# Where you live, or who you are? The role of childhood for adult health, southern Sweden, 1900-2013

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**Abstract**

Adverse early-life conditions have lasting negative effects on old-age income and health (Almond, 2006; D. J. P. Barker, 1992; Mazumder et al., 2010) Roseboom, 2006). Improvements in early life, whether less exposure to disease or better diets, have been considered to be an important driver of the historical mortality decline (Finch & Crimmins, 2004) and economic growth (Fogel & Costa, 1997). Consequently, understanding the role of early life factors and whether they have changed over time is important for the understanding the improvements in living standards and health over the last two centuries. Using longitudinal data from Southern Sweden this paper evaluates the impact of socioeconomic status and disease exposure on mortality later in life for individuals born in the 20th century. Previous studies have shown that individuals born in the 19th century in years with high infant mortality rates or with smallpox or whooping cough epidemics experienced lower socioeconomic performance and higher levels of adult and old-age mortality, while no effects were seen in relation to exposure to high prices or socioeconomic status of parents. The question addressed in this paper is whether these results persisted for later born cohorts and, if they did not, when did a change take place. In this work we show that females born between 1900 and 1950 in years with high infant mortality rates had greater risks of dying in ages 75-95. For men, an increased risk of death for those exposed was only seen among those who were aged 55-70 and who were born before 1930. We also show that parental socio-economic status started to play a role for adult health in the beginning of the 20th century.

Key words: adult mortality, childhood conditions, longitudinal, Sweden

## Introduction

Earlier research has identified three main factors that may be particularly important for health in later life: nutritional deprivation, exposure to disease, and socioeconomic adversity. First, inadequate nutrition in utero may result in physiological and metabolic restrictions that increase the risk of cardiovascular disease mortality in later life (D. J. Barker, 2006). The basic idea is that malnourished children do not develop cells and organs, such as the artery system, as they should and are therefore more likely to develop diabetes, cancer, cardiovascular disease, metabolic disorders, and so on later in

life. As a consequence of improvements in diets, humans got healthier and stronger. This way it has not only expanded life but also contributed to an increase in labour productivity and economic growth, making the next generations even better nourished and healthier, more productive, and so on (Floud et al., 2011; Fogel & Costa, 1997).

Second, exposure to infectious disease in the first years of life may cause damage that influences adult health and mortality. One possible mechanism is inflammation caused by infections, such as small-pox and whooping cough, which damages the artery system and can lead to chronic disease in later life (Bengtsson & Lindström, 2003; Quaranta, 2013). Exposure to airborne infectious diseases at a young age is associated with cough, phlegm, and impaired ventilatory function later in life (D. J. P. Barker et al., 1991; Shaheen et al., 1994). It has also been found that in utero exposure to infectious diseases have similar effects from studies of the long-term effects of the Spanish flu in 1918 (Almond & Mazumder, 2005). In fact, in utero exposure to the 1918 pandemic was associated not only with worsening educational and job-market outcomes but also with higher cardiovascular disease prevalence and mortality (Almond, 2006; Mazumder et al., 2010; Myrskylä et al., 2013; Richter and Robling, 2015).

Third, early-life family characteristics and socioeconomic conditions are important predictors of later health. Many studies have documented strong associations between childhood conditions, measured by parental occupation, education, housing characteristics, or family income and various adult health outcomes and mortality (Hayward & Gorman, 2004; Preston, Hill, & Drevenstedt, 1998; Strand & Kunst, 2007). Analyses exploiting exogenous macro variation in socioeconomic conditions, typically fluctuations in per capita gross domestic product, have obtained results that are consistent with those that consider socioeconomic conditions at an individual level. For example, being born in a recession, compared to being born in a period of economic growth, increases all-cause, and in particular cardiovascular disease, mortality (van den Berg et al., 2006). However, several studies find no social differences in adult mortality until the latter part of the 20$^{th}$ century, which one would anticipate if socioeconomic conditions in childhood was important for later life health (Bengtsson & Dribe, 2011; Bengtsson & van Poppel, 2011).

Analyses for later born cohorts show, however, mixed results. While studies using register data for Sweden find effects of disease exposure in the first years of life on income and sick leaves for immigrants and natives controlling for family factors (Helgertz, 2010; Helgertz & Persson, 2014), a study using survey data for a city in Southern Sweden finds no such effect for native born on health outcomes (Lindström, 2015). In a study that focused on individuals born between 1914 and 1922 in Sweden, those exposed in-utero to the 1918 influenza pandemic had worse health in adulthood and males also experienced increased hospitalization and mortality in old ages, particularly from cancer, while only weak (Bengtsson & Helgertz, 2015).

