deaths are available from age zero onwards, starting with the year 1878. Lexis triangles were constructed by the team of the Human Mortality Database using the aggregate death counts published in various statistical publications (Statistics Finland, 1879-2003a) by year of birth, year of death and age. This implies that the cohorts born before 1878 are included from the age they had reached in 1878 onwards. Thus, members of the cohort born in 1850 are in the database from age 28 onwards, while data are available from age zero onwards for members of the youngest cohort in this analysis, who were born in 1889.

For Sweden, Lexis triangles of deaths are available from age zero onwards starting in 1751. However, the quality of the data improved tremendously after 1860, when the compilation of population and death counts based on all parish registers sent to Statistics Sweden began. Age reporting has been extremely accurate since 1861, but age exaggeration was a problem before that date. (Wilmoth and Lundström 1996).

Two major period effects modify the hazard rates in our analysis. For Finland, we include one variable to capture the effect of the declaration of independence from Russia in 1917, which

resulted in a short civil war from January to May 1918, the same year that Finland was hit by the Spanish Flu Pandemic. The variable takes the value one for the years 1917 and 1918, and zero otherwise. The second variable captures the two wars fought by Finland against the Soviet Union during the Second World War: the Winter War from November 1939 to March 1940, and the **Continuation War from** June 1941 to September 1944. For Sweden, two additional period effects have been included in two separate variables: the Spanish Flu pandemic of 1918 and World War II from 1939 to 1945.

Cohorts lived through these period effects at various ages. The two oldest cohorts (1850-1854, 1855-1859) experienced the events of 1917/1918 at ages 60-68, while in this analysis, the younger cohorts were not exposed because the events happened before their 60^{th} birthday, which is the starting age of our analysis. The oldest cohort was aged 85 to 94 (Finland)/95(Sweden) during WW II, and the second oldest was aged 80 to 89/90. Birth cohorts 1885(Finland)/1886(Sweden) and younger do not experience the period effect of WW II in this analysis, because at that time they were younger than age 60.

Results

Table 1: Parameter estimates of the hazard model for Finland

	Period Effects		Gompertz Function	Frailty Distribution		
Cohorts	1917-1918	1939-1944	$ln(\alpha)$	γ	σ^2	$\sigma^2\alpha^2 E^{10}$
Females						
1850-1854	0.223	-0.180	-10.097	0.102	0.024	0.409
1855-1859	0.377	-0.109	-10.951	0.115	0.138	0.426
1860-1865		0.005	-10.480	0.108	0.118	0.928
1866-1868		0.075	-9.814	0.097	0.031	0.941
1869-1874		0.070	-10.019	0.100	0.030	0.596
1875-1879		0.228	-11.321	0.118	0.147	0.215
1880-1884		0.317	-11.582	0.121	0.209	0.182
1885-1889			-11.494	0.120	0.259	0.269
Cohorts	1917-1918	1939-1944	$ln(\alpha)$	γ	σ^2	$\sigma^2\alpha^2 E^{9}$
Males						
1850-1854	0.290		-8.480	0.083	0.038	1.618
1855-1859	0.399		-8.782	0.088	0.062	1.462
1860-1865		-0.025	-9.235	0.096	0.123	1.174
1866-1868		0.015	-8.917	0.092	0.114	2.058
1869-1874		0.075	-8.182	0.079	0.004	0.323
1875-1879		0.243	-8.884	0.088	0.050	0.963
1880-1884		0.247	-8.871	0.088	0.057	1.114
1885-1889			-8.835	0.088	0.087	1.847

Table 1 shows the parameter estimates of the hazard models for Finland. Both the variance of the frailty distribution σ^2 and the age-independent level of mortality α fluctuate considerably over the cohorts, and do not by themselves constitute a conclusive trend. Considering the age-independent parameter α as part of the frailty distribution ($\sigma^2\alpha^2$) clearly shows an increased standardized variance for the birth cohorts 1866-1868 among males, and the cohorts 1860-1865 and 1866-1868 among females.

The parameter estimates for the two period effects in the hazard models are highly significant for those cohorts among whom a large percentage of their members were still alive. The effect of the inclusion of the period effects on the standardized variance is large (Figure 1). Among males, a model without period effects ascribes the highest standardized variance to the birth cohort 1869 to 1874; the correction for period effects results in the highest standardized variance for the birth cohort 1866 to 1868. The rather erratic cohort pattern in the standardized variance on the basis of the models without period effects turns into a declining trend among the cohorts born before the famine, a strong increase for those born during the

famine, followed by a sharp decline for the cohorts born immediately after the famine once period effects are accounted for. Among younger cohorts, the standardized variance increases steadily. In the same figure, the comparison with Sweden shows that the trends in the standardized variance are unrelated, although there is a small increase for Swedish cohorts born 1860-1865 and 1866-1868.

