Effects of Early-Life Conditions on Adult Mortality Decline in the Netherlands 1860-1969

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Abstract: How important are improvements in early-life conditions in adult mortality decline? Properly controlling for period effects in an age-period-cohort model this paper estimates the contribution of early-life conditions to mortality decline above age 40 in the Netherlands between the onset of decline until the medical innovations of the 1970s. Early-life conditions explain about a third of the adult mortality decline between 1860-74 and 1965-69. Increased height and infant mortality decline explain slightly less than half of the cohort influences (or more than fifteen percent of the decline). While infant mortality has a significant effect on adult mortality, improvements in health conditions in childhood, as measured by infant mortality decline, only make a marginal contribution to adult mortality decline.

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Evidence that early-life conditions may affect an individual's mortality risks as an adult has been accumulating for some time now. Previous research has mostly focused on correlations of adult mortality with exposure to infectious diseases in childhood and with nutrition. Exposure to infectious diseases in childhood may have long-term effects. In the absence of measures of exposure to infectious diseases in childhood, researchers use infant and early childhood mortality as a proxy (e.g. Barker and Osmond 1986; Bengttson and Lindström 2000; Caselli and Capocaccia 1989; Crimmins and Finch 2005). Infectious diseases in childhood may affect adult mortality either directly or through adult height.

One's physique may also be related to adult mortality risks. An individual's height at the end of childhood is probably the best single indicator of that individual's dietary and infectious disease history (Elo and Preston 1992, p. 203). In the absence of direct measures of nutrition, some use food prices in early childhood, while others use adult height as a proxy. Fogel (1991), for example, uses final height as a net measure for nutrition, while Bengttson and Lindström (2000) use rye prices as a proxy for nutrition. Van den Berg et al. (2006) use GNP per capita as a proxy for economic conditions around birth. Bozzoli et al. (2009), however, call for caution when using adult height as a proxy for nutrition. They show that among European cohorts born between 1950 and 1980 the fall in postneonatal mortality can account for almost all of the increase in adult heights.

There are also processes, however, that may create a negative relationship between early-life conditions and adult mortality: selection and acquired immunity

(Elo and Preston 1992). Covering a period from 1907 to 1978 in Italy, Caselli and Capocaccia (1989, p. 152), for example, report that higher mortality early in life is associated with higher adult mortality before age 45, but with *lower* mortality levels after age 45.

How important are improvements in early-life conditions in adult mortality decline? In what is now a classic paper, Kermack, McKendrick and McKinlay (2001) have established the predominance of cohort influences in the English mortality decline from 1841 to 1931. Preston and van de Walle (1978) reported similar results for urban France. Unless a cohort had experienced reduced mortality as children, they experienced little or no advantage as adults. Fogel (1991, pp. 35 and 39) claims that improvements in height can explain nearly all of the decline in adult death rates in England and France between 1750 and 1875, and about half of the decline since 1875. In a regression model of mortality among Union Army veterans, Costa (2004) estimated that changes in anthropometric measures explain up to 47% of mortality decline at older ages from 1914 to 1988. None of these studies, however, uses age-period-cohort models to estimate the relative contribution of early-life conditions.

In this paper we adopt a longer time frame than most previous research to estimate the relative contribution of improvements in early-life conditions of people born from 1813 until 1921 to adult mortality decline in the Netherlands from 1860 to 1970 under proper control for period effects. Using an age-period-cohort model our results indicate a more limited role of early-life conditions in adult mortality decline than suggested by previous research. We estimate that cohort influences contributed about a third to mortality decline before 1970. Less than half of the cohort effect is explained by increased height. Infant mortality decline only makes a marginal contribution.

Data and variables

The data in this study are taken from the Historical Sample of the Netherlands (HSN), Data Set Life Courses Release 2008.01. The HSN is a stratified sample of 77,941 birth certificates (about 0.5 percent of all births) in the period 1811-1922 (Mandemakers 2000 and 2001).[1]

Between 1860 and 1969 life expectancy at age 40 for men and women rose from 25.0 and 26.6, respectively, to 32.9 and 37.4 (see Figure 1). Until 1950 trends are very similar for both men and women. After 1950 mortality among men increases, while it continues to decline among women.

