Effects of Early-Life Conditions on Adult Mortality Decline in the Netherlands 1850-1999

Jona Schellekens and Frans van Poppel

March 2013

*Jona Schellekens, Department of Sociology and Anthropology, Hebrew University, Mount Scopus, Jerusalem 91905, Israel; e-mail: jona@vms.huj.ac.il; Frans van Poppel, Netherlands Interdisciplinary Demographic Institute (NIDI), The Hague, and Department of Sociology, Utrecht University, the Netherlands. Earlier versions of this paper were presented at the 2010 annual meeting of the Population Association of America, Dallas, and the 2010 European Population Conference, Vienna.

Effects of early-life conditions and adult mortality decline in the Netherlands 1850-1999

Abstract: How important are improvements in early-life conditions in adult mortality decline? This paper provides an estimate of the contribution of early-life conditions to mortality decline above age thirty in the Netherlands between the onset of decline in the 1870s until the end of the twentieth century. Whereas in the nineteenth century period-specific influences were more powerful than cohort-specific ones, in the twentieth century cohort-specific influences were more powerful. Increased height explains most, if not all, of the twentieth-century decline until 2000. Early childhood mortality decline, on the other hand, did not contribute to adult mortality decline after age thirty.

Effects of early-life conditions and adult mortality decline in the Netherlands 1850-1999

Mounting evidence suggests that early-life conditions have an enduring effect on an individual's mortality risks as an adult (e.g. Bengtsson and Mineau 2009). The contribution of improvements in early-life conditions to adult mortality *decline*, however, has received much less attention. One approach is to study the importance of unspecified cohort influences in adult mortality decline. The earliest studies heavily depended on graphical methods. Kermack et al. (2001 [1934]), for example, argued that cohort influences dominate the English adult mortality decline from 1841 to 1931. After updating their figures, however, Murphy (2010) showed that the existence of cohort patterns is not as clear cut as Kermack et al. (2001 [1934]) thought. In a much later paper that also used graphic methods, Preston and van de Walle (1978) concluded that in urban France cohort-specific influences were more powerful than period-specific ones. Unless a cohort had experienced reduced mortality as children, they experienced little or no advantage as adults.

Although a formal age-period-cohort model would seem to be a more appropriate method than graphic methods to estimate the relative importance of cohort influences, the contribution of formal age-period-cohort models to the debate has remained limited. Some concern only one cause of death, like Collins (1982) who applied an age-period-cohort model to show the predominance of cohort influences in tuberculosis mortality decline in England and Wales, Italy and New Zealand, or Wolfe and Burney (1992) who found that in England there are substantial cohort influences on trends in stroke mortality. Navaneetham (1993) has shown how cohort influences slowed down adult mortality decline in the 1980s, but his study relates to a

less developed country. Other studies relate to a more recent period in the more developed countries. Thus, Meza et al. (2010) tried to determine whether year of birth or year of death better correlates with observed patterns of adult mortality among the 1920 and 1944 birth cohorts in nine countries, whereas Yang (2008a) found that adult mortality decline in the United States in the last four decades of the previous century is mostly a cohort effect.

Cohort effects not only reflect early-life conditions, but may also reflect the impacts of lifelong accumulation of exposure to risk factors (Ryder 1965; Yang 2008a). Hence, another approach is to use proxies for early-life conditions. The most commonly used proxies are childhood mortality and anthropometric measures. Using anthropometric measures among Union Army veterans, Costa (2004) estimated that improvements in early-life conditions explain almost fifty percent of mortality decline at older ages from 1914 to 1988. Crimmins and Finch (2006) argued that in Sweden, France, Switzerland and England mortality decline before age 15 explains more of the decline in mortality of the same cohort at age 70-74 than contemporary changes in mortality before age 15. These results, however, have not convinced everyone. Barbi and Vaupel (2005) asserted that period-specific influences were more important during the decline. While they admit that early-life factors may affect late-life mortality, they argued that the effect is modest.

Using early childhood mortality as a proxy for early-life conditions, Myrskylä (2010) found that the majority of variation in adult and old-age mortality in England and Wales, Finland, the Netherlands, and Sweden before World War I is attributable to changing period conditions. Early childhood mortality only had a weak effect on adult mortality. Several studies altogether failed to detect a significant effect of early-life conditions on adult mortality. Gagnon and Mazan (2009) found little evidence for

an effect of the infant mortality rate on mortality after age fifty in pre-industrial Québec. Kannisto et al. (1997) did not find any evidence that famine *in utero*, in infancy or early childhood, influences survival in later life. Cohen et al. (2010) failed to detect any significant effect of the 1918-1919 influenza pandemic on late-life mortality. Cutler et al. (2007) were unable to detect any meaningful relationship between early-life economic conditions in the 1930s and late-life health.

Numerous previous studies have already documented the importance of early-life conditions as determinants of adult mortality. Few of these, however, deal with the contribution of early-life conditions to the *decline* in adult mortality. Moreover, those that do often are limited to a shorter time frame. Most studies of the *decline* in adult mortality only used one proxy for early-life conditions. To show that an improvement in early-life conditions does not explain the adult mortality decline, one proxy for early-life conditions may not suffice. Hence, this study will present results for two proxies for early-life conditions: early childhood mortality and an anthropometric measure.

Using a half percent random sample of individuals born in the Netherlands between 1812 and 1922 this study analyzes mortality decline among adults after age thirty from 1850 to 2000. Our results indicate that period-specific influences are more important in the nineteenth century, while cohort-specific influences are more powerful in the twentieth century. Like Costa (2004), we find that increased height explains much of the decline in the twentieth century. Our results also indicate that in the Netherlands early childhood mortality had a negative effect on adult mortality. If not for early childhood mortality decline, adult mortality would have declined even further, suggesting that selection and acquired immunity cancelled out the scarring effect of infectious disease.

Early-life conditions and adult mortality

We review the literature using an analytic framework adapted from Ben-Shlomo and Kuh (2002) in which two major pathways lead from adverse childhood socioeconomic position to adult mortality. The first pathway is predominantly socio-biological and leads from adverse childhood socioeconomic position to poor adult health through exposure to infectious agents and malnutrition in early childhood. The second pathway is predominantly social and leads from adverse childhood socioeconomic position to adult socioeconomic position (see also Bengtsson and Broström 2009).

Most of the studies reviewed below assume a socio-biological pathway between adverse childhood socioeconomic position and poor adult health through exposure to infectious agents and malnutrition in early childhood. Thus, Finch and Crimmins (2004) asserted that as a result of better sanitation and nutrition, increased income and the spread of medical knowledge and technology, decreased exposure to infectious diseases and other sources of inflammation in early life has led directly to a decrease in mortality from chronic conditions in old age. As an example they mention childhood streptococcal infections and infant diarrhea and enteritis. There are few studies, however, of the impact of a specific infectious disease. Collins (1982) has shown how exposure to tuberculosis in early childhood influences tuberculosis mortality later in life. Almond (2006) reported increased rates of physical disability among cohorts *in utero* during the 1918 influenza pandemic, whereas Brenes-Camacho and Palloni (2011) have shown that malaria survivors are more likely to die from stroke.

In the absence of measures of exposure to infectious disease in childhood, measures of infant and early childhood mortality are commonly used as a proxy (e.g. Barker and Osmond 1986; Ben-Shlomo and Smith 1991; Bengttson and Lindström 2000; Caselli and Capocaccia 1989; Crimmins and Finch 2006). These studies suggest that exposure to childhood disease may cause permanent physical damage ("scarring") leading to fatal outcomes later in life.

There are also processes, however, that may create a negative relationship between infectious disease in childhood and adult mortality: selection and acquired immunity (Elo and Preston 1992). Covering a period from 1907 to 1978 in Italy, Caselli and Capocaccia (1989: 152), for example, report that although higher mortality early in life is associated with higher adult mortality before age 45, it is associated with *lower* mortality after age 45.

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). Arm length has also been used as a proxy for nutrition, although leg length seems to be the more prominent measure (e.g. Wen and Gu 2011). Height is inversely associated with coronary heart disease, stroke, and respiratory disease mortality among men and women. Whereas smoking related cancer mortality is not associated with height, the risk of death from cancer unrelated to smoking tends to *increase* with height, particularly for haematopoietic, colorectal and prostate cancers, except for stomach cancer mortality, which is inversely associated with height (Davey Smith et al. 2000).

