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Gerard J. van den Berg
Maarten Lindeboom
France Portrait

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Gerard J. van den Berg
Free University Amsterdam, IFAU-Uppsala,
CEPR, IFS, Netspar and IZA

Maarten Lindeboom
Free University Amsterdam, HEB-Bergen,
Tinbergen Institute, Netspar and IZA

France Portrait
Free University Amsterdam
and Tinbergen Institute

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ABSTRACT

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Background: Nutrition in utero and infancy may causally affect health and mortality at old ages. Until now, very few studies have demonstrated long-run effects on survival of early life nutrition, mainly because of data limitations and confounding issues. Methods: This paper investigates whether exposure to nutritional shocks in early life negatively affects longevity at older ages, using unique individual data and exploiting the exogenous variation implied by natural experiments. In particular, early nutritional conditions are instrumented by exposure to the potato famine of unprecedented severity that the Dutch faced in 1846-47. The individual data are from the Historical Sample of the Netherlands and are augmented by food price data and macro-economic data. The sample used in the study covers lifetimes of 398 individuals exposed and 1,342 individuals not exposed to severe famine during gestation and/or till age three. We compare non-parametrically the total and residual lifetimes of treated and controls per gender. We also estimate survival models in which we control for other individual characteristics and additional (early life) determinants of mortality. Results: Men exposed to severe famine during pregnancy (at least four months) and directly after birth have a significant lower residual life expectancy at age 50 than others, but not at earlier ages. We could not demonstrate any long-run effects for men exposed at ages 0-2 and for women. Conclusion: To our knowledge, this is the first evidence suggesting long-run effects of early nutritional stresses on mortality at old ages for men.

JEL Classification: N33, J10, I10

Keywords: nutrition in early life, famine, longevity, natural experiments, survival analysis, mortality, food intake, developmental origins, fetal origins

Corresponding author:

France Portrait
Institute of Health Sciences
Free University of Amsterdam
De Boelelaan 1085
1081 HV Amsterdam
The Netherlands
E-mail: france.portrait@falw.vu.nl

BACKGROUND

Nutrition in utero and infancy may causally affect health and mortality at old ages.[1-3] The main idea is that vital organs and the immune system might inadequately develop when the body faces in early life inappropriate nutritional stimuli. These damages may increase the predisposition to (chronic) diseases at old ages directly (see the "Barker's fetal origins" hypothesis[1]) or through poorer health and socioeconomic achievements along life course.[4,5] However, the hypothesis linking early nutrition to health at old ages is currently heavily questioned and the empirical literature is inconclusive.[6-8]

Most evidence is based on studies using birth weight, or other (anthropomorphic) measures at birth or in infancy, as a proxy for the early nutritional environment. Comprehensive reviews of the available demographic and epidemiological evidence have been published.[3,9] The majority of studies, but not all, demonstrate high correlations between early life indicators and increased morbidity at older ages. However, birth and infant outcomes may depend on unobserved (genetic and socioeconomic) factors that may also explain health at older ages. Therefore, the effects shown by these studies may be confounded and cannot be interpreted as causal. Other studies try to remove as much unobserved heterogeneity as possible by using longitudinal methods or very detailed (twins) data.[e.g.10-11] These studies often confirm the correlation, but for similar reasons their results may still be confounded.

Randomized experiments are considered as the superior methodology for unraveling causal effects. True experiments exist in the development economics literature, but the studies are for obvious reasons confined to harmless variations in diet, and only follow the individuals for a short period of time, or are still ongoing.[13-15] Experimental animal research also supports the long reach of early nutrition, but it is obviously difficult to directly translate the research results to humans.[16,17] Finally, a recent strand of literature uses natural

experiments to identify causal links. These studies use natural events or macro (economic) events as source of external variation. These events affect the supposed causal factor but are most probably independent of suspected confounders. In the context of our research, people compared health in later life of individuals exposed and not exposed to a famine in early life. These studies are until now inconclusive. For instance, no significant effect on mortality is found for individuals born in Finland during the famine of 1866-68.[18] Studies on the siege of Leningrad in 1941-44 show some conflicting results. Individuals exposed in utero or in infancy do not experience higher risks for coronary heart diseases in adult life[19] while men exposed at age 6-15 are more vulnerable to cardiovascular diseases and cardiovascular mortality at older ages.[20] Studies based on the Dutch "hunger winter" under German occupation at the end of World War II[21,22] and on China's great famine[23,24] show significant long-run effects on adult morbidity, but not on adult mortality.[25] Furthermore, individuals born during or just after the rainy hungry season in Gambia experience higher mortality rates at young ages than others.[26] Note finally that data covering full lifetimes of individuals are required if we want to observe the full patterns on survival. These data are scarce.

