# How conditions in early life affect mortality by age and gender: Southern Sweden, 1830-1968

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**Abstract**

By using individual level data from the Scanian Demographic Database for 1830-1968, this work closes the gap between modern and historical analyses of the effects of early life conditions on mortality later in childhood and in adult life. Early life conditions are measured using local infant mortality rates, percentages of deaths due to airborne infectious disease, and food prices as indicators, in order to value the impact of the disease environment during infancy and of access to nutrition during the fetal stage. Our previous findings, that the disease load in the first year of life has an influence both on childhood and adult mortality, remains true also into the twentieth century. Furthermore, a passage from a dominance of a selection to a scarring effect across the life course are shown, as well as differences by gender and by birth cohort on the magnitude of these effects.

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**Introduction**

The importance of early life conditions for health in later life has been known for centuries and can be traced back at least to Francis Bacon, who in 1625 wrote "For strength of nature in youth, passeth over many excesses, which are owing a man till his age" (Bacon 1833: 117). It is therefore no surprise that scholars of demography and epidemiology studying in the 1920s and 1930s the long-term mortality decline also put emphasis on the significance of early life factors (Derrick 1927; Kermack *et al* 1934). Using data on age-specific mortality, they conclude that the drops in mortality in England, Wales, Scotland and Sweden were influenced by the year of birth of individuals rather than their year of death, therefore proposing the cohort or early life explanation. These theories, which relate to the long term causal mechanisms through which both risk and protective factors influence future health and disease, differ from the emphasis on period factors affecting all age groups during a particular moment in time, which later became dominant in explaining the long-term mortality decline (UN 1953). Cohort factors are, however, now gaining interest again, partly due to theoretical and methodological developments, partly by the availability of new data at individual level (Bengtsson and Mineau 2009; Lindström and Davey Smith 2007).

Recent studies using life course models and longitudinal individual level data have proven the large significance of early life factors for mortality at older ages, both overall and for specific diseases (Bengtsson and Lindström 2000; Galobardes et al 2004; Kuh and Ben-Shlomo 1997, 2004; Kuh and Davey Smith 2004; Lawlor *et al* 2004 ). Still several scholars using country level data have shown a greater importance of period effects over cohort factors when looking at the mortality decline (Barbi and Vaupel 2005; Kannisto 1994; Wilmoth 1988).

Analyses of early-life effects on later life health at individual level have almost exclusively been made on modern data, going back to the 1950s as most, which means that it only covers ages up to about 60 years. Some of the few historical studies using longitudinal individual level data do, however, analyse the early-life effects also on old age mortality taking a full life course approach (for an overview, see Bengtsson and Mineau 2009). While the early studies using data for southern Sweden cover the period up to around 1900 (Bengtsson and Lindström 2000; 2003), here we bridge the gap between historical and modern studies by analysing the period up to 1968. A major question is whether the strong influence of exposure to infectious diseases in the first year of life on adult mortality found

for the nineteenth century remains important into the twentieth century as mortality decline and mortality patterns change. In this study we also bring together analyses of different age-groups, children in ages 1-10 years, and adults in ages 50-70 years, in an effort to distinguish between selection and scarring effects of inverse conditions early in life. We also make separate analyses for females and males.

During the fetal stage and first years of life the development of organs and cells is fastest and individuals are therefore very sensitive to disturbances occurring during these periods. The biological pathways linking early life conditions and later life outcomes can work both through scarring and selection effects. Scarring (Preston et al 1998) is the permanent health damage than shows up later in life, while selection effects can be expressed through the survival of the fittest to periods of stress and through life-long immunity to particular diseases following infections suffered during earlier stages. The different hypotheses that have been put forward to try to explain the causal mechanisms argue that mortality in later life is affected by nutritional intake or by increased nutritional demand due to disease and that these effects are most important either during the *in utero* stage or the first year of life.

Barker and his colleagues (1994, 1995, 1997, 1998, 2001) have introduced the nutritional programming (or fetal origins) hypothesis. It states that the development of cardiovascular and other diseases later in life is a result of "disproportionate" retardation in growth caused by the lack of sufficient nutrition during the second and third trimester of pregnancy rather than the "proportionate" retardation that can occur in the first trimester. Inadequate nutrition leads to low birth weight, thus increasing the systolic blood pressure at adult ages and therefore the risk of heart disease later in life. However, while birth weight indeed has a significant effect on blood pressure, it is rather small (Huxley at al 2002, see also Christensen 2007:36). According to Fridlizius (1989), the role of infectious diseases during early life is crucial to the development of other diseases later in life. In particular, exposure to certain diseases early in life reduces immunity to and therefore also the risk of being infected by other diseases at older ages. This author retains that the linking factor is not nutrition but, rather, deranged immunological balance between specific infectious agents and the human host. Finch and Crimmins (2004) instead argue that the hypotheses of inflammatory infection and nutrition are not competing or contradictory, but that they are complementary.

Ben-Shlomo and Kuh (2002) proposed the accumulation of risks models, which in contrast to the simpler mono-causal critical period model and fetal origins hypothesis, affirms that risks accumulate over the life course, although some stages might have greater relevance. The negative impacts of conditions early in life can affect adult health *directly* through permanent damage of cells or *indirectly* through reduced abilities to accumulate wealth (Bengtsson and Mineau 2009). Indirect pathways have also been found both in modern and historical studies. Educational effects of conditions in early life have, for example been found (Palloni et al 2009), so have effects on adult socioeconomic status (Bengtsson and Broström 2009; Bengtsson, Broström and Lindström 2002). Critics of the Barker hypothesis in effect attest that birth weight is confounded with socioeconomic status because adverse social conditions during early life might lead individuals into life paths that influence their health negatively (Joseph and Kramer 1996; Kramer 2000). When trying to measure direct effects, there is therefore a need to utilize indicators that are not related to the life course (Doblhammer 2007). Macro level variables experienced by a cohort during early life can be used as "instruments" for individual conditions (Bengtsson and Lindström 2000, 2003; van den Berg et al 2006, 2009). Infant mortality rates and years of famine, in addition to business cyels, are amongst some of the instruments that have been adopted by different authors to measure exposure to nutrition or diseases during the fetal stage and infancy.

On the contrary to contemporary works using retrospective data, which thus present problems of selectivity and lack of accurateness, analyses based on historical sources allow the adoption of a prospective full life course approach when studying old age mortality (Bengtsson and Mineau 2009). Bengtsson and Lindström (2000, 2003) have observed the effects of early life conditions on old age mortality at the individual level for four parishes in Southern Sweden for 1766-1895. These authors found neither any influence of nutrition during the fetal stage and infancy nor of the disease load on mothers during pregnancy. Instead, they evidenced a strong impact from the disease load experienced during the year of birth on mortality later in life, and concluded that exposure to airborne infectious diseases throughout infancy increased old age mortality, in particular mortality due to infectious and chest diseases. An overall effect was seen both from short term cycles and from long term declines in infant mortality and threshold patterns were also viewed. A strong impact was observed during years with high infant mortality rates, dominated by smallpox and whooping cough, while modest changes in IMR had an almost nonexistent impact. When

also introducing a seasonal interaction they saw greater effects for individuals born during winter and summer. For the same parishes and period, Bengtsson and Broström (2009) found direct effects of early life conditions on old-age mortality as well as on social mobility, but that evidence of an indirect impact through attained socioeconomic status was lacking.

The present work uses data from the same parishes studied in Bengtsson and Broström (2009) and Bengtsson and Lindström (2000, 2003), together with a fifth one, and covers a more extensive period of time, up to 1968. The purpose is to investigate whether previous results concerning early life effects persists into the twentieth century despite changes in mortality levels and causes of death. By looking at mortality at different ages, this study will also try to distinguish periods in which selection or scarring effects dominate. Confronting results for different birth cohorts and by gender will, furthermore, allow to evidence whether the pattern of the effects observed varied over time and if it differed for various groups of individuals. Not much research has been done, in fact, for the twentieth century, which presents many changes from previous epochs, amongst other things because of increases in real wages, declines in fertility, a widening of socioeconomic differences (Bengtsson and Dribe 2010) and a general improvement in medical development. In modern times, the diseases patterns of both morbidity and mortality are dominated by chronic disease, for example coronary heart disease (Davey Smith and Lynch 2005), something rather different to the past. In this study we therefore try to attest the validity of the hypotheses that point to the influence on mortality of nutrition and exposure to disease during the fetal stage and infancy for different age groups, cohorts and gender. We use local infant mortality rates during the year of birth, the percentages of deaths due to airborne infectious diseases and consumer price indices from the year of conception as instruments to measure the effect of the disease load during infancy and of nutrition during the fetal stage.

**Data and methods**

The source material used for this work is the Scanian Demographic Database[2], which comprises births, deaths, marriages and migrations for five parishes in the southernmost part of Sweden in the region of Scania. This material covers the years 1813 to 1894 for the

[2] The data come from the Scanian Demographic Database, which is a collaborative project between the Regional Archives in Lund and the Centre for Economic Demography at Lund University.

parishes of Halmstad and Sireköpinge and 1813 to 1968 for the parishes of Hög, Kävlinge and Kågeröd. The data collection process has not been completed yet, and it is being extended to cover the 20th century for all five parishes. The quality of the parish register material is high and the gaps for births, deaths and marriages are limited. To the family reconstitutions, information concerning farm size, property rights and various data from poll tax records and land registers has been linked.

The sampled parishes are compact in their geographical location, showing the variations that could occur in a peasant society with regards to size, topography, and socio-economic conditions, and they offer a good, early source material. The entire area was open farmland, except the northern part, which was more wooded. The southern part became industrialized and urbanized in the last decades of the nineteenth century.