[Figure 1 about here]

There are no age-specific mortality rates available before 1840. Hence, Mandemakers and van Poppel (2002) used HSN data to estimate the infant mortality rate (IMR) and early childhood mortality rate ($_1m_4$) in 1813-1922. Using an expanded HSN data base we computed new estimates of the infant mortality rate and early childhood mortality. Van der Bie and Smits (2001) published a time series of the infant mortality rate starting in 1840. Although our estimates are usually lower than those published by van der Bie and Smits (2001), Figure 2 shows that trends in the two series are very similar.

[Figure 2 about here]

Brinkman, Drukker, and Slot (1988) published a time series of median heights (in mm) of Dutch conscripts from 1863 to 1940. These data were revised by Mandemakers and van Zanden (1993). Drukker and Tassenaar (1997) extended this time series even further back in time to include the conscription years 1818-1863. Figure 3 compares the height series with IMR and shows that the two series are not

highly correlated. From the 1840s until the late 1870s both the infant mortality rate and median heights increased. Bozzoli et al. (2009) show that among European cohorts born between 1950 and 1980 the fall in postneonatal mortality can account for almost all of the increase in adult heights. Below we will show that among older cohorts born in the Netherlands between 1813 and 1921 adult height does not mediate the effect of postneonatal mortality on adult mortality.

[Figure 3 about here]

There are no height data for 1922 and no IMR is available for 1811-12. Hence, the analysis is limited to people born in the period 1813-1921.

Analytic Approach

A discrete-time hazard model is used to assess the effects of the independent variables on survival after age 40. We have assumed that the hazard is constant within annual intervals. We estimate discrete-time event-history models using logistic regression. This kind of analysis can accommodate two common features of event histories: censored data and time-varying variables (Allison 1982).

The dependent variable in the statistical model is the annual log odds of dying. The unit of analysis is the "person-year"; that is, each person contributes as many units to the analysis as the number for which he/she is observed. Person-years below age 40 were omitted from the analysis. Records were right-censored at age 90 or at the end of 1969, whichever came first. After left-truncation at the beginning of 1860, men and women contributed 635,526 person-years to the analysis.

Age-period-cohort models are particularly useful to detect the distinct impacts of age, period, and cohort on some outcome of interest. Disentangling the distinct effects of age, period and cohort, however, involves a methodological problem,

because the three are perfectly correlated. There are at least three conventional strategies for identification and estimation: (1) constraining two or more of the age, period, or cohort coefficients to be equal; (2) transforming at least one of the age, period or cohort variables so that its relationship is nonlinear; and (3) assuming that the cohort or period effects are proportional to certain measured variables (Yang and Land 2006).

Mason et al. (1973) point out that the identification problem can be solved by imposing equality constraints on categories of age, period and/or cohort. One criticism of this method is that estimates of model effect coefficients are sensitive to the arbitrary choice of the identifying constraint. A second strategy is to parameterize the effect of age as a polynomial (Mason et al. 1973; Raftery, Lewis, and Aghajanian 1995; Yang 2008). While the use of a polynomial may solve the problem of identification or extreme multi-collinearity, high levels of multi-collinearity may remain a problem in models of *change*. Simulation studies have shown, however, that the deleterious effects of multi-collinearity may be largely offset when the sample size is large and the independent variables explain a high proportion of the variance in the dependent variable (Mason and Perreault 1991; and Grewal et al. 2004). The analysis presented below is based on a very large sample, while period and cohort dummies explain a high proportion of the *temporal* component of the variance.

We chose to parameterize the effect of age as a cubic function. While the use of a polynomial solves the problem of the arbitrary choice of the identifying constraint, this approach still is not very informative about the mechanisms by which period-related changes and cohort-related processes act on the dependent variable of interest.

"Period" is a poor proxy for some set of contemporaneous influences, and "cohort" is an equally poor proxy for influences in the past. When these influences can themselves be directly measured, there is no reason to probe for period or cohort effects (Hobcraft, Menken, and Preston 1982). Hence, a third strategy is to constrain the effects of period and/or cohort to be proportional to some other substantive variable. Heckman and Robb (1985) term this the “proxy” variable approach because period and cohort are represented by some other variable. We use three proxies for the cohort effect: the infant mortality rate (IMR), the mortality rate at age 1-4 and height at age 20. The "proxy" variable approach, however, also has its drawbacks. Replacing the cohort dummies by proxies may lessen the rigorousness of the control for the period effects on cohort differences (O'Brien 2000, p. 125). Although replacing an accounting dimension with measured variables solves an identification problem, it makes room for specification errors (Smith, Mason and Fienberg 1982). If the use of proxies does not lessen the rigorousness of the control for cohort differences, however, then period effects in the "proxy" variable approach should resemble cohort differences in the approach that uses cohort dummies. In order to determine the extent to which the use of proxies lessens the rigorousness of the control for cohort differences, we compare period effects in both strategies. After replacing cohort dummies with proxy variables, there is no need to replace age dummies by a polynomial in order to identify the model. We retained the polynomial, however, to enhance comparability of the period dummies in the second and third model.