This paper investigates whether exposure to nutritional shocks in early life negatively affects longevity at older ages, using unique individual data and exploiting the exogenous variation implied by natural experiments. In particular, early life nutritional conditions are instrumented using exposure to the potato famine of unprecedented severity that the Netherlands faced in 1846-47. We compare non-parametrically the total and residual lifetimes of men and women exposed and not exposed to famine during gestation and in early infancy. We also estimate survival models in which we control for other individual characteristics and additional (early life) (macro) determinants of mortality.

DATA

Population and study sample

The individual data are from the Historical Sample of the Netherlands (HSN).[27] The HSN data are well suited to explore the causality at hand for two major reasons. First, the data cover the full lifetimes of a random sample of 13,718 Dutch individuals born between 1812 and 1922. The end of the observation window is December 31, 1999. The HSN members are born in the provinces of Utrecht, Friesland and Zeeland, which were jointly rather representative of the Netherlands in terms of economic activity and mortality patterns. Second, the HSN data are derived from the registers of birth, marriage, and death certificates. Besides the timing and place of vital life events, the data provide parental and individual characteristics recorded in the above registers. This allows the control for much unobserved heterogeneity and the characterization of groups most at risks.

We discarded from the analyses individuals with missing values for the explanatory variables. The observations with no information on death are either excluded or right-censored if information on marriage or child birth is available. 25.9% of the observations are censored at on average age 27.2 (standard deviation= 9.0). We observe dates of death of migrants out of the provinces.

We considered three birth cohorts: September 1st 1846–June 1st 1848 (398 individuals born, or in gestation during the most tragic famine period); September 1st 1848–September 1st 1855 (702 individuals born after the famine); September 1st 1837–September 1st 1844 (640 individuals born before the famine period). The latter two cohorts act as a control to detect the effect of the famine period. We also considered controls born before the famine as differences in survival between treated and controls born after the famine may be explained by upward trends in (residual) life expectancy.

Famine exposure and other macro (economic) instruments for early life conditions

Most importantly, the nutritional conditions in early life are characterized by exposure to the Dutch potato famine of 1846-47. At that time, all potato harvests and grain crops in Europe failed due to the Deadly Fungus potato disease and bad weather conditions.[28,29] Note that the potato disease had also stuck the crop in 1845. The two harvest failures in a row were disastrous since potatoes and grain were essential components of the Dutch diet.[28,30] The most tragic period was September 1846–September 1847. The provinces of Friesland, Zeeland, and Utrecht were relatively badly affected by the famine.[31] We use a dummy characterization of famine exposure at birth in most analyses. Additionally, we instrument access to food using variations in yearly average real market prices of rye and potatoes for the period 1812-1902 and for the three regions at hand. Food prices are often used in historical studies for modelling access to food.[32,33] The rye and potato market real prices almost doubled between 1844 and 1847.

Recent literature demonstrates long-run effects on longevity of the economic and diseases environment in early life.[34-37] Poverty and the prevalence of infectious and digestive diseases may considerably increase during famines, due to e.g. social and economic disorders, changes in food composition or debilitated immune systems.[8,38,39] If we do not control for this, this may be picked up by the famine parameters. The pathogen and economic conditions at birth are instrumented using historical time-series on infant mortality rates, and on the Gross National Product (GNP).[34,40,41] The GNP substantially decreased in 1845-47, compared to the previous years. As far as we know, there is no official statistics on infant mortality rates covering the full observation window.[42] Therefore, we computed the mortality rates using the vital information of the HSN data. The computed rates are slightly lower than the official statistics for the period 1865-1900.[42]

Note that 23% of the infants died before age 1 in 1846-47, compared to about 18% in the preceding years.[42] Dummy variables indicating periods of high infant mortality and the logarithm of annual real per capita GNP (in 1,000 euros with 1995 as base year) are used in the analyses.

Finally, our parametric analyses are corrected for current (economic) conditions (characterized by the GNP, World War II and most severe epidemics: the cholera epidemic of 1849 in Utrecht, the smallpox epidemic of 1870, and the influenza epidemic of 1918).