Occupational information from poll-tax registers and census information has also been employed. For married women with no registered occupation of their own, as well as for children and servants, the occupation of the household head is used. All occupations in the database were coded into HISCO (van Leeuwen, Maas and Miles 2002), and later five categories were created to represent the socioeconomic status of each family: higher occupations, skilled, farmers, lower skilled and unskilled.

The analysis will be conducted through the use of Cox proportional hazard models with time varying community variables. Proportionality tests have been performed on Schoenfeld residuals. Local infant mortality rates were constructed from the data and are used to measure the disease load during the first year of life of each cohort. These measures are complemented with further data that relate to the rural areas of Malmöhus County, where the parishes are located. In particular, based on information from official statistics, county level IMR were calculated, as well as the percentages of deaths due to airborne infectious diseases. Different source materials were used to obtain official statistics. For the years 1830-1859, the data was collected from Tabellverket, which was produced by the Tabular Commission (Tabellkommissionen) in Stockholm and was obtained from the Demographic Data Base in Umeå. Population statistics (Befolkningsstatistik) were used for the years 1860-1910, while for the years 1911-1918 the tables on death causes (Dödsorsaker) were consulted, both produced by Sweden´s Official Statistic Agency (SCB).

Information on consumer price indices for the entire country, obtained from Statistics Sweden, will furthermore be employed as a proxy of the access to nutrition. This last series

covers the years 1830 to 2009 and for this reason the analysis is at this stage restricted to individuals conceived on or after 1830.

The study will be divided into two different cohorts to view changes over time in the patterns of the results. To divide the data roughly in halves, but also to respect fluctuations in infant mortality rates and the type of disease environment, individuals born between 1830 and 1879 will be compared with those born between 1880 and 1918.

**Indicators of early life conditions**

The aim of this work is to analyse how the risk of dying at different stages of the life course is influenced by early life conditions, measured here by utilizing as instruments the disease environment during infancy as well as prices during the year of birth, used to capture the effect of nutrition during the fetal stage. The advantage of employing these types of indicators is that they are not confounded with variables that are directly related to the life course and which could influence the life paths undertaken by individuals. Through a series of graphs and models, the probability of dying for individuals born in years with a high disease load or high prices is compared to that of those born in other years.

The disease environment is measured using three different types of information: the infant mortality rate and the percentage of deaths due to airborne infectious diseases for Malmöhus County, where the studied parishes are located, as well as the infant mortality rates of the parishes themselves. The latter was calculated directly from the Scanian database, while the two former series were constructed form data obtained from official statistics.

The selection to employ both data of the studied parishes and of the entire County was based on the fact that the purpose of this work is not merely to measure years where mortality during infancy was high but, rather, to capture the effect of the disease environment during early life. The use of data for a larger geographical area allows to make inferences about the disease environment, something which would not be possible when only employing IMR of the studied parishes, especially in periods when mortality rates began to drop. We are not able to make conclusions in relation to the severity of diseases, but by using these three types of series we can pick up years when exposure rates were high.

The two series of infant mortality rates were detrended using the Hodrick Prescott filter with a filtering factor of 6.25, which is the value usually selected for yearly series. In order to signal years with high IMR, the proportion of the deviation over the trend was calculated for both series. Figures 1a shows the observed values and the estimated trend of infant mortality rates for the five parishes in Scania, Figure 1b the deviations from the trend.

The yearly percentages of deaths due to airborne infectious diseases were obtained by dividing the number of deaths due to these causes by the total number of deaths in Malmöhus County. These series are shown in Table 1. In years where the data allowed a distinction, only information relating to the rural areas of the county were selected, something which was possible for the period 1861-1918. Due to data restrictions, for 1830-1859, the entire county was observed and also for this period within the airborne infectious diseases category, only deaths due to measles, whooping cough and smallpox were considered. For the year 1860 no official statistics which distinguished deaths by cause were found. Furthermore, for most of the studied period death data regards the entire population, except in those years where official statistics allowed distinctions by age. It was possible to calculate the percentage of deaths due to airborne infectious diseases for children aged 0-1 for the years 1830 to 1859, and for children aged 0-10 for the years 1881 to 1890, whereas for other years the population as a whole was taken into account.

Departing from these three types of data, years with a high disease load were defined in two stages. The values of each series were first ordered and percentiles were calculated, thus allowing to signal years with a high deviation over trend in IMR either in the studied parishes or Malmöhus County or high percentages of airborne infectious diseases. A $90^{th}$ percentile threshold was considered for the two series of IMR, which corresponded to a value of 0.15 for the proportion of deviation over the trend of the entire county and 0.44 for this same proportion in the studied parishes (Figure 1b). A $75^{th}$ percentile threshold was, instead, used for the series of airborne infectious diseases, calculating these percentiles separately for different periods, since, as stated earlier, values did not always consider the same age groups. Using these thresholds, years where the percentage of airborne infectious diseases deaths exceeded 7% for 1839-1859, 25% for 1881 to 1890 and 14% for other periods were considered as years with a high disease load. Since the types of diseases included in the official statistics within the airborne infectious diseases category changed starting from

1911, the percentile calculations were done separately for 1911-1918, where the 75$^{th}$ percentile corresponded to 15%.

In the above described first stage, years with a high disease load were signalled by taking into account each of the three indicators separately. During a second stage, corrections were made by considering combinations of these pieces of information. Years with values that exceeded one of the thresholds described above but for which the deviation around the trend of IMR was negative, either for the studied parishes or the entire county, were not considered as years with a high disease load, with six exceptions: 1835, 1862, 1881, 1886, 1894 and 1918. The reasons for these exceptions were that in 1835, 1881, 1886 and 1894 the proportion of deviation over trend if IMR was above 0.5 in the studied parishes, while in 1862 and 1918 the percentages of deaths due to airborne infectious diseases was very high.

Through these two stages, 17 years were considered as having a high disease load: 1831, 1832, 1835, 1838, 1846, 1853, 1859, 1862, 1869, 1874, 1878, 1881, 1886, 1890, 1894, 1916 and 1918 (Table 1)[3].

Another type of indicator used in this study to measure early life conditions are prices during the year of conception, which serve as a proxy for the level of nutrition during the fetal stage. The year of conception corresponded to the year of the date of birth minus nine months. In Figure 2a the consumer price indices, expressed in a logarithmic scale, for Sweden from 1830 to 1968 are shown together with the respective trend. Deviations from the trend are shown in Figure 2b. A threshold level of 0.03 was used to consider years with high prices. This threshold level was chosen rather arbitrarily, following graphical evidence.

**Early life conditions and the risk of death during childhood**

This section evaluates the influence of early life conditions on childhood mortality. In particular, we observe the impact of nutrition during the fetal stage and the disease load during the first year of life on the risk of dying between ages 1 and 10. The first estimations evidence that within these age groups there is a dominance of a selection effect due to the disease load experienced in the first year of life. The proportional hazards assumption is, however, violated, which evidences that the differentials in the risks of dying are not constant over age, something also supported by graphical evidence.

[3] Sensitivity analyses will be conducted in the future to test the impact of these choices.

Figures 3a-d display hazard curves, by gender and cohort, which compare the risk of dying between ages 1 and 10 for children born in years with a high disease load relative to those born in other years. Different age patterns can be seen for boys and girls for the two cohorts, which suggest separate analyses for these groups.

The results of Cox proportional hazard estimates are found on Table 2. These models include covariates that measure the impact of early life conditions, but also control for socioeconomic status and year and parish of birth. We can first concentrate on the estimations for children born between 1830 and 1879. For girls (Model A), when first looking at the hazard ratio for the effect of the disease environment during the first year of life, the overall pattern is a dominance of a scarring effect, which, however, lacks statistical significance. Different interactions with age were introduced, pointing to a dominance of a scarring effect during an initial short period, but also lacking statistical significance. The impact of nutrition during the fetal stage shows a dominance of a scarring effect, which also lacks statistical significance. From these estimates it is therefore possible to conclude that early life conditions do not influence significantly the risk of dying between ages 1 to 10 for girls born between 1830 and 1879.

Boys display a somewhat different pattern, with a dominance of a scarring effect between ages 1 and 10 in relation to the disease environment during the first year of life. Those born in years with a high disease load have, in fact, a 17% lower risk of dying than boys born in other years (results not shown here). Figure 3b as well as tests on Schoenfeld residuals suggest the introduction of age interactions due to the violation of the proportional hazards assumption. In Model B an interaction between a dummy variable measuring the disease load during infancy and whether the boy was older than age 2.9 was introduced. This age division was selected based on survival, hazard and cumulative hazard curves as well as on different model implementations. Boys of cohorts 1830 to 1879 born in years with a high disease load have a 31% lower risk of dying between ages 1 and 2.9 and a 3% lower risk of dying between ages 2.9 and 10. A dominance of a selection effect is therefore maintained throughout childhood for the impact of the disease environment during the year of birth, and the strongest effects are displayed within the first couple of years after infancy. The hazard ratio for prices points to a very small but not statistically significant prevalence of a scarring effect.

Table 2 also shows the results for children born between 1880 and 1918. A dominance of a scarring effect as an impact to the disease environment experienced during infancy is now displayed for girls, where those born in more difficult years have a 15% lower risk of dying between ages 1 and 10 than those born in other years (results not shown here). By observing Figure 3c as well as cumulative hazard and survival curves, two interactions with age were introduced: 2.3 and 6.8. As can be seen in Model C, girls born in years with a high disease load show a 63% lower risk of dying between ages 1 and 2.3, a 33% higher risk of dying between ages 2.3 and 6.8 and a 46% lower risk of dying between ages 6.8 and 10. A very small but not statistically significant dominance of a scarring effect is displayed in relation to the impact of nutrition during the fetal stage.