If the cohorts and time periods are unique entities, then conventional statistical methodology guidelines suggest that it might be more appropriate to model them with a fixed-effects specification. Hence, we model periods and cohorts as fixed effects. Yang and Land (2006 and 2008), however, argue that when sample sizes within each

cohort and/or period are unbalanced mixed (fixed and random effects) models use the available information in the data more efficiently than fixed-effects models. They warn that the standard errors of estimated coefficients of conventional fixed-effects regression models may be underestimated, leading to inflated t-ratios and actual alpha levels that are larger than nominal levels of significance. To minimize this problem we use a nominal level of significance of 2.5 percent.

Period effects are measured by nineteen period dummies indicating whether the current year is in the period 1860-74, 1875-79, 1885-89, 1890-94, 1895-99, 1900-04, 1905-09, 1910-14, 1915-19, 1920-24, 1925-29, 1930-34, 1935-39, 1940-44, 1945-49, 1950-54, 1955-59, 1960-64, 1965-69 – 1860-74 being the reference category. Cohort effects are captured by nine cohort dummies indicating whether the woman was born in 1813-39, 1840-49, 1850-59, 1860-69, 1870-79, 1880-89, 1890-99, 1900-09, or 1910-21 – the pre-1840 birth cohort being the reference category.

Results

Table 1 presents six models of the decline in adult mortality. Coefficients are presented as odds ratios or exponents of the raw logistic coefficients. The odds ratios are multiplicative effects on the odds of giving birth in any one-year interval. A coefficient of 1.00 represents no effect, a coefficient greater than 1.00 represents a positive effect, and a coefficient less than 1.00 represents a negative effect on the odds.

[Table 1 about here]

The first model includes age, sex and period dummies (AP-1). The period effects show that adult mortality started to decline in the early 1880s, at about the same time when infant mortality starts to decline (see Figure 2). In the early 1940s

adult mortality increased remaining relatively high until the 1970s. The cause of this increase is unknown. It is mostly due to increased mortality among men. Hence, the second model adds interaction effects between periods after 1940 and sex (AP-2). Differences between men and women before 1960 are now much smaller. The AP-2 model shows that in the 1960s mortality among women had reverted to the level of the 1930s. The interaction effects show that mortality trends among men and woman did not diverge until the 1960s. The inclusion of interaction effects between periods and sex, turns our analysis of the decline of adult mortality effectively into an analysis of the decline of *female* adult mortality.

The third model adds cohort effects (APC). After a small increase among those born in 1840-49, perhaps due to the Potato Famine, mortality starts to decline among those born in 1850-59, at about the same time as the height of conscripts starts to increase, but before the decline in infant mortality (Figure 3). If not for cohort influences, mortality would have declined less until 1940. The APC model indicates that the rise in mortality after 1940 was a period effect, perhaps due to increased tobacco consumption, as suggested by Van Poppel (1985). If not for cohort influences, not only the level of male mortality, but the level of female mortality would also have been higher in the 1960s than in the 1930s.

How much of the decline is due to cohort effects? Figure 4 presents period mortality trends in terms of odds ratios. The AP-2 model (thick dashed line) shows that between 1860-74 and 1965-69 female mortality declined by 65 percent in terms of odds ratios. The period mortality trends in terms of odds ratios in the APC model (thick line) are net of cohort influences. The APC model shows that if not for cohort effects female mortality would have declined by 43 percent only. Thus, early-life conditions explain about a third [= 100 . (65-43) / 65] of the decline.

[Figure 4 about here]

In order to identify cohort effects, the fourth model (APEM) replaces the cohort dummies with measures of infant and early childhood mortality. While infant mortality has a significant effect in the expected direction, early childhood mortality does not have a significant effect. Figure 4 shows that the period mortality trends in terms of odds ratios in the APEM model (thin dashed line) are very similar to those in the AP-2 model (thick dashed line). Thus, infant and early childhood mortality make a marginal contribution to cohort influences.