According to Cole (2003), the increase in adult stature from one generation to the next mostly results from longer legs, the extra leg growth occurring before the age of two. Most studies report a negative correlation between height and adult mortality

(e.g. Fogel 1994). Su (2009), however, observed that being taller is related to a higher relative risk of mortality for Union Army veterans who survived past 1910.

In the absence of anthropometric measures of nutrition, some use an exogenous variable such as food prices in early childhood. Bengttson and Lindström (2000), for example, used rye prices as a proxy for nutrition. They did not find any impact of the price of rye around birth on mortality at ages 55-79. Although the anthropometric evidence suggests that the price of milk may be a good proxy for nutrition, considering its effect on height (Baten 2009), there are no studies that use the price of milk as a proxy. Van den Berg et al. (2006), Janssen et al. (2006) and van den Berg et al. (2009), on the other hand, use gross domestic or national product per capita as a proxy for economic conditions around birth, whereas Hayward and Gorman (2004), van Poppel and Liefbroer (2005) and Schenk and van Poppel (2011) use socio-economic status of the parents to measure childhood conditions. Van den Berg et al. (2006) and van den Berg et al. (2009) found that economic conditions around birth affect mortality later in life. Janssen et al. (2006) reported a mostly negative correlation of GDP per capita prevailing at earlier ages with adult mortality. This correlation remained significant after control for GDP per capita prevailing at the time of death.

The second pathway is predominantly social and leads from adverse childhood socioeconomic position to adult socioeconomic position through adverse childhood exposures. Few studies control for adult socioeconomic position. Thus, it is not always clear to what extent their findings reflect the first of second pathway. Among those that do the evidence is mixed. Davey Smith et al. (2000), Kuh et al. (2002), Case et al. (2005), Power et al. (2005), and Wen and Gu (2011) show that after controlling for adult socio-economic status the statistical effects of early life

conditions remain significant. Davey Smith et al. (2000) reported that height is inversely associated with all cause, coronary heart disease, stroke, and respiratory disease mortality among men and women. Adjustment for socioeconomic position and cardiovascular risk factors had little influence on these associations. Kuh et al. (2002) found that manual origins and poor care in childhood remained associated with adult mortality even after adjusting for social class in adulthood or home ownership. Case et al. (2005) found that controlling for educational attainment, and for socioeconomic status and health in earlier adulthood, childhood health markers were significant predictors of health and economic status at age 42. Power et al. (2005) found that adverse social conditions in both childhood and adulthood were associated with higher death rates from coronary heart disease and respiratory disease. Wen and Gu (2011) found that childhood socioeconomic conditions exert long-term effects on functional limitations, cognitive impairment, self-rated health, and mortality independent of adult and community socioeconomic conditions.

Hayward and Gorman (2004), on the other hand, observed that the associations between childhood socioeconomic and family conditions and men's mortality were largely indirect through socioeconomic-achievement processes and lifestyles in adulthood. They found little to no evidence in support of the socio-biological pathway linking childhood circumstances to adult mortality, such as a physiological "scarring" effect, an acquired immunity effect, or a negative selection process. At least one study found no evidence for either pathway. Lynch et al. (1994) found that the increased mortality risk among low-income adults was not related to childhood economic conditions.

Thus, the evidence is highly variable, although most of the studies cited here would agree that early-life conditions affect adult mortality in some way. How are we

to interpret the lack of agreement? Of course, the studies reviewed here differ in their methodology, period and country of interest. Whereas the studies by Hayward and Gorman (2004), Lynch et al. (1994) and Wen and Gu (2011), for example, studied differences *within* cohorts, those by Bengttson and Lindström (2000), van den Berg et al. (2006) and van den Berg et al. (2009) compared *between* cohorts. Our approach differs from most previous research, because we ask a different question. Even if early-life conditions affect adult mortality it does not necessarily follow that an improvement in early-life conditions explains part of the adult mortality decline. Hence, we ask to what extent *changes* in early-life conditions contributed to adult mortality *decline*. Most of the studies reviewed here agree that early-life conditions affect adult mortality. This justifies our question.

Data and variables

The data used in the analysis come from the Historical Sample of the Netherlands (HSN), Data Set Survival Dates Release 2011.01. The HSN is a national database with information on the complete life history of a half percent random sample (78,000 birth records) of men and women born in The Netherlands between 1812 –the first year for which vital registration is available for the whole country– and 1922. In all Dutch provinces, a random sample of births was drawn, which was stratified by period of birth (eleven cohorts) and level of urbanization of the municipality (Mandemakers 2000, 2001, and 2002).[1]

Whereas before 1939 the sources used provide information about all sampled persons, the data sources used after 1939 provide information only about deceased persons. Thus, the life history of persons who died after 1939 but whose date of death was not found was censored in 1939. To the extent that the average person whose date

of death is unknown outlived the average person whose date of death is known, we may have overestimated the actual mortality risk after 1940. Schenk and van Poppel (2011), however, have shown that the HSN-mortality data only slightly underestimate the increase in expectation of life.

The dependant variable is mortality decline after age 30. This decision is based on the observation that the survival function after that age tends to be much more linear. Moreover, over time the age pattern of the survival changes much more before than after age 30.

The data set was censored at the end of 1999. To the extent that the age pattern changes over time, this may be problematic because the later cohort estimates are based on a small fraction of their experience.

There are two major sources for missing cases. First, among those born before 1863, only people born in the provinces of Friesland, Utrecht and Zeeland, and the city of Rotterdam were followed (Kok et al. 2009). Hence, we control for province of birth. The capital city of Amsterdam is the only one bigger than Rotterdam. For comparison, we also defined a variable indicating birth in Amsterdam. The indicator variables for the provinces of North and South Holland do not include Amsterdam or Rotterdam.

Second, there are missing cases as a consequence of the loss of population registers over time, especially those of Middelburg in Zeeland for 1850-1899 and those of Arnhem for 1850-1939, who were lost during the war.

There are two major causes of censoring. First, we stopped the follow-up at the end of 1999. Second, not all migrations were properly registered in the population registers (Mandemakers 2002: 89). Thus, our results are biased toward the sedentary population.

Some childhood diseases are more prevalent in certain provinces, providing another justification for our control on province and major city of birth. Malaria, for example, did not disappear from the coastal marshes in the Netherlands until the 1950s (Knottnerus 2002).

Figures 1 and 2 show the probability of twenty year survival (l_{x+20}/l_x) at ages 30, 50, and 70 in selected years for women and men, respectively. Until 1950 trends were very similar for both men and women. Note that during the Great Depression life expectancy did not decline. Thus, there are also important period influences in the decline. After 1950 mortality among adult men slightly increased, while it continued to decline among women (see also Wolleswinkel-van den Bosch et al. 1998). Van Poppel and van Ginneken (1985) suggested that the increased mortality among men is the result of increased tobacco consumption.

[Figures 1 and 2 about here]

We used the probability of survival until age five as a proxy for exposure to infectious disease by birth cohort. We estimated the probability of survival until age five from the HSN data set by single year of birth. Figure 3 shows that early childhood survival started to improve among those born in 1870s.

[Figure 3 about here]

We used the heights of conscripts as a proxy for the nutritional status of single-year birth cohorts by attributing the median height of conscripts measured in year *t* to all those born in year *t*-19. 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 (1990 and 1993) by correcting for bias due to the under-representation of men from more wealthy families and by adjusting for small changes over time in the age at measurement. Drukker and

Tassenaar (1997) extended the time series further back in time to include the conscription years 1818-1863. There is no comparable source of information on the height of women. Among prisoners born between 1815 and 1865, however, the increase in the fully-grown height of men and women was more or less the same (de Beer 2010). Figure 3 shows that height declined among those born before the 1840s and started to increase again among those born in the 1850s (compare with Komlos 1993). There are no height data for 1922. Hence, the analysis is limited to people born in 1812-1921.

There is synergy between illness and poor diet in the stunting of growth. Hence, our two indicators of the influence of early-life conditions, height and early-child mortality, are correlated. Below, we will discuss how this influences our results.