For females, the results are different (Figure 2). The models without period effects find a general decrease in the standardized variance over cohorts, with the exception of a large increase for the birth cohort 1866 to 1868. Meanwhile, Swedish data show an increase over cohorts. Taking period factors into account changes these trends: for Finland, the highest standardized variances exist for the birth cohorts 1860 to1865 and 1866 to 1868, followed by a strong decrease in the variances for cohorts born after the famine. For Sweden, the trend also changes and more closely follows the Finnish pattern.

Figure 1: The standardized variance of male cohorts born 1850 to 1889 for Finland and Sweden, with and without period effects

Figure 2: The standardized variance of female cohorts born 1850 to 1889 in Finland and Sweden, with and without adjustment for period factors

Figure 3 compares the standardized life expectancy between Finland and Sweden. For males, it is lowest for the birth cohorts 1866 to 1868, and it is second-lowest for the cohorts 1860 to 1865. The difference between the famine cohort and the cohort born immediately after the famine (1869-1874) is 1.16 years, when corrected for period effects (1.06 years for cohorts born 1860-1865). Sweden does not show any large disturbances in standardized life expectancy for these cohorts.

Among Finnish females, standardized life expectancy is lowest for the cohorts born before the famine (1854-1859, 1860-1865). The difference from the cohorts born immediately after the famine is generally one year, while the difference between the famine cohorts and those born after the famine is 0.24 years. In addition, the Swedish pattern in the standardized life expectancy closely resembles that of Finnish females, once period effects are accounted for.

Figure 3: Standardized life expectancy (at age 60) for male cohorts born 1850 to 1889 in Finland and Sweden, with and without adjustment for period factors

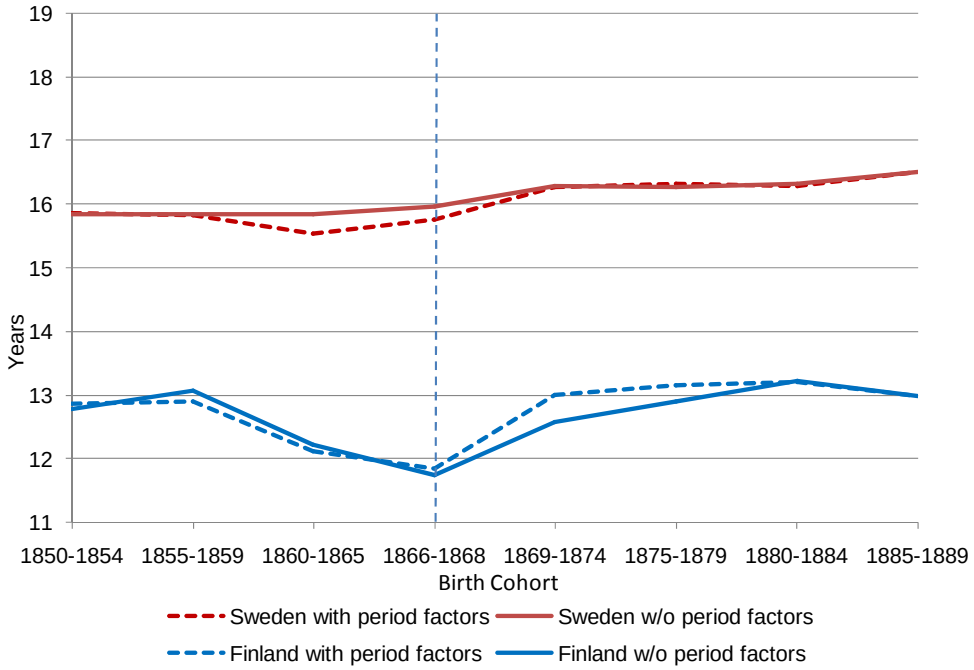
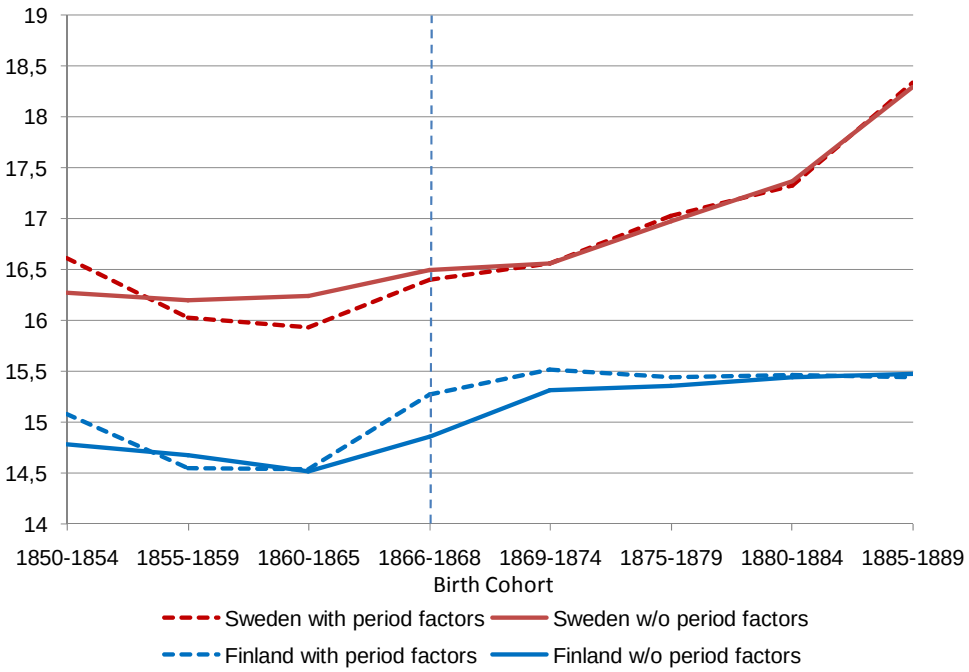