Individual variables

Beside age and gender, our survival analyses are corrected for parental and individual characteristics at birth: mother's marital status, father's socioeconomic status, whether he was a farmer and whether he was illiterate, province and season of birth, and whether the individual is born in a city or not. Recent literature consistently shows higher mortality at ages 50 and older for individuals born in spring.[3] The HSN data provides no information on cause of death. Table 1 presents descriptive statistics for birth cohorts exposed and not exposed to famine. The survival statistics are based on uncensored information and, most probably, underestimate the actual survival as censoring mostly occurs for individuals who survived infancy.

< Table 1 here>

NON-PARAMETRIC ANALYSES

Methods: We tested equality of means in total and residual life expectancies of individuals exposed (treated) and not exposed (controls) to famine early in life. We also checked the equality of their distributions of survival times using the Kolmogorov-Smirnov test.[43] We

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tested one-sided, since the hypothesis predicts that experiencing famine in early life will reduce survival. In preliminary analyses we checked for trends in life expectancies in the period 1840-1952 by testing whether there were statistically significant differences in total and residual life expectancies between periods 1837-1844 and 1848-1855.

Results: Table 2 summarizes the main results.

< Table 2 here >

First, we find no evidence of marked trends in total and residual life expectancies in the periods 1837-55 (p-values   0.11 for both genders at ages 1, 5, 40 and 50). This is in line with the observed historical developments.[28,30]

Second and most importantly, men born between September 1st 1846 and December 1st 1847 (those exposed to severe famine at birth and/or during at least six gestational months) have a significantly shorter life expectancy at age 50 than men born after the famine (with 3.1 years; p-value = 0.02) and before the famine (with 1.4 years ; p-value = 0.04). The figures equal 5.2 years (p-value = 0.006) and 3.3 years (p-value = 0.02) respectively, for individuals born in 1/3/47-1/12/47 (those with at least six exposed gestational months) and 4.9 years (p-value = 0.05) and 3.2 years (p-value = 0.08) respectively for individuals born in 1/9/47-1/12/47 (those born after the famine and with at least 6 exposed gestational months). Men born during the famine live also shorter at age 40 than controls, but the differences are not statistically significant at a 5% level (p-values 7 0.08). As shown previously, trends in life expectancy do not explain the results. Note that men born before the famine were also exposed to nutritional restrictions in 1845-46 and to the famine of 1846-47 later in childhood, which may have affected their survival as well.

These results are to a large extent unchanged when we considered alternative control periods within 1837-44 and 1848-55 or when other cut-off points around 50 years are used. The loss in life expectancy at age 50 reduces to 1.8 years for individuals born and/or with a least 4 gestational months during severe famine (born in 1/9/1846-1/2/1848; p-value = 0.04) compared to controls born after the famine. The differences at all ages are not statistically significant when we consider shorter gestational periods of exposure (e.g. born in 1/9/1846-1/3/1848) or individuals born in the first six months of the famine (born in 1/9/1846-1/3/1847, p-values 7 0.34 at ages 40 and 50).

Remarkably, we find no significant results for men exposed at ages 0-2 to severe famine (e.g. men born in 1/9/1844-1/9/1846 and in 1/9/1844-1/9/1845 (p-value 7 0.33 at all ages and for different control periods)).

The results on women show that all differences in total and residual life expectancies are not statistically significant (p-values 7 0.10). Note that on average the residual life expectancy at ages older than 40 is lower for women exposed to famine at birth than for controls.

Finally, the Kolmogorov-Smirnov test indicates that the survival curves after age 50 of the treated and controls born after the famine differ significantly (p-value = 0.03). The maximum difference between the two distributions is 0.15 at about age 56.

PARAMETRIC ANALYSES

Methods: We also estimated survival models (using Gauss 5.0) in order to correct for other relevant mortality determinants, albeit at the expense of functional-form assumptions. For

these, we used individual characteristics, our macro determinants of mortality in early life and current macro conditions. These variables are described in the data section.

We used proportional hazard models with time dependent covariates, in which we interacted the age dependence function with current macro-conditions and with the macro-conditions in early life. This allows us to investigate the life-course effects of early conditions.

For the age dependence we adopted a piecewise constant specification with 10 different age intervals. We only included individual characteristics at birth as opposed to later in life, since the latter may be endogenous or confounded. To avoid spurious associations between trends in macro determinants at birth and current macro variables, we decided to include only the cyclical components of the conditions at birth in the final specification. The cyclical components are computed using the Hodrick-Prescott filter with smoothing parameter 500 and are very robust with respect to the smoothing parameter. See [34] for additional details on the survival model.

Finally, we used the model's estimation results to calculate residual life expectancies had the famine well or not occurred . The residual life expectancy at age 50 is computed as the integral between 50 and infinity of the survival function associated with our model.[45] The life expectancies are computed for a typical individual with average socio-economic characteristics and average macro conditions of the individuals born in the same birth cohort.