Once smoking is initiated, processes of physical and psychological dependence produce long-term continuity of smoking behavior (Chassin et al. 1996). Thus, smoking is a cohort effect. Preston and Wang (2006) have highlighted the role of cohort smoking patterns in changes in sex mortality differences. Following Preston et al. (2010) we use lung cancer mortality rates to estimate cohort smoking patterns. Van der Hoff (1979) estimated net lung cancer cohort risks for Dutch males born in 1860-1955. For the years that they overlap, however, the serial correlation coefficient between median heights of conscripts and lung cancer risks is very high ($r^2 = 0.925$). Hence we omitted a proxy for cohort smoking patterns from the analysis. This should not affect the analysis of female mortality, because among women born before 1922 lung cancer rates are only a fraction of those among men (Janssen-Heijnen et al. 1995).

Van Poppel and Liefbroer (2005) used the occupation of the father at birth to measure childhood conditions. Socio-economic background, however, may influence

adult mortality indirectly via the accumulation of wealth and obtained socio-economic status in adulthood (Bengtsson and Broström 2009). Although the birth records provide information about the occupation of the father, in the HSN data set this variable is only available for those whose life history was retrieved from the population registers. Hence the occupation of the father was omitted from the analysis.

In the first few decades the oldest age groups are not represented, whereas in the last few decades the youngest age groups are not represented. To the extent that trends differ by age group, this will influence our results in the first and last few decades under consideration. In particular, this is true for the oldest age group.

Analytic Approach

A discrete-time hazard model is used to assess the effects of the independent variables on survival after age thirty. We have assumed that the hazard is constant within annual intervals. We estimated 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 covariates (Allison 2010).

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 thirty were omitted from the analysis. Records were right-censored at age ninety or at the end of 1999, whichever came first. After left-truncation at the beginning of 1850, women and men contributed 491,422 and 511,713 person-years, respectively, to the analysis.

Our observations are clustered. One approach to adjust for a cluster effect is to introduce it as a covariate to the model. Thus, we added variables indicating province and period. But instead of adding birth year as a random or fixed effect, we modeled survival as a function of two cluster characteristics: height and childhood mortality. Unlike Wong and Mason (1985) we do not allow regression coefficients to vary between clusters. There is no theoretical justification for this. To the extent that we omitted important cluster characteristics, coefficients may be biased.

Early-life conditions changed dramatically across cohorts. Thus, to the extent that early-life conditions influence adult mortality, we expect to find cohort influences in adult mortality. 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. Figures 1 and 2 show that adult mortality started to decline in the 1870s. Hence, we imposed an equality constraint on the period categories in 1850-69. Period influences are captured by twenty five-year dummies indicating whether the current year is in the period 1870-74, 1875-79, 1880-

85, 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, or 1965-69 – 1850-69 being the reference category.

A second strategy is to transform one of the age, period or cohort variables so that its relationship is nonlinear. We chose to parameterize the effect of age as a cubic function (e.g. Raftery, Lewis, and Aghajanian 1995). While the use of a polynomial solves the problem of the arbitrary choice of the identifying constraint, this strategy 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 (e.g. Portrait et al. 2006; Preston and Wang 2006). Heckman and Robb (1985) term this the "proxy" variable approach because period and cohort are represented by some other variable. This study uses the "proxy" variable approach. We use two proxies for cohort-specific influences: survival until age five and height at age twenty. The "proxy" variable approach, however, also has its drawbacks. Replacing the cohort dummies by proxies may lessen the rigorousness of the control for the cohort-specific influences on period-specific influences. Because we only control for two proxies of cohort influences, we may overestimate the importance of period-specific influences (O'Brien 2000: 125).

The use of either or both of the first two strategies solves the under-identification problem (Yang 2008b: 211). After solving the under-identification

problem, however, multi-co-linearity may remain a problem (Collins 1982). The imposition of an equality constraint on the period categories in 1850-69 was retained, because it is used as the reference category and a reference category that is too small may lead to multi-co-linearity.

Results

Table 1 presents descriptive statistics of the variables used in the multivariate analysis. There are no missing values for year and place of birth. Figures 1 and 2 show the probability of twenty year survival (l_{x+20}/l_x) at ages 30, 50, and 70 in selected years for women and men, respectively. Tables 2 and 3 each present three models of the decline in adult mortality above age thirty for women and men, respectively. Coefficients are presented as odds ratios or exponents of the raw logistic coefficients. The odds ratios are multiplicative effects on the odds of dying 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.

[Tables 1 and 2 about here]

The first model in Table 2 only includes age, sex and period dummies (AP). The AP model shows that between 1850-69 and 1995-99 adult mortality among women above age thirty declined by about 45 percent in terms of odds ratios of the period dummies. About sixty percent of this decline (26 percentage points) occurred between 1850-69 and 1900-05. Adult mortality reached its lowest level in 1960-64 (see solid line in Fig. 4).

[Figure 4 about here]

The second model (APH) in Table 2 adds height at age twenty. Height has a significant effect on mortality after age thirty. As predicted, the relationship between height and adult mortality is negative. To illustrate the effect of increased height on the decline in adult mortality, Fig. 4 compares period effects in the second model (APH) with those in the first model (AP). To the extent that increased height explains the decline in adult mortality, mortality in terms of odds ratios of the period dummies should decline less after controlling for height. Figure 4 shows that mortality levels in terms of odds ratios in the APH model (dashed line) decline less than in the AP model (solid line), especially in the twentieth century. If height would not have changed, mortality levels in terms of odds ratios would have declined only slightly less until 1900-04, whereas in the twentieth century adult mortality would not have declined at all. Thus, increased height seems to have made a substantial contribution to adult mortality decline in the twentieth century.

We do not present a model with the probability of survival until age five, because early childhood survival does not have a significant effect in a model without height. After controlling for height, however, survival until age five does have a significant effect, as shown in the third model (APPROX) in Table 2. Moreover, the effect of early childhood survival is positive. In other words, low levels of early childhood mortality are associated with high levels of adult mortality.

Our measure of early childhood mortality seems to be correlated with height (see Fig. 3). Hence, we only compare the combined effect of height and survival until age five with the effect of height in the previous model (APH). Figure 4 shows that adding a measure of early childhood mortality to the model does not modify to any large extent our conclusion that early-life conditions explain most of the decline in the twentieth century.

Figure 4 also shows that mortality levels in terms of odds ratios in the APPROX model (dotted line) are lower than those in the APH model (dashed line). Thus, if not for the decline in early childhood mortality, adult mortality would have been *lower*. This suggests that early childhood mortality mainly affected adult mortality through selection and acquired immunity, rather than scarring.

After controlling for height and childhood mortality, there is no decline among men. This suggests that after 1950 mortality decline mostly occurred among men born after 1922 and, hence, that the decline is a cohort effect,

Conclusion and discussion

Mounting evidence suggests that early-life conditions have an enduring effect on an individual's mortality risks as an adult (e.g. Bengtsson and Mineau 2009). The contribution of improvements in early-life conditions to adult mortality *decline*, however, remains a much debated issue. The present study uses Dutch data to determine the relative importance of early-life conditions. Two major findings emerge from our analysis. First, in the second half of the nineteenth century early-life conditions played a marginal role, whereas in the twentieth century they dominate the decline. Second, among men increased height statistically explains most of the decline in the twentieth century, whereas among women it explains about forty percent of the decline.

The Dutch data indicate that in the second half of the nineteenth century period-specific influences were more powerful than cohort-specific ones, whereas in the twentieth century cohort-specific influences dominated the decline. We may have overestimated period-specific influences, however, because we only used two proxies for cohort-specific influences. Myrskylä (2010) found that period-specific influences

were more powerful than cohort-specific ones during adult mortality decline in the Netherlands. His analysis, however, ends in 1915. In general, part of the conflicting results reported in the literature may be the result of differences in time frames, cohort-specific influences being less powerful in earlier periods, as cohorts that experienced improved early childhood conditions were still too young to contribute to adult mortality decline. Thus, if we would have used the end of the nineteenth century as our cut-off point, then we also would have concluded that period-specific influences were more powerful than early-life conditions.

If early-life conditions would not have improved, our results seem to suggest that mortality would not have declined among adult men in the twentieth century and much less among adult women. Of course, this does not imply that there were no period-specific influences among men. In the twentieth century, however, period-specific influences do not seem to explain long-term trends in adult mortality. Besides the 1918-1919 influenza pandemic and World War II, two period-specific influences stand out.