The fifth model (APH) replaces the cohort dummies with height. Height has a significant effect in the expected direction. A comparison of the period effects in the APC and APH models shows that the use of height as a proxy only partially lessens the rigorousness of the control for cohort differences on the period effects. Hence, the APH model overestimates period effects, while underestimating the cohort influences.

How much of the cohort effect is due to increased heights? If height would not have increased female mortality would have declined by 51 percent by 1965-69. Thus, height explains more than twenty percent [=100 . (65-51)/65] of the decline. Thus increased height makes a much larger contribution to cohort effects than infant mortality.

The last model (APPROX) replaces cohort dummies with three proxies. How much of the cohort effect is due to the combined effects of increased heights and a decline in infant and early childhood mortality? If height would not have increased and infant mortality would not have declined (female) adult mortality would have declined by only 55 percent by 1965-69. Thus, the three proxies explain more than fifteen percent [=100 . (65-55)/65] of the decline or slightly less than half of the cohort effects.

As younger and healthier cohorts replace older cohorts over time the contribution of cohort influences becomes larger. Hence, our estimate of the contribution of cohort influences to adult mortality decline would be much smaller, if we would have stopped our analysis in 1940.

Conclusion and discussion

Mortality above age 40 in the Netherlands started to decline in the early 1880s. Few studies provide an empirical answer to a question of prime interest: How much of the adult mortality decline is due to cohort effects? There is plenty of research on cohort effects in adult mortality. An estimate of the relative contribution of period and cohort effects to adult mortality decline using an age-period-cohort model, however, is rarely provided. Caselli and Capocaccia (1989) applied an age-period-cohort model to Italian data, but were unable to separate period and cohort effects completely. They did estimate a model (APEM) that replaces the cohort effect by a proxy: infant and childhood mortality, but do not provide an estimate of the contribution of infant mortality decline to adult mortality decline.

Three major findings emerge from our analysis. First, cohort effects explain about a third of the adult mortality decline between 1860-74 and 1965-69. Second, increased height explains slightly less than half of the cohort effects. Third, infant and early childhood mortality only make a marginal contribution to adult mortality decline. Our estimates of the contribution of early-life conditions to adult mortality decline are much lower than previous ones. Differences with previous results may be because of the method, the setting – The Netherlands – or a longer time frame. Note also that cohort effects may be weaker at older ages because of selection of the weak before age 40 (Leon et al. 1995).

Bengttson and Lindström (2000) use rye prices around the time of birth as a proxy for nutrition. While they report a significant effect of childhood mortality on adult mortality, they found no significant effect of rye prices. Baten (2009) has shown that milk production per capita explains a lot of the variation in height of conscripts between areas in nineteenth-century Bavaria, Prussia and France. Perhaps, a time series of average milk consumption around the year of birth would have provided significant results. Following Fogel (1991), we use adult height as a proxy for nutrition in childhood. We report a significant effect of adult height on adult mortality. However, height is not only a function of nutrition but also of health. Childhood mortality does not attenuate the effect of height on adult mortality to any large extent. Hence, to the extent that health is measured by infant mortality, height is mostly a function of nutrition. Probably, these results cannot be extrapolated to later cohorts. In a survey of Americans aged 55-64 in 1996, Blackwell, Hayward and Crimmins (2001) found no support for using adult height as a proxy for the effects of childhood health experiences. Bozzoli et al. (2009) show that among European cohorts born between 1950 and 1980 the fall in postneonatal mortality can account for almost all of the increase in adult heights.

Like Barker and Osmond (1986), Bengttson and Lindström (2000) and Crimmins and Finch (2005), we report a significant effect of infant mortality, but no significant effect of early childhood mortality, on current adult mortality. They do not, however, provide an empirical answer to the question: How much of the adult mortality decline is due to infant mortality *decline*? In the Netherlands, infant mortality decline only makes a marginal contribution to adult mortality decline. Kermack et al. (2001) reached a similar conclusion regarding the adult mortality decline in England and Wales until 1925. Hence, to the extent that health is measured

by infant mortality, improved child health did not contribute to adult mortality decline. Perhaps processes that create a negative relationship between infant and adult mortality, such as selection and acquired immunity, cancel out the positive relationship (Elo and Preston 1992).