Results: Table 3 reports the estimation results on the early life factors per gender. The results on additional controls are available on request.

< Table 3 >

Most importantly, men exposed to severe famine at birth live significantly shorter at age 50 than others. The corresponding parameter for women is positive but not statistically significant at a 5% level. Exposure reduces residual life expectancy of men by about 4.2 at age 50.

Again, we do not find any statistically significant effect of famine on survival at ages younger than 50. Furthermore, we find no evidence of long-run effects for individuals born in 1/9/1847-1/2/1848 (those born after the famine and exposed to at least four gestational months) (analyses not presented here).

All above results remain unchanged when other cut-off points around 50 years are used and when the nutritional shock is characterized by the numbers of days exposed to the famine after birth.

We find no significant effect of food prices at birth and no strong statistical evidence of effects of exposure to infectious diseases and of GNP at birth on mortality at older ages. The above results are robust to alternative characterizations of food prices and infant mortality.

DISCUSSION AND CONCLUSIONS

The non-parametric and parametric analyses strongly agree. Both suggest that survival at age 50 of men is significantly reduced by famine exposure in utero (at least four months) and the first months of life. Results concerning women show the same patterns, but are not

strongly statistically significant. This is in line with the Barker's fetal origins hypothesis which argues that most long-run effects are initiated during gestation[1] and with the recent literature showing long-run effects on morbidity.[20-24] However it differs from the one of Kannisto et al.[17] We believe that strong "selection of the fittest effects" may have masked the long-run effects in the Finnish study.

Note that our data do not allow the precise identification of the most critical periods of exposure. Indeed, only 44 men and 38 women are born in e.g. 1/3/1847-1/12/1847 and these individuals are not only exposed to famine during at least six gestational months but also on average during the first three months of life. The results suggest that the period directly following birth may also be important in explaining the observed relationship. Possibly, the baby's body and immune system were very vulnerable to health shocks after birth due to very poor living conditions and low prevalence of breastfeeding in those days.[28,44]

Moreover, the strongest long-run effects are initiated in the period 1/3/1847-1/9/1847. This may be due to either exposure during at least six gestational months, or increased severity of the nutritional restrictions, or seasonal effects. Note that the long-run effects for men remain in the survival analyses in which we corrected for seasonal effects.

Finally, the results are much stronger for men. This may be due to a so-called "male vulnerability" because males have an unprotected Y chromosome or because boys are born with less mature respiratory systems than girls.[46,47] An other explanation may be the higher proportion of body fat of females, which may make the females less at risk in period of famine.[39]

The validity of our results rests mostly on the absence of confounding of the instruments used for the early nutritional conditions. First, with respect to this, we corrected the parametric analyses for the diseases and economic conditions at birth and for the season of birth. This did not substantially affect the link between early nutrition and longevity. However, the observed relationship may still be partly confounded if the proxies for diseases and economic early environment are not perfect.

Second, individuals with higher socioeconomic status may postpone the birth of their children during famine. Since people with higher socioeconomic status live on average longer, babies born during famine will have on average shorter life expectancies. We compared the percentage of birth per socioeconomic status during the famine and in the years before and after the famine, but we could not find any statistical evidence of changes (see also Table 1).

Third, birth rates may decline during famine due to e.g. decreased libido, decreased reproductive ability and separation of spouses.[39] Since individuals with lower socioeconomic status are likely to be more affected, this will induce higher (residual) life expectancy during famine. If this is present, then the true effect will be larger than what we observe.

The disadvantage of working with historical data may be that the results are less relevant for the current time. However, recent literature demonstrates long-run effects of early nutrition on morbidity at young ages nowadays.[3,23,24,26] Moreover, if we want to analyze the full mechanisms, we are forced to use data on past generations. Finally, our results may be highly relevant in developing countries confronted to severe nutritional stresses. Currently, about one third of the world's children is underweighted or stunted.[48] Our results also

underline the potential dangers of monocultures, which may more easily lead to severe food restrictions in case of calamities.

To conclude, this is, to our knowledge, the first evidence suggesting a causal relationship between nutritional stresses in early life and mortality at old ages for men. What most importantly can be learned from this study is that interventions directed to infants in periods of nutritional stresses may have long-run consequences on mortality at old ages. Future research should focus on disentangling the roles of early pathogen and nutritional conditions, and on understanding how the causation is mediated through life.