First, after World War II adult mortality remained high instead of declining below pre-war levels. Apparently, unidentified period-specific influences caused adult mortality to remain relatively high after the war. Thus, one reason why improvements in early-life conditions dominated the twentieth-century adult mortality decline is that period-specific influences caused adult mortality to remain high after the war. We were unable to identity these period-specific influences. Higher mortality than expected after the war, however, has also been observed in other developed countries. Tapia Granados (2005) asserted that economic expansion in the US after World War II was associated with an *increase* in mortality. Thus, the use of the end of 1999 as

our cut-off point may have highlighted the role of early-life conditions, because the post-war economic expansion was associated with an increase in mortality.

Second, in spite of the Great Depression, mortality continued to decline in the early 1930s. A similar phenomenon has been observed in other developed countries. Tapia Granados and Diez Roux (2009), for example, have shown that mortality actually improved during the Great Depression of 1930-1933. If we would have used the end of 1939 as our cut-off point, then the improvement in early-life conditions would only have explained about half of the twentieth-century decline, because of the negative effect of the Great Depression on mortality.

The Dutch data indicate that increased height explains part of the adult mortality decline in the twentieth century before 2000. We used the heights of conscripts as a proxy for nutritional status. The hypothesis of McKeown (1976) that better nutrition has been the principal source of mortality decline is not consistent with low mortality during the Great Depression and elevated mortality during the economic expansion after World War II (Tapia Granados 2005; and Tapia Granados and Diez Roux 2009). However, McKeown's hypothesis is consistent with a strong effect of increased height on adult mortality decline.

Many previous studies only use one proxy for early-life conditions. The use of one proxy may explain why some concluded that improvements in early-life conditions were not important. If we would only have used early-childhood survival as a proxy for early-life conditions, then we would also have concluded that improvements in early-life conditions were not important. Only after controlling for height did we find a significant and negative effect of early childhood mortality on adult mortality. Most previous studies, however, report a *positive* effect of early childhood mortality on adult mortality (e.g. Bengtsson and Lindström 2000; Catalano

and Bruckner 2006; and Crimmins and Finch 2006). We can only speculate about reasons for the difference between the results of our study and those of previous studies. The use of an earlier time frame (1760-1894) by Bengtsson and Lindström (2000) may explain why their results differ from ours. The choice of age groups may provide another explanation for the different results. Catalano and Bruckner (2006) studied the effect of early childhood mortality on mortality after age five, whereas Crimmins and Finch (2006) modeled mortality in one age group (70-74) only. At least one other study, however, also reported a negative effect of early childhood mortality on adult mortality. Caselli and Capocaccia (1989) found that in Italy lower mortality early in life is associated with lower mortality up to age 45, but with *higher* mortality levels after age 45.

Notes

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

References

Allison, Paul D. 2010. *Survival Analysis Using the SAS® System: A Practical Guide*. 2nd ed. Cary, NC: SAS Institute Inc.

Almond, Douglas. 2006. "Is the 1918 Influenza Pandemic Over? Long-Term Effects of In Utero Influenza Exposure in the Post-1940 U.S. Population." *Journal of Political Economy* 114 (4): 672-712.

Barbi, Elisabetta, and James W. Vaupel. 2005. "Comment on "Inflammatory Exposure and Historical Changes in Human Life-Spans." *Science* 308: 1743a.

Barker, David J. P., and C. Osmond. 1986. "Infant Mortality, Childhood Nutrition, and Ischaemic Heart Disease in England and Wales." *Lancet* 1986 (i): 1077-1081.

Baten, Joerg. 2009. "Protein Supply and Nutritional Status in Nineteenth Century Bavaria, Prussia and France." *Economics and Human Biology* 7: 165–180.

Bengtsson, Tommy, and G. Broström. 2009. "Do Conditions in Early Life Affect Old-Age Mortality Directly and Indirectly? Evidence from 19th-Century Rural Sweden." *Social Science and Medicine* 68 (9): 1583-1590.

Bengtsson, Tommy, and Martin Lindström. 2000. "Childhood Misery and Disease in Later Life: The Effects on Mortality in Old age of Hazards Experienced in Early Life, Southern Sweden, 1760-1894." *Population Studies* 54: 263-277.

Bengtsson, Tommy, and Geraldine P. Mineau. 2009. "Early-Life Effects on Socio-Economic Performance and Mortality in Later Life: A Full Life-Course Approach Using Contemporary and Historical Sources." *Social Science and Medicine* 68 (9): 1561-1564.

Ben-Shlomo, Y., and Diana Kuh. 2002. "A Life Course Approach to Chronic Disease Epidemiology: Conceptual Models, Empirical Challenges and Interdisciplinary Perspectives." *International Journal of Epidemiology* 31: 285-293.

Ben-Shlomo, Y., and G. D. Smith. 1991. "Deprivation in Infancy or in Adult Life: Which is more Important for Mortality Risk?" *Lancet* 337: 530-534.

Brenes-Camacho, Gilbert, and Albert Palloni. 2011. "Malaria Survivors during Early Life, Health at Old Age, and Stroke Mortality in Costa Rica." Paper presented at the Annual Meeting of the Population Association of America, Washington, D.C.

Brinkman, H.-J., J. W. Drukker, and B. Slot. 1988. "Height and Income: A New Method for the Estimation of Historical Income Series." *Explorations in Economic History* 25: 227-264.

Case, Anne, Angela Fertig, and Christina Paxson. 2005. "The Lasting Impact of Childhood Health and Circumstance." *Journal of Health Economics* 24 (2): 365-389.

Caselli, Graziella, and Riccardo Capocaccia. 1989. "Age, Period, Cohort and Early Mortality: An Analysis of Adult Mortality in Italy." *Population Studies* 43 (1): 133-153.

Catalano, Ralph, and Tim Bruckner. 2006. "Child Mortality and Cohort Lifespan: A Test of Diminished Entelechy." *International Journal of Epidemiology* 35: 1264-1269.

Chassin, Laurie, Clark C. Presson, Jennifer S. Rose, and Steven J. Sherman. 1995. "The Natural History of Cigarette Smoking From Adolescence to Adulthood: Demographic Predictors of Continuity and Change." *Health Psychology* 15 (6): 478-84.

Cohen, Allan A., John Tillinghast, and Vladimir Canudas-Romo. 2010. "No Consistent Effects of Prenatal or Neonatal Exposure to Spanish Flu on Late-Life Mortality in 24 Developed Countries." *Demographic Research* 22 (20): 579-634.

Cole, T. J. 2003. "The Secular Trend in Human Physical Growth: A Biological View." *Economics and Human Biology* 1: 161-168.

Collins, James J. 1982. "The Contribution of Medical Measures to the Decline of Mortality from Respiratory Tuberculosis: An Age-Period-Cohort Model." *Demography* 19 (3): 409-427.

Costa, Dora. 2004. "The Measure of Man and Older Age Mortality: Evidence from the Gould Sample." *Journal of Economic History* 64 (1): 1-22.

Crimmins, Eileen M., and Caleb E. Finch. 2006. "Infection, Inflammation, Height, and Longevity." *Proceedings of the National Academy of Sciences* 103 (2): 498-503.

Cutler, David M., Grant Miller, and Douglas M. Norton. 2007. "Evidence on Early-Life Income and Late-Life Health from America's Dust Bowl Era." *Proceedings of the National Academy of Sciences* 104: 13244-13249.

Davey Smith, George, Carole Hart, Mark Upton, David Hole, Charles Gillis, Graham Watt, and Victor Hawthorne. 2000. "Height and Risk of Death among Men and Women: Aetiological Implications of Associations with Cardiorespiratory Disease and Cancer Mortality." *Journal of Epidemiology and Community Health* 54: 97-103.

de Beer, Hans. 2010. "Physical Stature and Biological Living Standards of Girls and Young Women in the Netherlands, Born between 1815 and 1865." *The History of the Family* 15: 60-75.

Drukker, J. W., and Vincent Tassenaar. 1997. "Paradoxes of Modernization and Material Well-Being in the Netherlands During the Nineteenth Century." Pp. 331-377 in *Health and Welfare during Industrialization*, edited by Richard H. Steckel and Roderick Floud. Chicago and London: The University of Chicago Press.

Elo, Irma T., and Samuel H. Preston. 1992. "Effects of Early-Life Conditions on Adult Mortality: A Review." *Population Index* 58 (2): 186-212.

Finch, Caleb E., and Eileen M. Crimmins. 2004. "Inflammatory Exposure and Historical Changes in Human Life-Spans." *Science* 305: 1736.