Notes

1. For more information about the HSN, see www.iisg.nl/~hsn.

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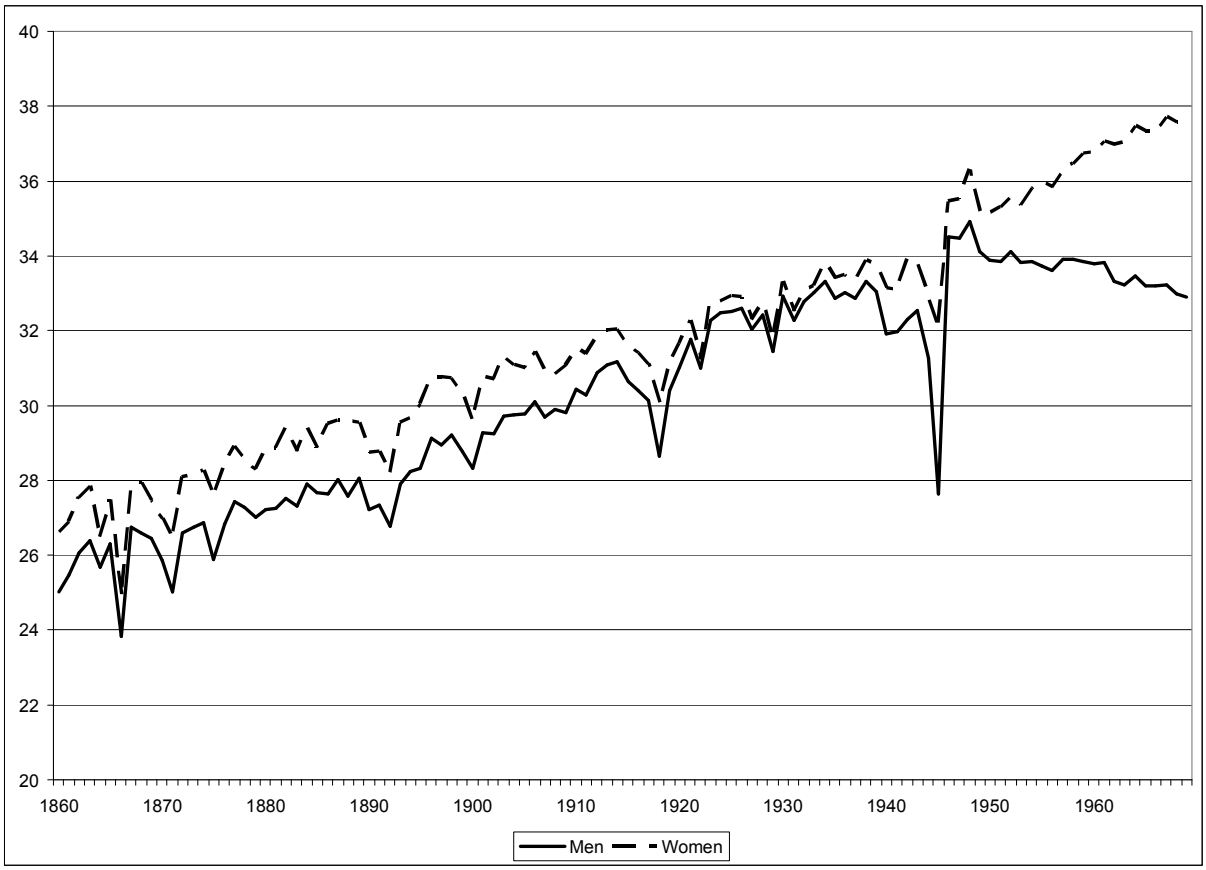
Table 1. Discrete-time survival analysis, Netherlands 1860-1969.