For these same cohorts, boys born during years with a high disease load have a 31% lower risk of dying between ages 1 and 10 than those born in other years. Strong variations are seen, however, across ages in Figure 3d, reason for which age interactions were introduced. In Model D boys born during years with a high disease load have a 65% lower risk of dying between ages 1 and 2.3, a 38% higher risk between ages 2.3 and 4.5 and a 48% lower risk after age 4.5 than those born in other years. A small but not statistically significant dominance of a scarring effect is displayed in relation to the impact of nutrition during the fetal stage.

The results of this section evidence that, with the exception of girls born between 1830 and 1879, a dominance of a selection effect is displayed for children born in years with a high disease load. This effect is strongest in the initial stages of childhood, in particular below age 2.9 for boys born in 1830-79 and below 2.3 for boys and girls born in 1880-1918. If high mortality rates are encountered during the first year of life, only the healthiest and strongest children survive infancy. In years where the disease load is not so elevated, weak children survive infancy, but die shortly after. After these ages the patterns are alternating, but evidence further periods with a dominance of a selection effect. No significant effects of nutrition during the fetal stage were observed for children aged 1-10.

**Early life conditions and the risk of death during old age**

Having seen in the previous section that early life conditions have a significant effect in the risk of dying during childhood, their influence on old age mortality, in particular from ages 50

to 70, can now be analyzed. This will allow to attest whether a scarring effect prevailed or if a selection effect is still dominant and whether the hypotheses on the influence of nutrition or disease during early life can be corroborated for old age mortality for females, males and for different birth cohorts.

We begin by taking a look at hazard curves, shown in Figure 4a-d, which compare the risk of dying between ages 50 to 70 for individuals born in years with a high disease load relative to those born in other years. For cohorts 1830-1879 fluctuations in these curves can be observed both for males and females, pointing to the fact that the differentials in the risk of dying for the two groups of individuals were not constant over age. For this reason interactions with age were introduced, at 60.5 for females and 63 for males. These choices were based both on Figure 4a-b but also on cumulative hazard curves (not shown here). The patterns are more constant for males and females born between 1880 and 1918, who clearly evidence stronger risks of dying if they were born in years with a high disease load.

Cox proportional hazard models, by gender and cohort, that measure the probability of death between ages 50 to 70 are shown in Table 3. Females born in years with a high disease load between 1830 and 1879 have a 29% higher risk of dying between ages 50 to 60.5 and a 5% lower risk between ages 60.5, although neither the base nor the interaction effect were statistically significant. Males of the same cohorts show, instead, a 51% higher risk of dying between ages 50 and 63 and a 17% lower risk between ages 63 and 70 if they were born in years with a high disease load, and both the base and interaction terms are statistically significant. When observing the results for cohorts 1880-1918, statistically significant results are found for both groups. For females, being born in a year with a high disease load increased the hazard of dying between ages 50 to 70 by 86%, while for males by 50%. Significant effects of nutrition during the fetal stage were not seen in any of these models.

The results obtained in this section indicate that the exposure to disease during the first year of life has an influence on old age mortality. For cohorts 1830-1879 no significant effects were observed for females, while a dominance of a scarring effect was seen for males born in years with a high disease load up to age 63. Significant effects were found for both gender groups born after 1880, with a stronger magnitude for females. Possible reasons behind these differences by cohort and gender could be either a result of dissimilarities in the exposure to disease during infancy or on the type of pathologies from which these individuals are dying.

Taking the exposure side into consideration first, Table 1 evidences the most diffused cause of death each year. For the period 1830 to 1879, years with a high disease load show 2 peaks in measles, 2 in scarlet fever, 2 in smallpox and 3 in whooping cough. For the years 1880-1918, 3 peaks in diphtheria are observed, as well as 1 in influenza, 1 in scarlet fever and 1 in tuberculosis. Changes in the type of disease environment experienced during infancy are therefore perceived.

Causes of death by gender and birth cohort can also be confronted. When first calculating the percentage of death for each cause for individuals aged 50-70 (Table 4), cohorts 1880-1918 show a lower percentage of deaths due to airborne infectious diseases and higher proportions due to cardiovascular diseases & diabetes and cancers. This descriptive analysis can also be extended to view possible differences by gender and also by whether individuals were born in years with a high disease load. As can be observed in Table 5, for those born in 1830-1879, the percentage of deaths due to cardiovascular diseases & diabetes exceeds that of cancers for all groups, except for males born in years with a high disease load, where the proportions are roughly the same for these two groups of causes of death. If we, instead, concentrate on individuals born between 1880 and 1918, it can be seen that the percentage of deaths due to cardiovascular diseases & diabetes exceeds that of cancers for men born both in years with a high disease load and in other years. For females, this is true only for those born in years with a high disease load, whereas for those born in other years the percentage of deaths due to cancer is greater.

In this section significant effects of early life conditions on old age mortality have been shown. The results confirm the hypothesis that exposure to disease during infancy influences mortality later in life, but no clear evidence of an impact of nutrition during the fetal stage has been found. Gender and cohort differences in the patterns of the results were viewed, which could in part be explained, as shown by descriptive analyses, from dissimilarities in the disease environment during infancy but also in the types of diseases that caused the death of these individuals. Although more in depth studies on these issues are required and will be conducted in the future, these results could indicate not only differences in the types of diseases to which individuals of different cohorts were exposed to, but perhaps also diverse causal mechanisms linking early life conditions to later life outcomes for men and women.

**Early life conditions and the change between the predominance of selection and scarring across the life course**

The previous sections have analysed how the disease environment during the year of birth affects mortality during childhood and old age. A dominance of a selection effect was observed between ages 1 and 10 while from 50 to 70 a scarring effect prevailed. These patterns, although with varying magnitudes, were common across gender and cohort, with the exception of females born between 1830 and 1879, for which the disease environment did not impose significant effects on the risk of dying, neither during childhood nor old age. No significant effects were found, for both genders and cohorts, in relation to nutrition during the fetal stage.

We have now observed two different moments that point to fluctuations in the type of effects that the disease environment experienced during the year of birth impose on individuals, with a passage from a dominance of selection to a dominance of scarring. We can therefore question at which particular moment within the life course is this change observed. Furthermore, keeping in mind the differences seen across gender and cohort, it is also important to attest whether variations in these patterns also occur for different groups of people.

The effects that the disease environment experienced during the year of birth imposes on the entire life course of individuals, can be observed through cumulative hazard curves, shown in Figure 5a-d for ages 1 to 70. Through a simple glance at these graphs, striking differences by cohort and gender are seen in the points of fluctuation between a dominance of selection and scarring effects. In all cases these changes occur on or after age 30, reason for which hazard and cumulative hazard curves for ages 30 to 70 are shown in Figure 6a-h.

We begin by taking a closer look at females born between 1830 and 1879. Earlier sections had shown no significant effects, neither during childhood nor old age, and this is also observed at other stages of the life course in Figure 5a as well as in different Cox proportional hazard regressions (results not shown here).

For this same cohort, the pattern for males was rather diverse. Figure 5b evidences that a scarring effect starts to dominate more or less at around age 45. When studying ages 45 to 70, a 34% higher risk of dying is observed for males born in years with a high disease load relative to those born in other years. This result is statistically significant, although the

proportional hazards assumption is violated, reason for which an interaction with age is introduced, based on Figure 5b but also in consistency with earlier sections, at age 63. Table 6 shows that the risk of dying for individuals born in years with a high disease load relative to those born in other years is 66% higher between ages 45 and 63 and 18% lower between ages 63 and 70. Between ages 10 and 45 (model not shown here), we observe a 14% higher risk of dying for males born in years with a high disease load, although this result is not statistically significant. Summarizing the effects of the disease environment during the year of birth on males born between 1830 and 1879, we observed a dominance of a selection effect that was strong between ages 1 and 2.9 and weaker between ages 2.9 and 10, and a dominance of a scarring effect where the magnitude and statistical significance was weak between ages 10 and 45 and very strong between ages 45 and 63. From ages 63 to 70 we found a weak and not statistically significant prevalence of a selection effect.

Females born between 1880 and 1918 show patterns which are different than for those born in previous years. In Figure 5c it can be seen that a scarring effect starts to dominate from around age 30. This result is confirmed in Model A in Table 7, where it can be seen that between ages 30 to 70 the risk of dying for females born in years with a high disease load is 57% higher than those born in other years, where also the proportional hazards assumption is met. Between ages 10 and 30 (Model B in Table 7), the risk of dying is 23% higher for women born in years with a high disease load, although this result is not statistically significant. What is most striking to note in this case is the fact that, for the first time, a significant effect of nutrition during the fetal stage is evidenced. Women conceived in years with high prices have, between ages 10 and 30, a 96% higher risk of dying than those conceived in other years[4]. Other age groups had not shown significant effects of nutrition during gestation. Summarizing the effects of the disease environment during the year of birth on females born between 1880 and 1918, we observed a strong dominance of a selection effect between ages 1 and 2.3, a higher scarring effect between ages 2.3 and 6.8, and again a dominance of selection between 6.8 and 10. We later find a prevalence of a scarring effect where the magnitude and statistical significance was weak between ages 10 and 30 and very strong between ages 30 and 70.

[4] Taking into consideration Figure 2, the same model was run only for individuals conceived before 1918. The pattern of the results remained constant, with a 90% higher risk of dying for females conceived in years with high prices.