Finch, Caleb E., and Eileen M. Crimmins. 2005. "Response to Comment on "Inflammatory Exposure and Historical Changes in Human Life-Spans." *Science* 305: 1736.

Fogel, R. W. 1994. "Economic Growth, Population Theory, and Physiology: The Bearing of Long-Term Processes on the Making of Economic Policy." *American Economic Review* 84 (3): 369-395.

Gagnon, Alain, and Ryan Mazan. 2009. "Does Exposure to Infectious Diseases in Infancy Affect Old-Age Mortality? Evidence from a Pre-Industrial Population." *Social Science and Medicine* 68 (9): 1609-1616.

Hayward, Mark D., and Bridget K. Gorman. 2004. "The Long Arm of Childhood: The Influence of Early-Life Social Conditions on Men's Mortality." *Demography* 41 (1): 87-107.

Heckman, James, and Richard Robb. 1985. "Using Longitudinal Data to Estimate Age, Period, and Cohort Effects in Earnings Equations." Pp. 137-150 in *Cohort Analysis in Social Research*, edited by W. M. Mason and S. E. Fienberg. New York: Springer-Verlag,.

Hobcraft, John, Jane Menken, and Samuel Preston. 1982. "Age, Period, and Cohort Effects in Demography: A Review." *Population Index* 48 (1): 4-43.

Janssen, Fanny, and Anton E. Kunst. 2005. Cohort Patterns in Mortality Trends among the Elderly in Seven European Countries, 1950-99." *International Journal of Epidemiology* 34: 1149-1159.

Janssen, Fanny, Anton E. Kunst, and Johan P. Mackenbach. 2006. "Association between Gross Domestic Product throughout the Life Course and Old-Age Mortality across Birth Cohorts: Parallel Analyses of Seven European Countries, 1950-1999." *Social Science and Medicine* 63: 239-254.

Janssen-Heijnen, M. L. G., H. W. Nab, J. van Reek, L. H. van der Heijden, R. Schipper, and J. W. W. Coebergh. 1995. "Striking Changes in Smoking Behaviour and Lung Cancer Incidence by Histological Type in South-east Netherlands, 1960-1991." *European Journal of Cancer* 31A (6): 949-52.

Kannisto, Väinö, Kaare Christensen, and James W. Vaupel. 1997. "No Increased Mortality in Later Life for Cohorts Born during Famine." *American Journal of Epidemiology* 145 (11): 987-994.

Kermack, W. O., A. G. McKendrick, and P. L. McKinlay. 2001 (1934). "Death Rates in Great Britain and Sweden: Some Regularities and Their Significance." *International Journal of Epidemiology* 30: 687-683.

Knottnerus, Otto S. 2002. "Malaria Around the North Sea: A Survey." Pp. 339-353 in *Climatic Development and History of the North Atlantic Realm: Hanse Conference Report*, edited by Gerold Wefer, Wolfgang H. Berger, Karl-Ernst Behre, Eynstein Jansen. Berlin Heidelberg: Springer-Verlag.

Kok, Jan, Kees Mandemakers, and Hilde Bras. 2009. "Van Geboortebank tot Collaborator." *Tijdschrift voor Sociale en Economische Geschiedenis* 6 (4): 3-36.

Komlos, John. 1993. "The Secular Trend in the Biological Standard of Living in the United Kingdom, 1730–1860." *Economic History Review* 46 (1): 115–144.

Kuh, Diana, Rebecca Hardy, Claudia Langenberg, Marcus Richards, and Michael E. J. Wadsworth. 2002. "Mortality in Adults Aged 26-54 Years Related to Socioeconomic Conditions in Childhood and Adulthood: Post War Birth Cohort Study." *British Medical Journal* 325: 1076-1080.

Lynch, John W., George A. Kaplan, Richard D. Cohen, Jussi Kauhanen, Thomas W. Wilson, Nicholas L. Smith, and Jukka T. Salonen. 1994. "Childhood and

Adult Socioeconomic Status as Predictors of Mortality in Finland." *The Lancet* 343: 524-27.

Mandemakers, Kees, and J. L. van Zanden. 1990. "Lengte van Lotelingen en het National Inkomen: Schijnrelaties en Misvattingen." *Economisch- en Sociaal-historisch Jaarboek* 53: 1-23.

Mandemakers, Kees, and J. L. van Zanden. 1993. "The Height of Conscripts and National Income: Apparent Relations and Misconceptions." *Explorations in Economic History* 30: 81-97.

Mandemakers, Kees. 2000. "The Netherlands Historical Sample of the Netherlands." Pp. 149-177 in *Handbook of International Historical Microdata for Population Research*, edited by P. Kelly Hall, R. McCaa and G. Thorvaldsen. Minneapolis: Minnesota Population Center.

Mandemakers, Kees. 2001. "Historical Sample of the Netherlands HSN." *Historical Social Research* 26 (4): 179-190.

Mandemakers, Kees. 2002. "Building Life Course Datasets from Population Registers by the Historical Sample of the Netherlands (HSN)." *History and Computing* 14 (1+2): 87-107.

Mason, Karen O., W. M. Mason, H. H. Winsborough and W. Kenneth Poole. 1973. "Some Methodological Issues in Cohort Analysis of Archival Data." *American Sociological Review* 38 (2): 242-258.

McKeown T. 1976. *The Rise of Modern Population*. London: Edward Arnold.

Meza, R., B. Pourbohloul and R. C. Brunham. 2010. "Birth Cohort Patterns Suggest that Infant Survival Predicts Adult Mortality Rates." *Journal of Developmental Origins of Health and Disease* 1: 174-183.

Murphy, Michael. 2010. “Reexamining the Dominance of Birth Cohort Effects on Mortality.” *Population and Development Review* 36 (2): 365-390.

Myrskylä, Kimmo. 2010. “The Relative Effects of Shocks in Early- and Later-Life Conditions on Mortality.” *Population and Development Review* 36 (4): 803-829.

Navaneetham, K. 1993. “The Influence of Cohort Effects on Mortality Trends in India.” *Population Research and Policy Review* 12: 159-176.

O’Brien, Robert M. 2000. “Age Period Cohort Characteristic Models.” *Social Science Research* 29 (1): 123-139.

Portrait, France, Rob Alessie, and Dorly Deeg. 2010. “Do Early Life and Contemporaneous Macroconditions Explain Health at Older Ages? An Application to Functional Limitations of Dutch Older Individuals.” *Journal of Population Economics* 23: 617-642.

Power, Chris, Elina Hyppönen, and George Davey Smith. 2005. “Socioeconomic Position in Childhood and Early Adult Life and Risk of Mortality: A Prospective Study of the Mothers of the 1958 British Birth Cohort.” *American Journal of Public Health* 95 (8): 1396-1402.

Preston, Samuel H., Dana A. Glei, and John R. Wilmoth. 2010. “A New Method for Estimating Smoking-attributable Mortality in High-income Countries.” *International Journal of Epidemiology* 39: 430-38.

Preston, Samuel H., and Etienne van de Walle. 1978. “Urban French Mortality in the Nineteenth Century.” *Population Studies* 32 (2): 275-297.

Preston, Samuel H., and Haidong Wang. 2006. “Sex Mortality Differences in the United States: The Role of Cohort Smoking Patterns.” *Demography* 43 (4): 631-646.

Raftery, Adrian E., Steven M. Lewis, and Akbar Aghajanian. 1995. "Demand or Ideation? Evidence from the Iranian Marital Fertility Decline." *Demography* 32 (2): 159-82.

Ryder, N.B. 1965. "The Cohort as a Concept in the Study of Social Change." *American Sociological Review* 30: 843–61.

Schenk, Niels, and Frans van Poppel. 2011. "Social class, social mobility and mortality in the Netherlands, 1850–2004." *Explorations in Economic History* 48: 401–417.

Smith, Herbert L., William M. Mason, and Stephen E. Fienberg. 1982. "Estimable Functions of Age, Period, and Cohort Effects: More Chimeras of the Age-Period-Cohort Accounting Framework: Comment on Rodgers." *American Sociological Review* 47 (6): 787-793.

Su, Dejun. 2009. "Risk Exposure in Early Life and Mortality at Older Ages: Evidence from Union Army Veterans." *Population and Development Review* 35 (2): 275-295.

Tabeau, Ewa, F. Willekens and F. van Poppel. 1994. *Mortality in the Netherlands: The Data Base.* The Hague: Netherlands Interdisciplinary Demographic Institute.