Model	**AP-1**		**AP-2**		**APC**	
Variable	e^b	***p*-value**	e^b	***p*-value**	e^b	***p*-value**
Age	1.528	.000	1.528	.000	1.558	.000
Age squared	0.993	.000	0.993	.000	0.993	.000
Age cubic	1.000	.000	1.000	.000	1.000	.000
Male	1.113	.000	1.058	.056	1.055	.069
Period						
1860-74 (ref.)	1.000	-	1.000	-	1.000	-
1875-79	0.864	.312	0.864	.314	0.893	.436
1880-84	0.753	.038	0.753	.038	0.789	.087
1885-89	0.699	.005	0.698	.005	0.736	.021
1890-94	0.662	.001	0.662	.001	0.716	.010
1895-99	0.538	.000	0.538	.000	0.603	.000
1900-04	0.522	.000	0.522	.000	0.599	.000
1905-09	0.511	.000	0.511	.000	0.599	.000
1910-14	0.477	.000	0.477	.000	0.571	.000
1915-19	0.491	.000	0.490	.000	0.601	.001
1920-24	0.418	.000	0.417	.000	0.529	.000
1925-29	0.383	.000	0.383	.000	0.507	.000
1930-34	0.348	.000	0.347	.000	0.478	.000
1935-39	0.337	.000	0.336	.000	0.475	.000
1940-44	0.453	.000	0.451	.000	0.641	.020
1945-49	0.443	.000	0.441	.000	0.630	.022
1950-54	0.403	.000	0.411	.000	0.598	.015
1955-59	0.396	.000	0.380	.000	0.568	.011
1960-64	0.365	.000	0.333	.000	0.516	.004
1965-69	0.406	.000	0.352	.000	0.567	.019
Interactions: Period x Male						
1940-44			1.003	.966	1.006	.923
1945-49			1.004	.943	1.007	.903
1950-54			0.955	.457	0.959	.501
1955-59			1.076	.238	1.084	.192
1960-64			1.183	.008	1.195	.005
1965-69			1.302	.000	1.318	.000
Cohort						
1813-39 (ref.)					1.000	-
1840-49					1.141	.076
1850-59					0.750	.001
1860-69					0.814	.046
1870-79					0.843	.162
1880-89					0.759	.055
1890-99					0.756	.091
1900-09					0.627	.014
1910-22					0.511	.003
-2 Log likelihood	114818.185		114789.373		114711.976	

Table 1. Discrete-time survival analysis, Netherlands 1860-1969 (continued).