Turning back to Figure 5d, for males born between 1880 and 1918 a dominance of a scarring effect starting from around age 57 can be seen. This is confirmed in model A of Table 8, where men born in years with a high disease load evidence a 74% higher risk of dying than those born in other years[5]. Between ages 10 and 57, model B shows a 20% lower risk of dying for men born in years with a high disease load, although these results lack statistical significance. Statistical significance (at the 10% level) is, however, once again observed in relation to nutrition during the fetal stage. Men conceived in years with high prices have a 43% higher risk of dying between ages 10 and 57 than those born in other years. Summarizing the effects of the disease environment during the year of birth on males born between 1880 and 1918, we observed a strong dominance of a selection effect between ages 1 and 2.3, a higher scarring effect between ages 2.3 and 4.5, again a prevalence of selection between ages 4.5 and 10 and also between ages 10 and 57. We later find a dominance of a scarring effect between ages 57 and 70.

To analyse more closely the effect of prices, in Figure 7a-d the hazard of dying between ages 1 and 70 is shown for males and females born between 1880 and 1918. As can be seen in the hazard and cumulative hazard curves, the largest differences in the risks of dying between individuals conceived in years with high prices and those conceived in other years are evidenced in ages 10 to 20. The fact that we find significant results for this second cohort shows that the effect of nutrition during the fetal stage starts to become important, especially amongst teenagers and young adults, in years where the disease environment experienced in infancy was rather different than in the past and where also infant mortality rates were much lower than in previous periods.

[5] Similar results are obtained when restricting this model to males conceived before 1918, who display, between ages 10 and 57, a 47% higher risk of dying if conceived in years with high prices, and this result is statistically significant at the 5% level. To compare these values with those of females, estimations were made for ages 10 to 30 where males conceived before 1918 and in years with high prices showed a 130% higher risk of dying than those conceived in other years.

**Conclusions**

This work had the aim of analysing the influence of early life conditions on mortality by age, gender and birth cohort for Southern Sweden in 1830-1968. When studying the impact of the disease environment during the year of birth, a selection effect was found to dominate during earlier stages of childhood, whereas later in life a scarring effect prevailed, something observed for males of all cohorts and for females born between 1880 and 1918. The timing of the passage between the dominance of a selection to a prevalence of a scarring effect differed by gender and cohort. For males, the change to a strong and significant dominance of a scarring effect occurred more or less at around age 45 for those born between 1830 and 1879 and at around age 57 for those born between 1880 and 1919. For women, this passage took place at around age 30 for those born after 1880, while no significant effects of the disease environment during early life were perceived on mortality for those born before this year. An effect of prices during the year of conception on the risk of dying was found for individuals born in the second cohort (1880-1918), for females between ages 10 and 30 and males between ages 10 and 57.

In this study we therefore find evidence of the importance of the disease environment during infancy for mortality in later life. The hypothesis that indicates the importance of nutrition during the fetal stage was only proven for middle ages. Exposure to disease during infancy showed a passage from a selection effect during childhood to a scarring effect during old age, but also gender and cohort differences both on the magnitude of these effects as well as on the timing of the prevalence of each of them.

This work has provided several results which, we believe, are new to the literature. On the one hand, the use of extended datasets allowed to close the gap between historical and contemporary studies, evidencing that influential effects of the disease environment experienced in infancy were found for mortality later in life both in the 19$^{th}$ and 20$^{th}$ century. Furthermore, by analysing mortality of all age groups, we were not only able to show at which points in life a passage from a dominance of selection to a prevalence of scarring occurred, but also how these changes differed by gender and cohort. Successive stages of this project will try to compare the effects of different types of disease environments experienced during infancy on mortality, both overall and by various causes of death.

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**Table 1: IMR, percentage of deaths due to airborne infectious diseases, main death cause and choice of years with a high disease load**

| Year | IMR parishes | Deviation/Trend parishes | IMR county | Deviation/Trend county | % airbone inf. dis. deaths <1 | % airbone inf. dis. deaths <10 | % airbone inf. dis. deaths all ages | Main death cause county | High disease load year |
|---|---|---|---|---|---|---|---|---|---|
| 1830 | 196.721 | -0.031 | 170.596 | -0.094 | 4.40% | | | measles | 0 |
| 1831 | 300.699 | 0.436 | 209.890 | 0.155 | 11.60% | | | whooping cough | 1 |
| 1832 | 245.902 | 0.209 | 177.997 | 0.034 | 8.00% | | | whooping cough | 1 |
| 1833 | 100.671 | -0.473 | 157.860 | -0.023 | 6.41% | | | | 0 |
| 1834 | 135.338 | -0.267 | 134.926 | -0.114 | 2.24% | | | | 0 |
| 1835 | 288.889 | 0.576 | 144.104 | -0.017 | 4.52% | | | whooping cough | 1 |
| 1836 | 124.183 | -0.299 | 138.529 | -0.036 | 12.14% | | | measles | 0 |
| 1837 | 142.857 | -0.176 | 148.545 | 0.044 | 4.07% | | | smallpox | 0 |
| 1838 | 232.759 | 0.359 | 160.852 | 0.145 | 11.73% | | | smallpox | 1 |
| 1839 | 154.546 | -0.061 | 127.894 | -0.067 | 7.20% | | | | 0 |
| 1840 | 134.921 | -0.143 | 124.756 | -0.073 | 3.13% | | | whooping cough | 0 |
| 1841 | 159.420 | 0.048 | 128.070 | -0.042 | 2.11% | | | | 0 |
| 1842 | 172.662 | 0.174 | 150.653 | 0.127 | 4.34% | | | | 0 |
| 1843 | 117.241 | -0.175 | 133.112 | 0.003 | 3.40% | | | | 0 |
| 1844 | 134.228 | -0.050 | 119.111 | -0.097 | 0.64% | | | | 0 |
| 1845 | 99.338 | -0.313 | 121.731 | -0.080 | 1.04% | | | | 0 |
| 1846 | 211.539 | 0.404 | 152.665 | 0.149 | 9.46% | | | measles | 1 |
| 1847 | 175.325 | 0.160 | 150.496 | 0.148 | 6.77% | | | measles | 0 |
| 1848 | 114.650 | -0.222 | 109.364 | -0.141 | 1.11% | | | | 0 |
| 1849 | 146.497 | 0.013 | 114.056 | -0.089 | 1.84% | | | | 0 |
| 1850 | 138.122 | -0.032 | 121.692 | -0.032 | 4.56% | | | smallpox | 0 |
| 1851 | 139.665 | -0.016 | 127.301 | -0.002 | 2.89% | | | smallpox | 0 |
| 1852 | 100.592 | -0.290 | 132.147 | 0.024 | 2.25% | | | | 0 |
| 1853 | 229.299 | 0.625 | 162.523 | 0.263 | 8.29% | | | smallpox | 1 |
| 1854 | 106.509 | -0.199 | 103.795 | -0.170 | 0.22% | | | | 0 |
| 1855 | 130.952 | 0.059 | 116.975 | -0.044 | 2.92% | | | measles | 0 |
| 1856 | 101.266 | -0.126 | 114.022 | -0.062 | 1.53% | | | | 0 |
| 1857 | 70.968 | -0.373 | 116.448 | -0.048 | 1.71% | | | | 0 |
| 1858 | 97.938 | -0.161 | 128.473 | 0.039 | 1.52% | | | | 0 |
| 1859 | 192.090 | 0.583 | 154.133 | 0.252 | 4.99% | | | | 1 |
| 1860 | 154.762 | 0.309 | | | | | | | 0 |
| 1861 | 52.632 | -0.523 | 104.538 | -0.123 | | | 11.25% | scarlet fever | 0 |
| 1862 | 104.938 | -0.014 | 113.407 | -0.019 | | | 17.57% | measles | 1 |
| 1863 | 100.592 | -0.050 | 98.794 | -0.129 | | | 7.56% | scarlet fever | 0 |
| 1864 | 100.000 | -0.073 | 123.107 | 0.085 | | | 5.84% | scarlet fever | 0 |
| 1865 | 131.148 | 0.183 | 116.272 | 0.019 | | | 4.28% | typhoid/cerebral fever | 0 |
| 1866 | 115.607 | 0.034 | 103.794 | -0.102 | | | 5.67% | typhoid/cerebral fever | 0 |
| 1867 | 115.108 | 0.036 | 106.912 | -0.095 | | | 5.36% | typhoid/cerebral fever | 0 |
| 1868 | 73.826 | -0.328 | 145.757 | 0.214 | | | 14.68% | scarlet fever | 0 |
| 1869 | 148.760 | 0.360 | 136.862 | 0.156 | | | 16.05% | scarlet fever | 1 |
| 1870 | 118.881 | 0.124 | 103.163 | -0.092 | | | 7.50% | scarlet fever | 0 |
| 1871 | 60.811 | -0.398 | 86.573 | -0.211 | | | 2.86% | croup | 0 |
| 1872 | 111.111 | 0.117 | | | | | 3.96% | typhoid/cerebral fever | 0 |
| 1873 | 76.923 | -0.222 | | | | | 5.38% | cholera | 0 |
| 1874 | 151.515 | 0.531 | | | | | 10.03% | whooping cough | 1 |
| 1875 | 67.485 | -0.296 | | | | | 8.22% | croup | 0 |
| 1876 | 101.351 | 0.077 | | | | | 11.89% | croup | 0 |
| 1877 | 78.313 | -0.164 | | | | | 24.81% | scarlet fever | 0 |
| 1878 | 97.561 | 0.018 | | | | | 17.48% | scarlet fever | 1 |
| 1879 | 89.888 | -0.094 | | | | | 17.19% | scarlet fever | 0 |