Tapia Granados, Jose´ A. 2005. "Increasing Mortality during the Expansions of the US Economy, 1900-1996." *International Journal of Epidemiology* 34: 1194-1202.

Tapia Granados, José A., and Ana V. Diez Roux. 2009. "Life and Death during the Great Depression." *Proceedings of the National Academy of Sciences* 106 (41): 17290-17295.

van den Berg, Gerard J., Gabrielle Doblhammer, and Kaare Christensen. 2009. "Exogenous Determinants of Early-Life Conditions, and Mortality Later in Life." *Social Science and Medicine* 68 (9): 1591-1598.

van den Berg, Gerard J., Maarten Lindeboom, France Portrait. 2006. "Economic Conditions Early in Life and Individual Mortality." *American Economic Review* 96 (1): 290-302.

van der Hoff, N. M. 1979. "Cohort Analysis of Lung Cancer in the Netherlands. *International Journal of Epidemiology* 8 (1): 41-47.

van Poppel, Frans, and J. van Ginneken. 1985. "Reversal of Declining Mortality: The Case of the Netherlands in the Period 1950-80." *Maandbericht Gezondheid (Statistics Netherlands)* 85 (11): 5-19.

van Poppel, Frans, and Aart C. Liefbroer. 2005. "Living Conditions during Childhood and Survival in Later Life: Study Design and First Results." *Historical Social Research* 30 (3): 265-285.

Wen, M., and D. Gu. 2011. "The Effects of Childhood, Adult, and Community Socioeconomic Conditions on Health and Mortality among Older Adults in China." *Demography* 48 (1): 153-181.

Wolfe, Charles D. A., and Peter G. J. Burney. 1992. "Is Stroke Mortality on the Decline in England?" *American Journal of Epidemiology* 136 (5): 558-565.

Wolleswinkel-van den Bosch, Judith H., Frans W. A. van Poppel, Ewa Tabeau and Johan P. Mackenbach. 1998. "Mortality Decline in the Netherlands in the Period 1850-1992: A Turning Point Analysis." *Social Science and Medicine* 47 (4): 429-443.

Wong, George Y., and William M. Mason. 1985. “The Hierarchical Logistic Regression Model for Multilevel Analysis.” *Journal of the American Statistical Association* 80 (391): 513-24.

Yang, Yang. 2008a. “Trends in U.S. Adult Chronic Disease Mortality, 1960-1999: Age, Period, and Cohort Variations.” *Demography* 45 (2): 387-416.

Yang, Yang. 2008b. “Social Inequalities in Happiness in the United States, 1972 to 2004: An Age-period-cohort Analysis.” *American Sociological Review* 73 (2): 204-226.

Yang, Yang, and Kenneth C. Land. 2006. “A Mixed Models Approach to the Age-Period-Cohort Analysis of Repeated Cross-Section Surveys, with an Application to Data on Trends in Verbal Test Scores.” *Sociological Methodology* 36: 75-97.

Table 1. Descriptive statistics for variables in discrete-time hazard models.

Variable	Percentage	
	Women	Men
Period:		
1850-69 (ref.)	2.4	2.6
1870-74	0.8	0.9
1875-79	0.9	1.0
1880-84	1.0	1.2
1885-89	1.2	1.4
1890-94	1.6	1.7
1895-99	2.3	2.4
1900-04	3.2	3.1
1905-09	3.8	3.7
1910-14	4.5	4.3
1915-19	5.1	4.9
1920-24	5.8	5.4
1925-29	6.4	6.1
1930-34	7.0	6.8
1935-39	7.1	7.1
1940-44	7.0	7.0
1945-49	6.7	6.9
1950-54	6.4	6.7
1955-59	5.7	6.0
1960-64	4.9	5.3
1965-69	4.3	4.4
1970-74	3.5	3.6
1975-79	2.8	2.7
1980-84	2.2	2.0
1985-89	1.6	1.4
1990-94	1.1	0.9
1995-99	0.7	0.5
Place of birth:		
Friesland	10.9	10.2
Groningen	4.9	5.7
Drenthe	2.8	2.8
Overijssel	8.0	8.0
Gelderland	9.2	10.1
Utrecht (ref.)	7.3	7.2
North Holland	8.2	8.2
Amsterdam	6.7	6.9
South Holland	15.0	14.4
Rotterdam	5.6	5.0
Zeeland	6.4	6.7
North Brabant	10.1	9.6
Limburg	4.9	5.2
Person years	491,422	511,713

Note: descriptives for person years.

Table 2. Discrete-time hazard model of mortality at ages 30-89, the Netherlands 1850-1999: Women.

Model	**AP**		**APH**		**APPROX**	
Variable	e^b	***p*-value**	e^b	***p*-value**	e^b	***p*-value**
Age	1.218	.000	1.219	.000	1.218	.000
Age squared	0.996	.000	0.996	.000	0.996	.000
Age cubic	1.000	.000	1.000	.000	1.000	.000
Period:						
1850-69 (ref.)	1.000	-	1.000	-	1.000	-
1870-74	0.911	.617	0.905	.593	0.870	.532
1875-79	1.021	.903	1.028	.868	1.007	.967
1880-84	0.820	.235	0.842	.305	0.821	.239
1885-89	0.848	.278	0.889	.438	0.869	.357
1890-94	0.914	.520	0.973	.848	0.953	.733
1895-99	0.745	.029	0.804	.109	0.780	.070
1900-04	0.750	.025	0.816	.118	0.788	.068
1905-09	0.664	.001	0.723	.012	0.694	.005
1910-14	0.598	.000	0.661	.001	0.632	.000
1915-19	0.694	.002	0.783	.050	0.745	.020
1920-24	0.547	.000	0.632	.000	0.599	.000
1925-29	0.551	.000	0.653	.001	0.617	.000
1930-34	0.523	.000	0.637	.001	0.600	.000
1935-39	0.493	.000	0.617	.000	0.579	.000
1940-44	0.578	.000	0.746	.033	0.691	.008
1945-49	0.557	.000	0.741	.035	0.675	.007
1950-54	0.510	.000	0.698	.015	0.629	.002
1955-59	0.478	.000	0.672	.010	0.602	.001
1960-64	0.421	.000	0.612	.002	0.545	.000
1965-69	0.444	.000	0.667	.016	0.590	.002
1970-74	0.398	.000	0.615	.006	0.538	.001
1975-79	0.406	.000	0.648	.018	0.562	.002
1980-84	0.372	.000	0.614	.011	0.529	.001
1985-89	0.336	.000	0.578	.007	0.492	.001
1990-94	0.297	.000	0.536	.004	0.453	.000
1995-99	0.339	.000	0.638	.049	0.539	.008
Height in mm			0.994	.001	0.991	.000
Early childhood survival					3.447	.002
Place of birth:						
Friesland	0.915	.058	0.916	.064	0.916	.064
Groningen	0.742	.000	0.739	.000	0.739	.000
Drenthe	0.813	.004	0.808	.003	0.810	.003
Overijssel	0.969	.528	0.967	.505	0.970	.541
Gelderland	0.904	.044	0.901	.036	0.900	.035
Utrecht (ref.)	1.000	-	1.000	-	1.000	-
North Holland	0.892	.026	0.888	.020	0.891	.025
Amsterdam	0.882	.020	0.879	.017	0.881	.019
South Holland	0.844	.000	0.839	.000	0.843	.000
Rotterdam	0.999	.983	0.992	.888	0.995	.932
Zeeland	0.937	.223	0.935	.205	0.934	.201
North Brabant	0.873	.008	0.875	.006	0.877	.008
Limburg	0.882	.035	0.878	.030	0.882	.036
-2 Log likelihood	85871.386		85860.523		85850.492	