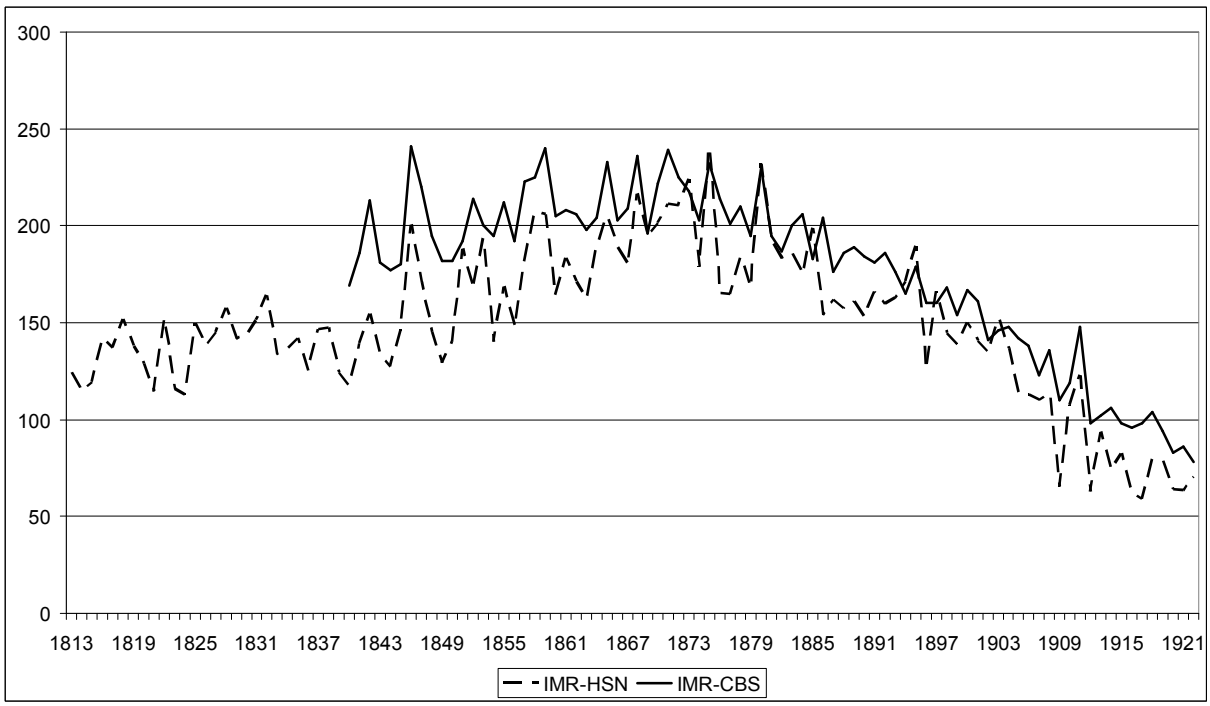
Model	**APEM**		**APH**		**APPROX**	
Variable	e^b	***p*-value**	e^b	***p*-value**	e^b	***p*-value**
Age	1.544	.000	1.535	.000	1.544	.000
Age squared	0.993	.000	0.993	.000	0.993	.000
Age cubic	1.000	.000	1.000	.000	1.000	.000
Male	1.060	.051	1.058	.055	1.060	.051
Period						
1860-74 (ref.)	1.000	-	1.000	-	1.000	-
1875-79	0.864	.314	0.869	.334	0.869	.332
1880-84	0.755	.040	0.763	.049	0.763	.049
1885-89	0.701	.006	0.716	.010	0.715	.009
1890-94	0.665	.001	0.685	.002	0.683	.002
1895-99	0.539	.000	0.562	.000	0.558	.000
1900-04	0.519	.000	0.544	.000	0.538	.000
1905-09	0.500	.000	0.530	.000	0.520	.000
1910-14	0.460	.000	0.497	.000	0.482	.000
1915-19	0.469	.000	0.519	.000	0.499	.000
1920-24	0.397	.000	0.451	.000	0.430	.000
1925-29	0.363	.000	0.424	.000	0.402	.000
1930-34	0.330	.000	0.394	.000	0.372	.000
1935-39	0.321	.000	0.392	.000	0.368	.000
1940-44	0.431	.000	0.540	.000	0.506	.000
1945-49	0.423	.000	0.543	.000	0.505	.000
1950-54	0.397	.000	0.520	.000	0.482	.000
1955-59	0.371	.000	0.494	.000	0.459	.000
1960-64	0.329	.000	0.446	.000	0.415	.000
1965-69	0.353	.000	0.487	.000	0.454	.000
Interactions: Period x Male						
1940-44	1.001	.983	1.003	.962	1.002	.978
1945-49	1.004	.944	1.005	.936	1.005	.940
1950-54	0.956	.465	0.956	.472	0.957	.473
1955-59	1.078	.224	1.079	.221	1.080	.214
1960-64	1.186	.007	1.186	.007	1.188	.006
1965-69	1.305	.000	1.305	.000	1.307	.000
IMR	1.001	.002			1.001	.009
${}_1m_4$	1.001	.677			0.999	.727
Height in mm			0.995	.000	0.996	.007
-2 Log likelihood	114776.380		114776.456		114769.152	

Figure 1. Life expectancy by sex at age 40 in the Netherlands, 1860-1969.



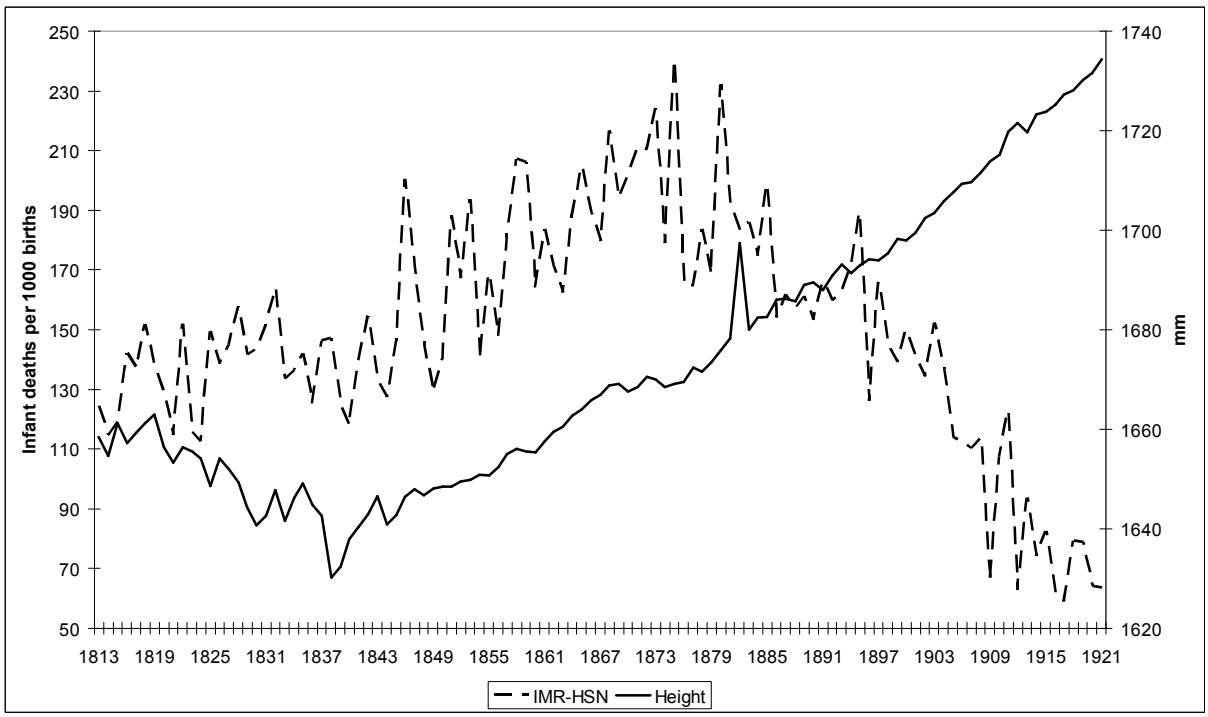
Source: Central Bureau of Statistics.