**Table 1 continued**

| Year | IMR parishes | Deviation/Trend parishes | IMR county | Deviation/Trend county | % airbone inf. dis. deaths <1 | % airbone inf. dis. deaths <10 | % airbone inf. dis. deaths all ages | Main death cause county | High disease load year |
|---|---|---|---|---|---|---|---|---|---|
| 1880 | 91.549 | -0.110 | | | | | 11.28% | diptheria | 0 |
| 1881 | 173.913 | 0.669 | 107.267 | -0.012 | | 16.83% | 7.08% | diptheria | 1 |
| 1882 | 63.694 | -0.357 | 107.541 | -0.011 | | 17.93% | 7.11% | diptheria | 0 |
| 1883 | 90.909 | -0.035 | 132.036 | 0.220 | | 24.62% | 10.78% | scarlet fever | 0 |
| 1884 | 96.552 | 0.062 | 103.041 | -0.020 | | 37.56% | 16.66% | scarlet fever | 0 |
| 1885 | 38.710 | -0.569 | 96.653 | -0.045 | | 22.97% | 7.69% | scarlet fever | 0 |
| 1886 | 147.059 | 0.591 | 94.651 | -0.033 | | 12.25% | 3.80% | scarlet fever | 1 |
| 1887 | 89.655 | -0.029 | 88.861 | -0.073 | | 12.15% | 3.66% | whooping cough | 0 |
| 1888 | 80.537 | -0.120 | 97.650 | 0.022 | | 23.21% | 8.06% | measles | 0 |
| 1889 | 84.746 | -0.077 | 94.888 | -0.011 | | 14.75% | 4.69% | diptheria | 0 |
| 1890 | 109.489 | 0.174 | 102.685 | 0.065 | | 30.36% | 11.20% | diptheria | 1 |
| 1891 | 85.938 | -0.091 | 93.230 | -0.033 | | 0.00% | 9.87% | diptheria | 0 |
| 1892 | 87.500 | -0.101 | 100.802 | 0.049 | | | 15.63% | diptheria | 0 |
| 1893 | 75.000 | -0.261 | 92.115 | -0.034 | | | 15.39% | diptheria | 0 |
| 1894 | 172.414 | 0.634 | 94.563 | 0.000 | | | 14.72% | diptheria | 1 |
| 1895 | 70.866 | -0.317 | 93.423 | -0.005 | | | 7.57% | diptheria | 0 |
| 1896 | 126.050 | 0.246 | 96.589 | 0.034 | | | 5.16% | diptheria | 0 |
| 1897 | 70.707 | -0.275 | 88.937 | -0.042 | | | 4.46% | diptheria | 0 |
| 1898 | 88.496 | -0.082 | 86.627 | -0.066 | | | 5.03% | diptheria | 0 |
| 1899 | 108.527 | 0.117 | 101.592 | 0.092 | | | 5.34% | diptheria | 0 |
| 1900 | 95.588 | -0.026 | 93.150 | 0.007 | | | 4.30% | diptheria | 0 |
| 1901 | 94.828 | -0.044 | 91.581 | 0.004 | | | 4.64% | diptheria | 0 |
| 1902 | 98.361 | -0.015 | 88.140 | -0.018 | | | 6.03% | diptheria | 0 |
| 1903 | 121.951 | 0.232 | 87.861 | -0.004 | | | 4.87% | diptheria | 0 |
| 1904 | 90.226 | -0.053 | 84.366 | -0.030 | | | 5.59% | diptheria | 0 |
| 1905 | 86.207 | -0.052 | 85.596 | -0.004 | | | 6.73% | diptheria | 0 |
| 1906 | 72.993 | -0.167 | 88.627 | 0.046 | | | 4.45% | diptheria | 0 |
| 1907 | 96.491 | 0.125 | 79.054 | -0.049 | | | 3.83% | diptheria | 0 |
| 1908 | 93.458 | 0.113 | 92.232 | 0.137 | | | 4.29% | whooping cough | 0 |
| 1909 | 72.000 | -0.125 | 66.060 | -0.158 | | | 2.47% | diptheria | 0 |
| 1910 | 83.333 | 0.012 | 80.387 | 0.052 | | | 2.39% | diptheria | 0 |
| 1911 | 65.041 | -0.226 | | | | | 14.94% | tuberculosis | 0 |
| 1912 | 102.804 | 0.174 | | | | | 14.87% | tuberculosis | 0 |
| 1913 | 97.345 | 0.082 | | | | | 14.11% | tuberculosis | 0 |
| 1914 | 84.211 | -0.071 | | | | | 13.80% | tuberculosis | 0 |
| 1915 | 66.038 | -0.270 | | | | | 12.08% | tuberculosis | 0 |
| 1916 | 132.653 | 0.491 | | | | | 14.91% | tuberculosis | 1 |
| 1917 | 91.954 | 0.123 | | | | | 13.51% | tuberculosis | 0 |
| 1918 | 54.3478 | -0.244 | | | | | 34.43% | influenza | 1 |

**Table 2: Risk of dying between ages 1 and 10 by gender and cohort, controlling for the disease environment during the first year of life and nutrition during the fetal stage, Scania 1840-1928**

| | Cohorts 1830-1879 | | | | Cohorts 1880-1918 | | | |
|---|---|---|---|---|---|---|---|---|
| | Model A: Girls | | Model B: Boys | | Model C: Girls | | Model D: Boys | |
| | Means | Hazard ratio | Means | Hazard ratio | Means | Hazard ratio | Means | Hazard ratio |
| Disease load, birth year | | | | | | | | |
| Low (ref.) | 80.5 | 1.00 | 78.9 | 1.00 | 85.9 | 1.00 | 85.6 | 1.00 |
| High | 19.5 | 1.11 | 21.1 | 0.69 * | 14.2 | 0.37 * | 14.4 | 0.35 ** |
| Disease load & Age >= 2.3 | (--) | (--) | (--) | (--) | 12.0 | 3.57 ** | 12.2 | 3.94 ** |
| Disease load & Age >= 2.9 | (--) | (--) | 16.2 | 1.41 | (--) | (--) | (--) | (--) |
| Disease load & Age >= 4.5 | (--) | (--) | (--) | (--) | (--) | (--) | 8.5 | 0.35 * |
| Disease load & Age >= 6.8 | (--) | (--) | (--) | (--) | 4.8 | 0.41 | (--) | (--) |
| Ln consumer price index, conception year | | | | | | | | |
| ln CPI < 0.03 (ref.) | 81.4 | 1.00 | 80.7 | 1.00 | 91.7 | 1.00 | 91.6 | 1.00 |
| ln CPI >= 0.03 | 18.6 | 0.83 | 19.3 | 1.03 | 8.3 | 1.02 | 8.4 | 0.97 |
| Socioeconomic status | | | | | | | | |
| Higher occupations (ref.) | 5.9 | 1.00 | 7.0 | 1.00 | 13.6 | 1.00 | 12.8 | 1.00 |
| Skilled | 8.7 | 1.56 | 7.9 | 1.42 | 16.5 | 1.56 | 15.8 | 2.71 *** |
| Farmers | 33.6 | 0.99 | 32.6 | 1.44 | 22.2 | 1.41 | 23.1 | 1.35 |
| Lower skilled | 24.9 | 1.38 | 26.2 | 1.35 | 22.2 | 1.64 | 24.5 | 2.29 ** |
| Unskilled | 21.3 | 1.19 | 21.2 | 1.56 * | 17.5 | 1.91 * | 16.2 | 2.68 *** |
| NA | 5.7 | 1.51 | 5.1 | 1.13 | 8.0 | 2.63 *** | 7.5 | 2.51 ** |
| Year of birth | 1855.8 | 1.00 | 1855.9 | 1.00 | 1898.1 | 0.97 *** | 1898.8 | 0.96 *** |
| Parish of birth | | | | | | | | |
| Same parish as residence (ref.) | 77.6 | 1.00 | 78.6 | 1.00 | 64.0 | 1.00 | 65.55 | 1.00 |
| Different parish | 22.4 | 0.82 | 21.5 | 0.78 * | 36.0 | 0.64 ** | 34.45 | 0.96 |
| Number of children | 4179 | | 4509 | | 4051 | | 4228 | |
| Number of deaths | 391 | | 444 | | 172 | | 180 | |
| Total person years | 24720 | | 26729 | | 17659 | | 18682 | |
| Overal p value | 0.055 | | 0.180 | | <0.01 | | <0.01 | |

**Table 3: Risk of dying between ages 50 and 70 by gender and cohort, controlling for the disease environment during the first year of life and nutrition during the fetal stage, Scania 1880-1968**