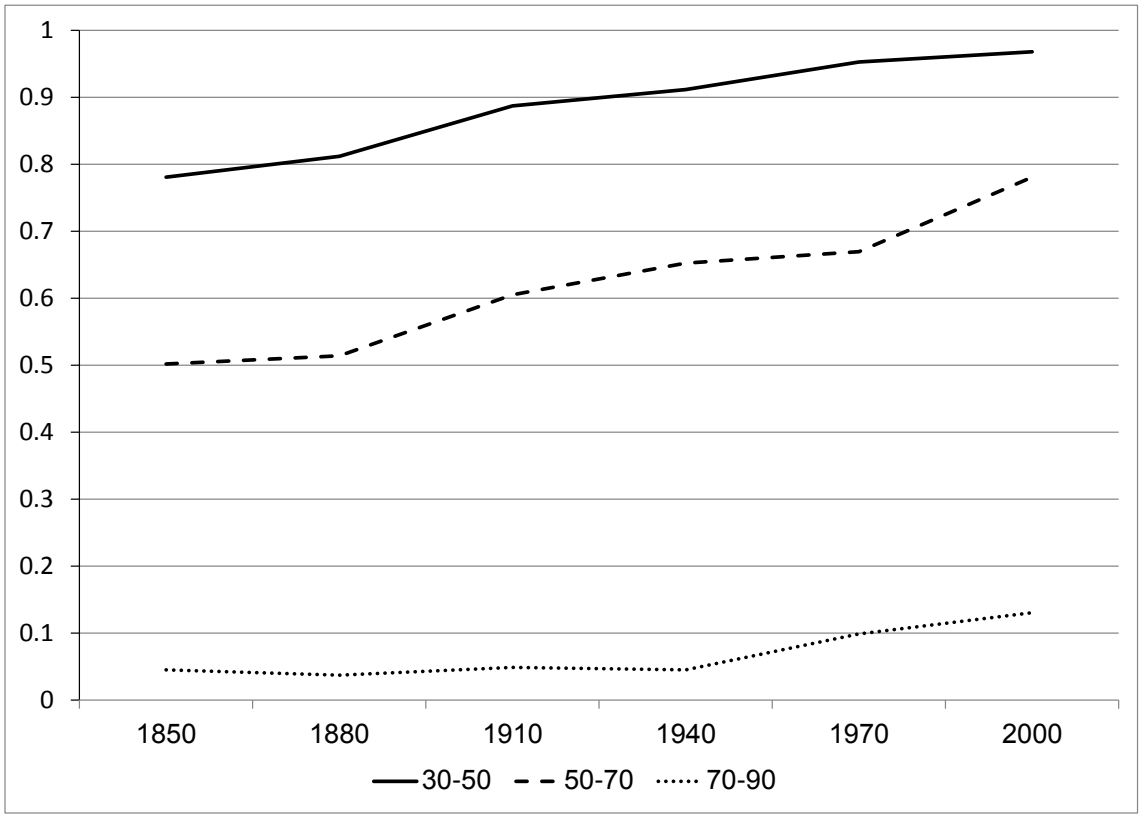
Table 3. Discrete-time hazard model of mortality at ages 30-89, the Netherlands 1850-1999: Men.

Model	**AP**		**APH**		**APPROX**	
Variable	e^b	***p*-value**	e^b	***p*-value**	e^b	***p*-value**
Age	1.161	.000	1.166	.000	1.166	.000
Age squared	0.998	.000	0.997	.000	0.997	.000
Age cubic	1.000	.000	1.000	.000	1.000	.000
Period:						
Period: 1850-69 (ref.)	1.000	-	1.000	-	1.000	-
1870-74	0.981	.916	0.981	.916	0.966	.846
1875-79	0.845	.334	0.855	.367	0.837	.307
1880-84	0.940	.690	0.964	.814	0.940	.690
1885-89	0.848	.273	0.883	.408	0.862	.324
1890-94	0.768	.065	0.811	.146	0.791	.105
1895-99	0.690	.006	0.740	.029	0.717	.016
1900-04	0.716	.010	0.774	.051	0.746	.026
1905-09	0.682	.002	0.739	.019	0.709	.008
1910-14	0.646	.000	0.704	.006	0.675	.002
1915-19	0.732	.009	0.812	.094	0.776	.043
1920-24	0.596	.000	0.675	.002	0.644	.001
1925-29	0.567	.000	0.658	.001	0.626	.000
1930-34	0.487	.000	0.579	.000	0.549	.000
1935-39	0.496	.000	0.604	.000	0.569	.000
1940-44	0.621	.000	0.777	.061	0.722	.017
1945-49	0.596	.000	0.767	.057	0.700	.012
1950-54	0.528	.000	0.697	.013	0.631	.002
1955-59	0.551	.000	0.747	.052	0.671	.009
1960-64	0.535	.000	0.745	.061	0.666	.011
1965-69	0.628	.000	0.900	.517	0.797	.172
1970-74	0.622	.000	0.915	.601	0.802	.204
1975-79	0.614	.000	0.930	.681	0.808	.240
1980-84	0.568	.000	0.888	.525	0.765	.160
1985-89	0.522	.000	0.847	.400	0.723	.108
1990-94	0.478	.000	0.807	.304	0.686	.077
1995-99	0.537	.000	0.937	.768	0.797	.311
Height in mm			0.995	.002	0.991	.001
Early childhood survival					4.282	.000
Place of birth:						
Friesland	0.933	.132	0.937	.154	0.935	.143
Groningen	0.758	.000	0.753	.000	0.756	.000
Drenthe	0.870	.042	0.865	.035	0.867	.037
Overijssel	0.995	.915	0.993	.884	0.993	.876
Gelderland	0.831	.000	0.826	.000	0.825	.000
Utrecht (ref.)	1.000	-	1.000	-	1.000	-
North Holland	0.807	.000	0.803	.000	0.805	.000
Amsterdam	0.904	.051	0.902	.045	0.904	.049
South Holland	0.856	.000	0.851	.000	0.852	.000
Rotterdam	0.988	.822	0.981	.734	0.984	.772
Zeeland	0.868	.006	0.866	.005	0.866	.005
North Brabant	0.884	.009	0.879	.007	0.883	.009
Limburg	0.835	.001	0.833	.001	0.834	.001
-2 Log likelihood	92210.981		92201.439		92186.657	

Figure 1. Probability of twenty-year survival (l_{x+20}/l_x) at ages 30, 50, and 70, for selected years, the Netherlands 1850-2000: Women.

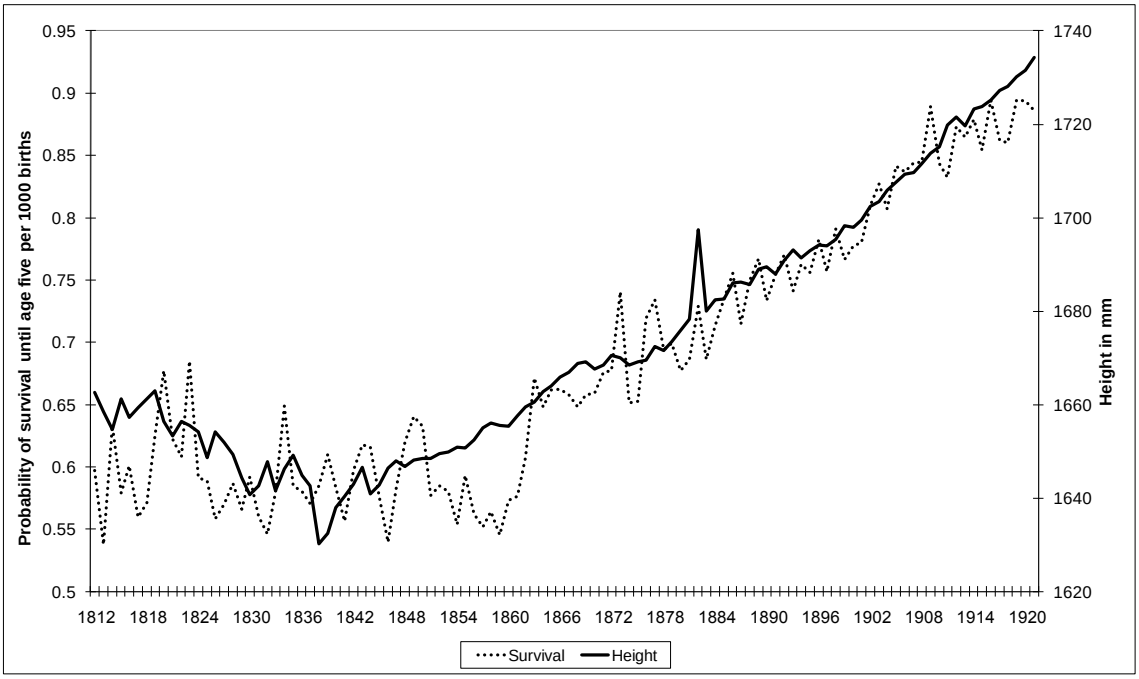
Source: Mortality database constructed by the Netherlands Interdisciplinary Demographi Institute (for a description see Tabeau et al. (1994)).