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Table 1. Descriptive statistics cohorts exposed and not exposed to famine (cleansed sample)

Date of birth / Variables	*1/9/1837- 30/8/1844*	*1/9/1844- 30/8/1845*	*1/9/1845- 30/8/1846*	*1/9/1846– 30/8/1847*	*1/9/1847– 1/6/1848*	*1/9/1848- 1/9/1855*	Total
Males	338	59	54	43	35	366	895
Females	302	56	67	52	32	336	845
Social class (1-6):	2.3	2.4				2.1	2.3
Father literate (%)	80.6	82.9				85.3	83.0
Father farmer (%)	18.7	16.6				14.2	16.4
Born in a city (%)	18.3	19.9				22.2	20.2
Lifetime duration	30.9	30.0				31.6	31.0
% death under age 1	25.9	29.8				29.8	28.4
% death under age 5	40.6	43.8				44.4	42.9
% death under age 50	66.4	65.7				62.4	64.7

Table 2: (Residual) life expectancies of cohorts exposed or not exposed to the famine[*]

(Figures into brackets are numbers of individuals per group and figures in bold are p-values of statistical tests for difference in means between individuals born during famine and controls)

Date of birth	Total Life Expectancy (#)	Residual Life Expectancy			
		at age 1 (#)	at age 20 (#)	at age 40 (#)	at age 50 (#)
MALES					
1/9/1846–1/12/1847 (born or at least 6 gestational months during famine)	29.3 (47)	38.2 (35)	37.4 (22)	26.3 (17)	17.4 (16)
C O N T R O L S 1/9/1848-1/9/1855 (born after famine)	30.9 (316) **0.37**	42.5 (224) **0.23**	45.0 (142) **0.03**	30.5 (124) **0.08**	20.5 (114) **0.02**
1/9/1837-1/9/1844 (born before famine)	26.9 (306) **0.68**	38.5 (208) **0.48**	37.4 (133) **0.42**	28.8 (100) **0.24**	18.8 (87 **0.04**
FEMALES					
1/9/1846–1/12/1847 (born or at least 6 gestational months during famine)	32.5 (41)	46.4 (28)	44.3 (20)	29.5 (17)	20.7 (16)
C O N T R O L S 1/9/1848-1/9/1855 (born after famine)	31.0 (270) **0.60**	43.4 (188) **0.67**	46.1 (121) **0.34**	32.5 (102) **0.15**	22.5 (97) **0.10**
1/9/1837-1/9/1844 (born before famine)	34.1 (275) **0.60**	41.8 (219) **0.76**	41.9 (145) **0.69**	30.5 (114) **0.38**	20.6 (104) **0.20**

[*] The life expectancies are calculated after exclusion of the censored observations and might therefore underestimate the true survivals.

Table 3. **Results survival model per gender (excerpt*)**

Effect early macro-conditions on mortality later**	MALE		FEMALE	
	par.	**T-stat.**	**par.**	**T-stat.**
Born in 1/9/1846-1/12-1847 on mort. at ages 2-10	-0.20	-0.4	-0.15	-0.9
Born in 1/9/1846-1/12-1847 on mort. at ages 11-49	0.36	1.1	-0.49	-0.9
Born in 1/9/1846-1/12-1847 on mort. at ages 50+	0.94	2.3	0.78	1.7
Rye price at birth on mortality at ages 50+ ***	-0.00	-0.4	-0.00	-0.6
Potato price at birth on mortality at ages 50+	0.00	0.1	-0.01	-0.5
Rye price at birth on mortality farmers at ages 50+	-0.02	-1.1	-0.02	-1.2
Potato price at birth on mort. farmers at ages 50+	-0.01	-0.3	0.04	1.2
High infant mortality at birth on mort. at ages 2-15	0.23	0.9	-0.53	-0.4
High infant mortality at birth on mort. at ages 16-49	-0.07	-0.2	0.53	2.0
High infant mortality at birth on mort. at ages 50+	-0.24	-1.1	-0.18	-0.7
GNP at birth on mortality at ages 2-10	-1.65	-0.5	-2.38	-0.7
GNP at birth on mortality at ages 11-49	2.45	0.7	-0.46	-0.0
GNP at birth on mortality at ages 50+	3.22	0.8	2.94	0.8
-log likelihood		2,358		2,129
Number of individuals		824		765

* The results on individual characteristics and current conditions are available on request by the authors.

** We investigated the effect of the cyclical component of each macro-variable included in the analyses.

*** For reasons of parsimony, we only investigated the long-run term effects of the food prices at ages older than 50.