| | Cohorts 1830-1879 | | | | Cohorts 1880-1918 | | | |
|---|---|---|---|---|---|---|---|---|
| | Model A: Women | | Model B: Men | | Model C: Women | | Model D: Men | |
| | Means | Hazard ratio | Means | Hazard ratio | Means | Hazard ratio | Means | Hazard ratio |
| Disease load, birth year | | | | | | | | |
| Low (ref.) | 78.8 | 1.00 | 79.4 | 1.00 | 88.11 | 1.00 | 88.11 | 1.00 |
| High | 21.3 | 1.29 | 20.6 | 1.46 *** | 11.89 | 1.86 *** | 11.89 | 1.50 * |
| Disease load & Age >= 60.5 | 8.37 | 0.73 | (---) | (---) | (---) | (---) | (---) | (---) |
| Disease load & Age >= 63 | (---) | (---) | 5.6 | 0.67 * | (---) | (---) | (---) | (---) |
| Ln consumer price index, conception year | | | | | | | | |
| ln CPI < 0.03 (ref.) | 82.1 | 1.00 | 81.6 | 1.00 | 90.24 | 1.00 | 90.24 | 1.00 |
| ln CPI >= 0.03 | 17.9 | 1.02 | 18.4 | 0.92 | 9.76 | 1.30 | 9.76 | 1.22 |
| Socioeconomic status | | | | | | | | |
| Higher occupations (ref.) | 17.1 | 1.00 | 16.75 | 1.00 | 26.22 | 1.00 | 26.22 | 1.00 |
| Skilled | 11.2 | 0.82 | 14.2 | 0.78 | 11.63 | 0.67 | 11.63 | 0.84 * |
| Farmers | 22.5 | 0.97 | 27.68 | 0.67 *** | 12.95 | 0.68 | 12.95 | 0.71 |
| Lower skilled | 20.6 | 1.21 | 19.74 | 0.71 ** | 24.22 | 0.82 | 24.22 | 0.65 |
| Unskilled | 6.8 | 1.33 | 8.35 | 1.01 | 6.14 | 0.75 | 6.14 | 0.94 |
| NA | 21.8 | 1.30 | 13.28 | 1.38 ** | 18.84 | 1.50 * | 18.84 | 1.30 |
| Year of birth | 1858.0 | 0.98 *** | 1858.3 | 0.99 *** | 1896.3 | 1.00 | 1896.3 | 1.00 |
| Parish of birth | | | | | | | | |
| Same parish as residence (ref.) | 15.9 | 1.00 | 18.39 | 1.00 | 13.87 | 1.00 | 13.87 | 1.00 |
| Different parish | 84.1 | 0.74 ** | 81.61 | 1.05 | 86.13 | 0.92 | 86.13 | 1.02 |
| Number of individuals | 1621 | | 1648 | | 1812 | | 1835 | |
| Number of deaths | 290 | | 291 | | 163 | | 176 | |
| Total person years | 17693 | | 17106 | | 18019 | | 17739 | |
| Overal p value | <0.01 | | <0.01 | | <0.01 | | 0.19 | |

**Table 4: Percentage of deaths by cause in ages 50-70 by cohort, Scania 1880-1968**

| Death cause \ Cohort | 1830-1879 | 1880-1918 |
|---|---|---|
| Airborne infectious diseases | 18% | 4% |
| Food & waterborne infectious diseases | 0% | 0% |
| Other infectious diseases | 1% | 2% |
| Cardiovascular diseases & diabetes | 26% | 43% |
| Accidents, crimes, etc | 4% | 4% |
| Weakness due to old age | 4% | 0% |
| Cancer | 17% | 36% |
| Other non infectious diseases | 13% | 10% |
| Not specified | 15% | 1% |

**Table 5: Percentage of deaths by cause in ages 50-70 by gender, cohort and disease load on birth year, Scania 1880-1968**

| | **Gender and cohort** | Females 1830-1879 | | Females 1880-1918 | | Males 1830-1879 | | Males 1880-1918 | |
|---|---|---|---|---|---|---|---|---|---|
| | **Disease load on birth year** | Low | High | Low | High | Low | High | Low | High |
| **Death cause** | Airborne infectious diseases | 19% | 19% | 6% | 9% | 17% | 15% | 2% | 0% |
| | Food & waterborne inf. dis. | 0% | 0% | 0% | 0% | 0% | 0% | 0% | 0% |
| | Other infectious diseases | 0% | 0% | 2% | 0% | 3% | 1% | 1% | 6% |
| | Cardiovascular dis. & diabetes | 29% | 25% | 31% | 44% | 25% | 18% | 52% | 47% |
| | Accidents, crimes, etc | 3% | 0% | 2% | 6% | 7% | 6% | 4% | 6% |
| | Weakness due to old age | 5% | 3% | 0% | 0% | 4% | 1% | 1% | 0% |
| | Cancer | 16% | 18% | 44% | 26% | 18% | 19% | 32% | 31% |
| | Other non infectious diseases | 13% | 12% | 13% | 15% | 12% | 19% | 7% | 9% |
| | Not specified | 15% | 22% | 1% | 0% | 13% | 19% | 1% | 0% |

**Table 6: Risk of dying between ages 45 and 70 for males born between 1830 and 1879, Scania**

| | Means | Hazard ratio |
|---|---|---|
| Disease load, birth year | | |
| Low (ref.) | 79.0 | 1.00 |
| High | 21.0 | 1.66 *** |
| Disease load & Age >= 63 | 4.15 | 0.50 ** |
| Ln consumer price index, conception year | | |
| ln CPI < 0.03 (ref.) | 81.88 | 1.00 |
| ln CPI >= 0.03 | 18.12 | 0.84 |
| Socioeconomic status | | |
| Higher occupations (ref.) | 16.7 | 1.00 |
| Skilled | 13.9 | 0.75 |
| Farmers | 27.9 | 0.71 * |
| Lower skilled | 20.2 | 0.79 |
| Unskilled | 9.3 | 0.96 |
| NA | 12.1 | 1.39 * |
| Year of birth | 1857.7 | 0.99 *** |
| Parish of birth | | |
| Same parish as residence (ref.) | 18.99 | 1.00 |
| Different parish | 81.01 | 1.11 |
| | | |
| Number of individuals | 2035 | |
| Number of deaths | 343 | |
| Total person years | 22881 | |
| Overal p value | <0.01 | |

**Table 7: Risk of dying for females born between 1880 and 1918, Scania**

| | Ages 30-70 | | Ages 10-30 | |
|---|---|---|---|---|
| | Means | Hazard ratio | Means | Hazard ratio |
| Disease load, birth year | | | | |
| Low (ref.) | 86.66 | 1.00 | 85.19 | 1.00 |
| High | 13.34 | 1.57 *** | 14.81 | 1.23 |
| Ln consumer price index, conception year | | | | |
| ln CPI < 0.03 (ref.) | 91.37 | 1.00 | 91.33 | 1.00 |
| ln CPI >=0.03 | 8.63 | 1.25 | 8.67 | 1.96 ** |
| Socioeconomic status | | | | |
| Higher occupations (ref.) | 26.8 | 1.00 | 21.52 | 1.00 |
| Skilled | 13.95 | 0.90 | 14 | 1.10 |
| Farmers | 14.55 | 0.98 | 23.25 | 1.14 |
| Lower skilled | 25.19 | 0.96 | 24.43 | 1.50 |
| Unskilled | 7.3 | 0.92 | 8.64 | 1.84 |
| NA | 12.22 | 1.57 ** | 8.16 | 1.53 |
| Year of birth | 1898.6 | 0.98 *** | 1899.7 | 0.97 *** |
| Parish of birth | | | | |
| Same parish as residence (ref.) | 15.08 | 1.00 | 36.18 | 1.00 |
| Different parish | 84.92 | 0.89 | 63.82 | 0.68 * |
| | | | | |
| Number of individuals | | 3437 | | 6189 |
| Number of deaths | | 247 | | 100 |
| Total person years | | 45485 | | 32316 |
| Overal p value | | <0.01 | | <0.01 |

**Table 8: Risk of dying for males born between 1880 and 1918, Scania**

| | Ages 57-70 | | Ages 10-57 | |
|---|---|---|---|---|
| | Means | Hazard ratio | Means | Hazard ratio |
| Disease load, birth year | | | | |
| Low (ref.) | 85.58 | 1.00 | 85.86 | 1.00 |
| High | 14.42 | 1.74 ** | 14.14 | 0.80 |
| Ln consumer price index, conception year | | | | |
| ln CPI < 0.03 (ref.) | 88.19 | 1.00 | 91.72 | 1.00 |
| ln CPI >=0.03 | 11.81 | 1.20 | 8.28 | 1.43 * |
| Socioeconomic status | | | | |
| Higher occupations (ref.) | 22.94 | 1.00 | 20.24 | 1.00 |
| Skilled | 14.91 | 0.81 | 15.79 | 1.06 |
| Farmers | 16.12 | 0.84 | 21.76 | 0.91 |
| Lower skilled | 25.16 | 0.59 * | 24.51 | 1.04 |
| Unskilled | 9.93 | 1.13 | 9.93 | 0.94 |
| NA | 10.94 | 1.43 | 7.77 | 1.46 |
| Year of birth | 1894.0 | 1.02 | 1900.2 | 0.98 *** |
| Parish of birth | | | | |
| Same parish as residence (ref.) | 19.35 | 1.00 | 32.35 | 1.00 |
| Different parish | 80.65 | 1.16 | 67.65 | 0.87 |
| | | | | |
| Number of individuals | | 1295 | | 7282 |
| Number of deaths | | 123 | | 249 |
| Total person years | | 9332 | | 69370 |
| Overal p value | | 0.107 | | <0.01 |