Figure 2. Infant mortality rate in the Netherlands as estimated from HSN data (IMR-HSN) and as published by the Central Bureau of Statistics (IMR-CBS), 1813-1921.



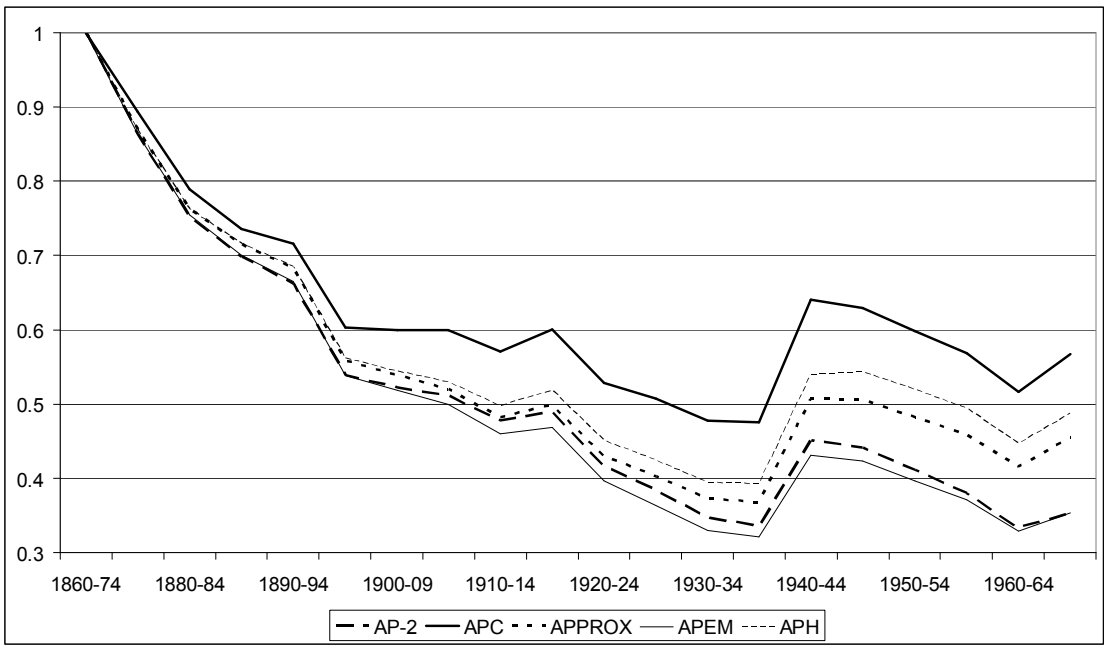
Source: HSN Data Set Life Courses Release 2008.01 and van der Bie and Smits (2001).

Figure 3. Infant mortality rate (IMR-HSN) and the height of conscripts in mm by year of birth in the Netherlands, 1813-1921.



Source: Same as Figure 2.

Figure 4. Period mortality trends in terms of odds ratios in five models, the Netherlands 1860-1969.



Source: Table 1.