Figure 4: Standardized life expectancy (at age 60) for female cohorts born 1850 to 1889 in Finland and Sweden, with and without adjustment for period factors


19
18,5
18
17,5
17
16,5
16
15,5
15
14,5
14
1850-1854
1855-1859
1860-1865
1866-1868
1869-1874
1875-1879
1880-1884
1885-1889
Birth Cohort
Sweden with period factors
Sweden w/o period factors
Finland with period factors
Finland w/o period factors

Discussion

Selection forces of fertility and mortality alter the frailty distributions of cohorts. We show that cohorts born in Finland during the Great Finnish Famine (1866-1868) are highly heterogonous in their distribution of deaths after age 60. By contrast, those born in the years immediately after the famine (1869-1874) seem to be more homogenous. The cohorts born immediately before the famine (1860-1865) also display increased heterogeneity: among males, heterogeneity is lower than among the famine cohorts, but it is still higher than among the post-famine cohorts. Among females, these cohorts are the most heterogeneous of all.

Taking changes in the frailty distribution into account results in lower standardized life expectancy for cohorts born during the Great Finnish Famine. This is particularly true for males, among whom approximately one year is lost relative to the cohorts born after the famine. The cohorts born immediately before the famine display the second-lowest standardized life expectancy. The absence of any effect among male Swedish cohorts born during the period of the Finnish famine further supports the assumption that the famine in Finland had long-term negative effects.

For males, we conclude that the Finnish famine resulted in an increased heterogeneity in age at death and a long-term decrease in life expectancy. For females, the results are less clear. As mentioned above, heterogeneity in the age at death is increased for all cohorts born between 1860 and the end of the famine in 1868. However, this does not necessarily result in a lower standardized life expectancy. The cohorts born between 1860 and 1865 lose about one year of life relative to the cohorts born immediately after the famine. Those born during the famine are only marginally affected, with a loss of 0.24 years. Furthermore, trends in Swedish standardized life expectancy closely resemble those of the Finnish cohorts, once period factors are taken into account.

Another noteworthy finding of this study is that the standardized life expectancy of those born during the famine and of those born before the famine was reduced. More precisely, both sexes born in Finland between 1860 and 1865 show increased heterogeneity and lower standardized life expectancy relative to those born immediately after the famine. There may be two reasons for this phenomenon: (1) the definition of the time period of the Great Finnish

Famine may be too narrow, and (2) the repeated outbreak of childhood diseases before the famine may have had negative long-term effects on the health of the respective cohorts.

While the Great Finnish Famine is, according to a generally accepted definition (Pitkänen 1993), understood to have occurred during the years 1866 to 1868, this period actually represents a low point in a series of economic difficulties that started at the beginning of the decade. The first of several harvest failures took place in 1862, with this severe reduction in crop yields mainly hitting the northern provinces. The next widespread crop failure was in 1865, but it was not as extensive as the one three years earlier. The year 1866 was the first of the decade with a somewhat better harvest, while the harvest in 1867 was the worst of the entire decade almost everywhere in Finland. In 1868, the harvest was exceptionally abundant in many of those regions which had repeatedly suffered from poor harvests since the beginning of the 1860s.