**Figure 1: Infant mortality rate, trend and deviation/trend, five parishes in Scania 1830-1918**

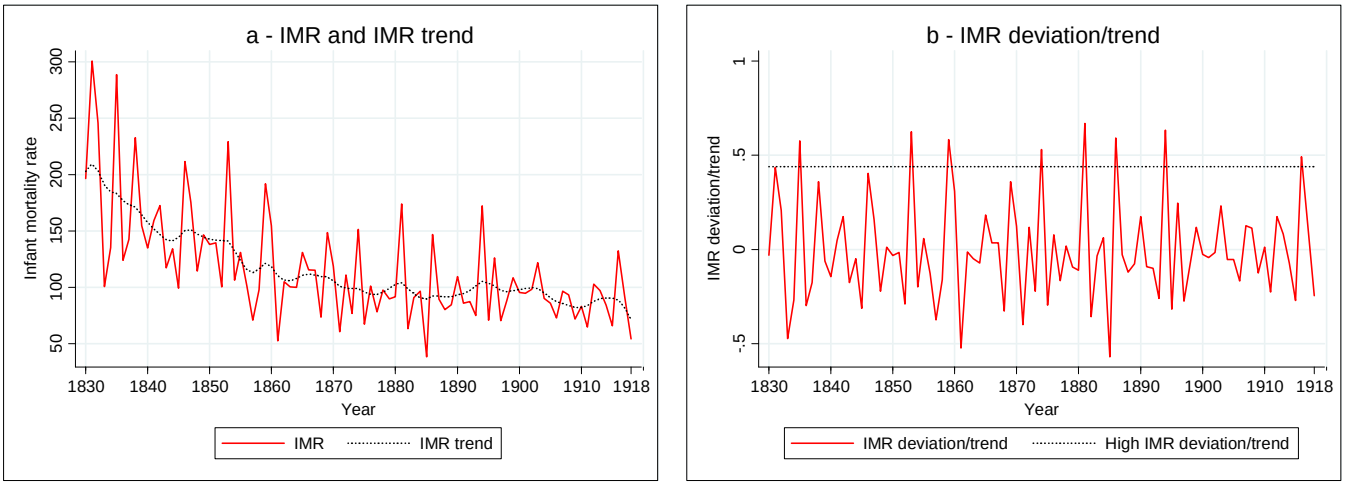


**Figure 2: Consumer price indices, trend and deviation for Sweden, 1830-1918**

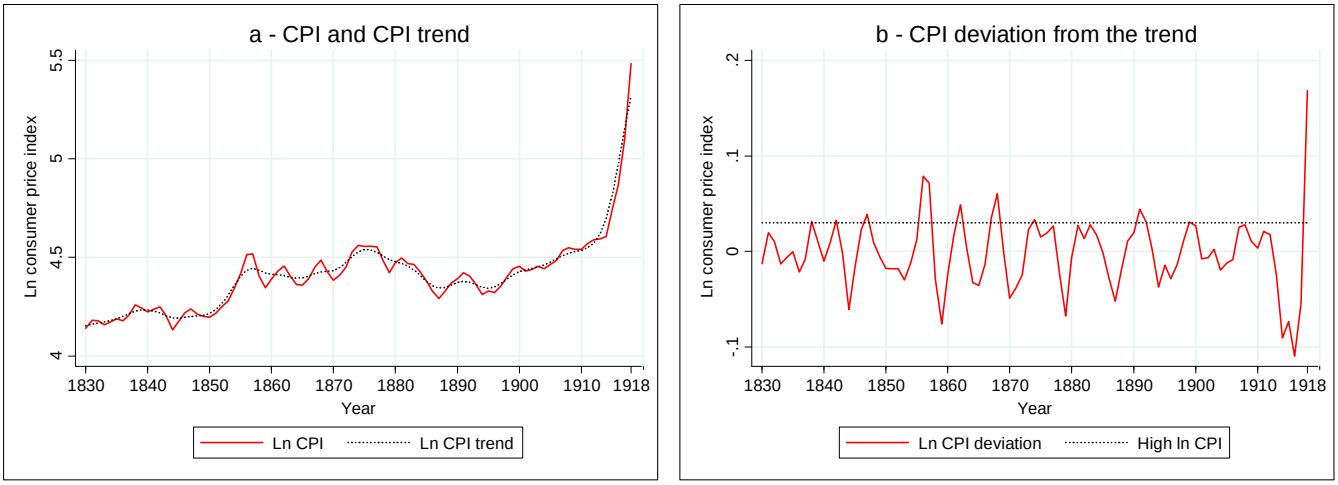

**Figure 3: Hazard of dying between ages 1 and 10 for children born in years with a high disease load compared to those born in other years by gender and cohort, Scania 1840-1928**

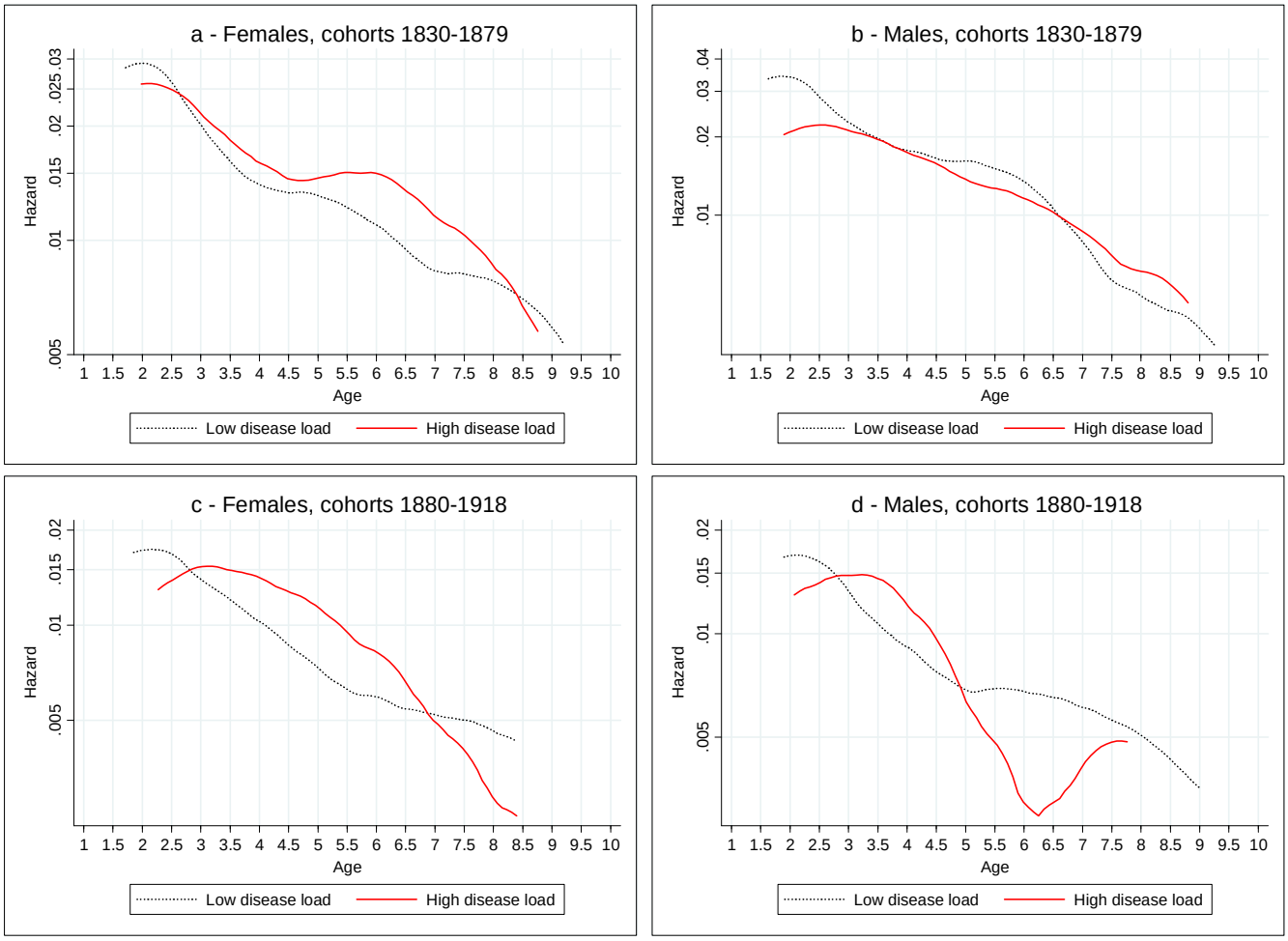

**Figure 4: Hazard of dying between ages 50 and 70 for individuals born in years with a high disease load compared to those born in other years by gender and cohort, Scania 1880-1968**