Figure 2. Probability of twenty-year survival (l_{x+20}/l_x) at ages 30, 50, and 70, for selected years, the Netherlands 1850-2000: Men.



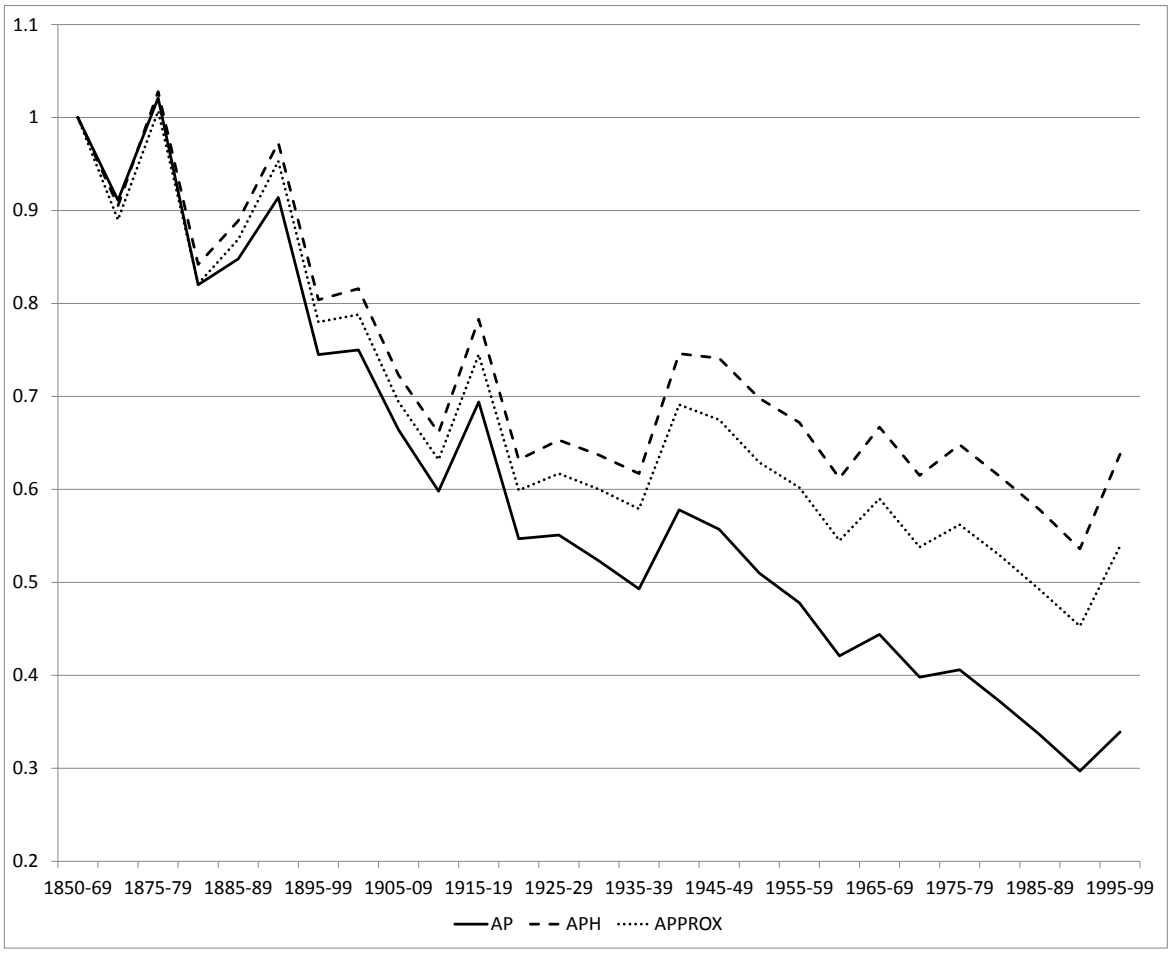
Source: Mortality database constructed by the Netherlands Interdisciplinary Demographi Institute (for a description see Tabeau et al. (1994)).

Figure 3. Probability of survival until age five and height of conscripts in mm by year of birth, the Netherlands 1812-1921.



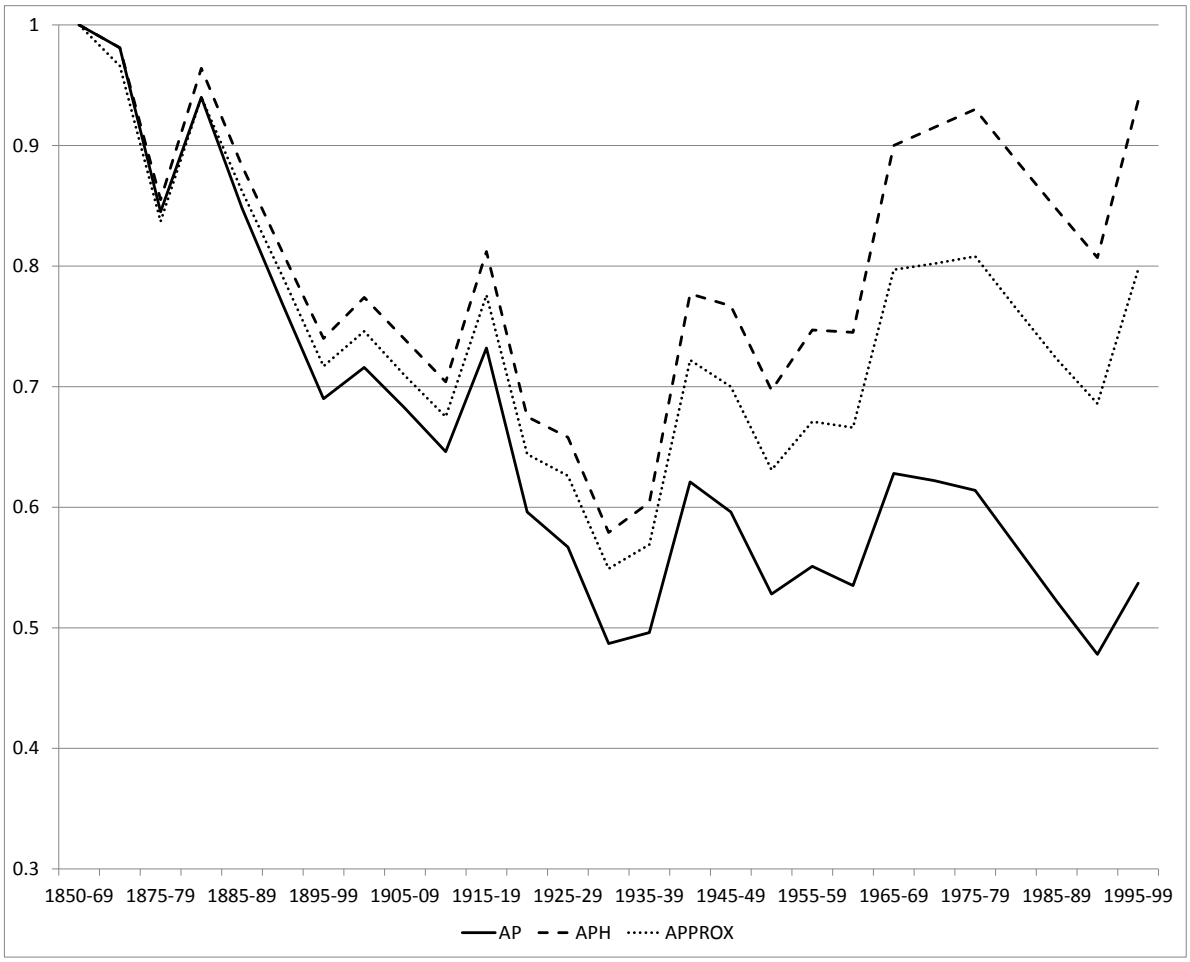
Source: HSN Data Set Life Courses Release 2011.01; Mandemakers and van Zanden (1993) and Drukker and Tassenaar (1997).

Figure 4. Period mortality trends in terms of odds ratios in three models, the Netherlands 1850-1999: Women.



Source: Table 2.

Figure 5. Period mortality trends in terms of odds ratios in three models, the Netherlands 1850-1999: Men.



Source: Table 3.