When defining famine, taking only harvest patterns into account is certainly insufficient. Famine is characterized by a considerable shortfall in food intake by a sizable number of people, and often is rooted in a preceding crop failure. However, many authors emphasize that the social disruption and economic chaos caused by a famine (Pitkänen 1993, O Grada 2007a,b) is as important—and is sometimes even more important—than the food shortages themselves (O Grada 2007a,b). Large-scale migration; the separation and uprooting of families; an increase in crime; the loss of land, livestock and other assets of production; mental disorientation; the consumption of alternative subsistence foods and loss of body weight are all factors that contributed to the large numbers of excess deaths. Thus, the core period of a famine is often defined by death counts.

In the 1860s, the first year with a peak in the total number of deaths was 1863. In this year, excess deaths were mainly the result of childhood diseases, such as measles and whooping cough. Death counts from these two causes of death were twice as high in 1863 as they were during the actual famine years (Pitkänen 1993). Mortality started to increase again, in spring 1866, and reached a second peak in spring 1867. After a rapid increase from December 1867 onwards, mortality reached catastrophic levels between March and August 1868. Despite a decrease in mortality from September 1868 onwards, mortality remained at high levels until summer 1869. As is typical for historical populations (O Grada 2007 a,b), starvation was not a main cause of death during the famine. Excess mortality resulted primarily from typhus and

dysentery, with substantial numbers of deaths from childhood diseases, such as smallpox, whooping cough and measles. In terms of childhood disease, one noteworthy feature of the Great Finnish Famine is that the rates of all three were elevated during the famine years (Pitkänen 1993).

Based on harvest patterns and death counts, it is possible to argue that the cohorts born from the beginning of the 1860s until the end of 1868 were repeatedly exposed to hazardous circumstances early in life: crop failures since 1862, the epidemic of childhood disease in 1863, as well as the Great Famine between 1866 and 1868. Many of these years were characterized by high levels of infant mortality and childhood disease. In terms of infant and child mortality, the year 1863 stands out; a fact that was already noted by Pitkänen (1993) when he compared mortality during the famine with pre-famine years. He pointed out that, when the pre-famine years, including the year 1863, are used as a basis for studying the infant and child mortality of the famine years, the impact of the famine may be underestimated.

The Kannisto et al study compared the combined birth cohorts 1861 to 1865 to those of the famine years 1866 to 1868, and thus must result in an underestimation of the effect of the famine. However, even if other cohorts are taken as a reference, the impact of the famine is only visible if unobserved heterogeneity is accounted for.

This is not the only study that finds that the long-term effects of early life circumstances differ by gender, and that the results for women are less conclusive than for men. For example, the effect of the business cycle at birth on late life mortality is stronger for men than for women in the Danish twin data (van den Berg, Doblhammer, Christensen 2009; van den Berg, Doblhammer, Christensen 2008). Pitkäinen (1993) found large sex differences in the Finnish famine mortality figures, with males being far more likely than females to die. Although this sex differential is particularly large for ages 10 to 40 in the high-impact areas of the famine, it also exists for younger ages.

It is difficult to compare the results from the Great Finnish Famine to those of the Dutch and Leningrad famines. The latter are both modern famines of the 20th century, in which excess deaths resulted mainly from starvation, while infectious disease hardly played a role (O Grada 2007a,b). Thus, the long-term effects on health of these two famines may be due to different mechanisms than those that were important for the Great Finnish Famine. Little knowledge

exists about causes of death in the Great Chinese Famine, and about what role infectious disease played there (O Grada 2007a,b)

This study has two major limitations. First, because cohort data based on Lexis triangles only exist on a yearly basis, the critical periods during pregnancy or the first years of life cannot be exactly defined. In order to identify the impact of the period effect on the variance of the frailty distribution, we had to combine several birth cohorts that lived through these events at various ages. Second, since the data apply to the whole country, no regional variation in the timing and the severity of the famine can be explored. On the other hand, this limitation also lends strength to this study. Selection effects due to migration out of the study area, a problem usually encountered in historical studies based on parish data, do not play a role.

In conclusion, the long-term negative effects of famine on adult health and mortality may be hidden by mortality and fertility selection, and may only be seen after accounting for differences in the frailty distributions of the cohorts. In the context of the historical famine in Finland, the negative long-term consequences on mortality may be attributable to childhood disease and to typhus/dysentery, rather than to starvation.

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