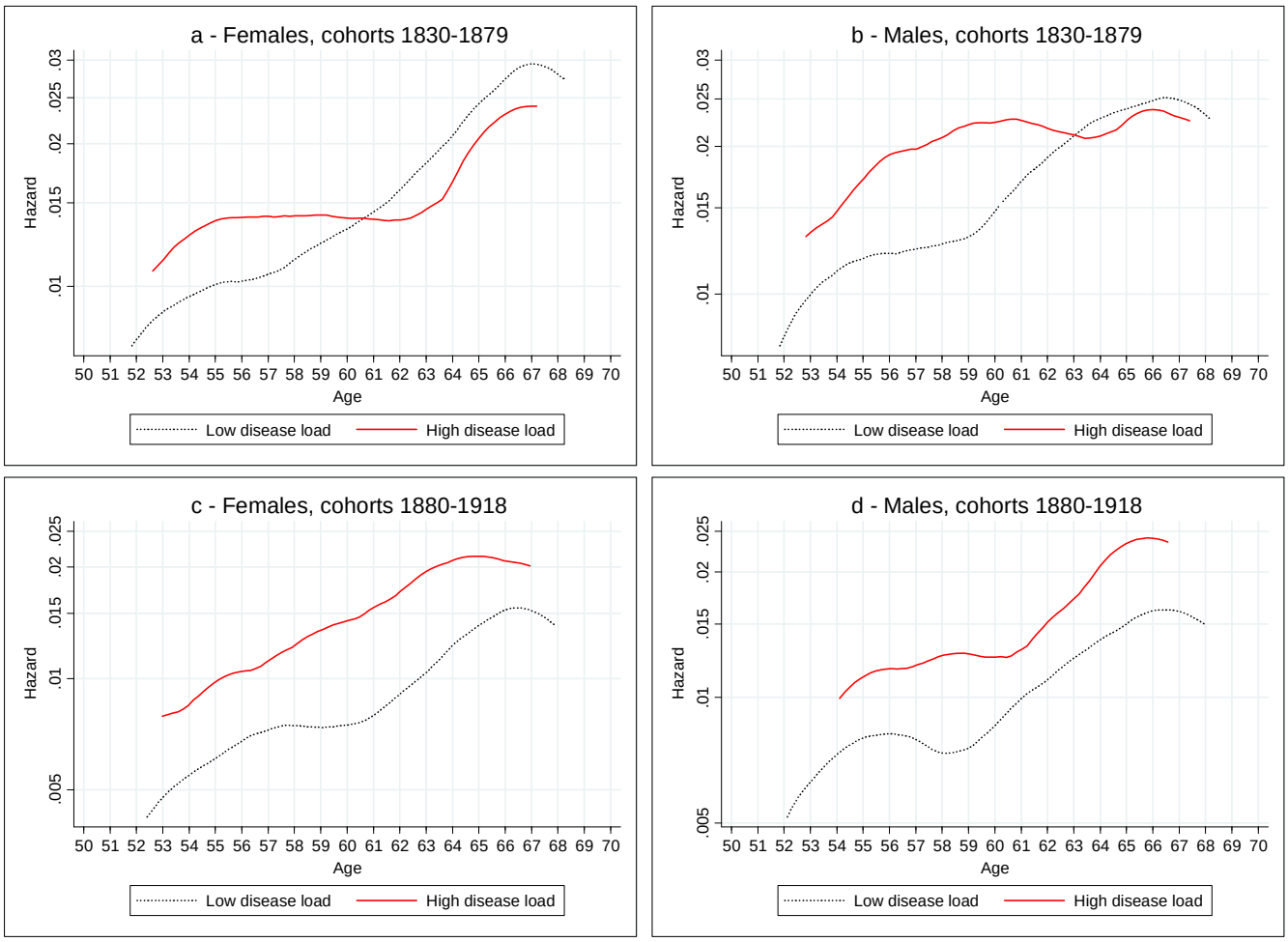

**Figure 5: Cumulative hazard of dying between ages 1 and 70 for individuals born in years with a high disease load compared to those born in other years by gender and cohort, Scania 1830-1968**

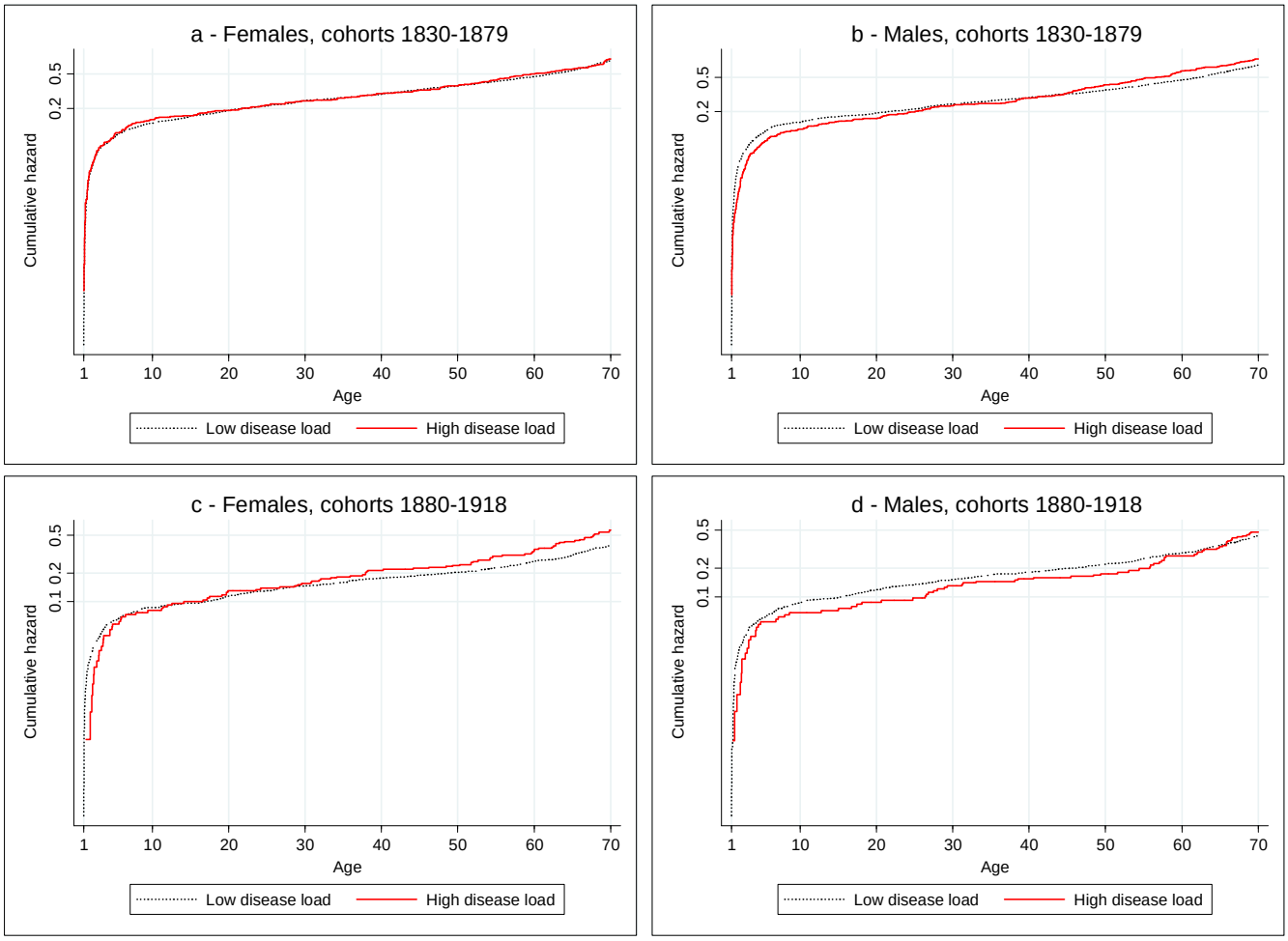

**Figure 6: Hazard and cumulative hazard of dying between ages 30 and 70 for individuals born in years with a high disease load compared to those born in other years by gender and cohort, Scania 1830-1968**

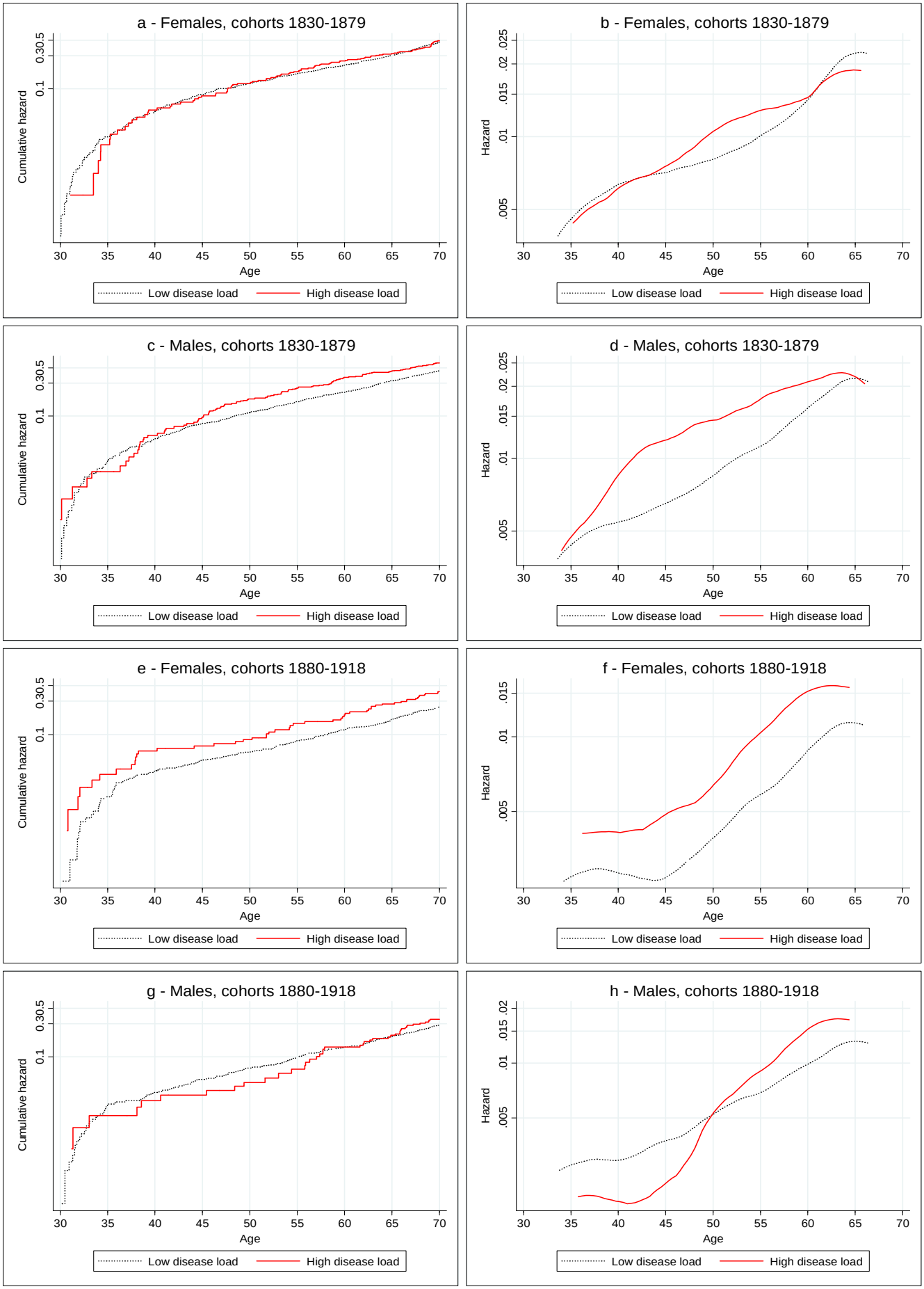

**Figure 7: Hazards of dying between ages 1 and 70 for individuals conceived in years with high or low prices, cohorts 1880-1918**