This paper contributes to this rapidly increasing strand of literature by evaluating the role of socioeconomic status and disease exposure in the first years of life on later life health for individuals born in the 20th century in southern Sweden. Previous studies focusing on the same area have shown that individuals born in the latter parts of the 18th century and during the 19th century in years with high infant mortality rates or with smallpox or whooping cough epidemics experienced higher levels of adult and old-age mortality, while no effects were seen in relation to exposure to high prices (Bengtsson & Lindström, 2003; Quaranta, 2013; Quaranta, 2014). In this work we analyse whether such patterns persisted also for individuals born during the 20th century.

**Data and methods**

This work uses data from the Scanian Economic Demographic Database (SEDD) (Bengtsson et al., 2016), which comprises births, deaths, marriages, and migrations occurring from 1813 to 1968 in the rural parishes of Hög, Kävlinge, Halmstad, Sireköpinge, and Kågeröd and from 1948 to 1968 in the city of Landskrona. The SEDD was constructed using register-type data from catechetical examination registers and updated with information on births, marriages, and deaths from church books. The material is of high quality, and the gaps for births, deaths, and marriages are limited (Bengtsson & Lindström, 2000).

The SEDD also contains detailed information on occupations, obtained from catechetical examination, poll-tax, and income registers. Occupation at the time of birth was also registered for all individuals, including in-migrants. All occupations were classified according to the SOCPO scheme, which comprises five categories based on level of skills, degree of supervision, whether self-employed or not as well as pure status (van de Putte & Miles, 2005). After 1968 SEDD has been linked to the data from Statistics Sweden and the National Board of Health and Welfare, which allows us to follow individuals anywhere in Sweden.

The role of early life exposures is evaluated by considering different indicators. Occupation of the father at the time of birth is used to measure the household conditions in early childhood. Disease exposure is defined by considered deviations from the trend in infant mortality rates (IMR), obtained from official sources and measured at the county level. The IMR series of each county were detrended by applying a Hodrick-Prescott (HP) filter (Hodrick and Prescott 1997) with a filtering factor of 6.25, which is the recommended value to remove the trend from yearly series (Ravn and Uhlig 2002). The trend components of early life conditions are highly correlated with current macro-conditions, preventing these effects from being separated (Lindeboom, Portrait, & van den Berg, 2010). The trends in IMRs are, in fact, likely to reflect long-term changes in the development of healthcare and the economy. Instead, we are interested in measuring the effects of short-term variations in the disease environment and the level of nutrition, particularly years of epidemics, which are better captured by cyclical

fluctuations. Years with high infant mortality rate were defined as years where the relative deviation from the trend in the county was higher than the 80$^{th}$ percentile in the distributions of the relative deviations from the trend for that county.

The long-term impacts of early life exposures is evaluated on mortality at different ages (20-50, 50-75 and 75-95) and distinctively by gender. Cox proportional hazard models are estimated. In the models individuals born in years with high IMR are compared to individuals born in years with medium-low IMR and individuals born into high status families (SOCPO 3-5) are compared to individuals born into low status families (SOCPO 1-2). We follow individuals born anywhere in Sweden and who are currently living in one of the five rural parishes or Landskrona. There are variations in the data before and after 1948, since data for the city of Landskrona is currently available only after such year.

**Results**

*Previous findings*

In previous studies we have evaluated the impact of IMR in the year of birth and of parental SES at birth on mortality in ages 1-70 for individuals born in the five Scanian rural parishes in the years 1813-1898 and who were followed until 1968. IMR in the year of birth was calculated directly from the data. The series were also detrended and IMR thresholds were identified in the same way as in the current paper.

The results of such work, which are presented in Table 2, show a fluctuation between selection and scarring across age. In early childhood for females and early childhood and adolescence for males, a dominance of selection was observed. In other words individuals born in a year of high IMR showed lower probabilities of dying. From early adulthood for males and the post childbearing period for females, a strong dominance of scarring is seen. Significantly higher mortality was in fact observed between ages 20-49 for males and between ages 50-70 for females who were born in a year with high IMR. Higher probabilities of dying were also seen for exposed males between ages 50-70, but this effect was weaker and slightly below the threshold of statistical significance. The bottom rows of Table 5 present the results of the estimations that consider all individuals and include an interaction between the indicator for the level of the IMR in the year of birth and sex. The males exposed to a high IMR in their infancy had a higher mortality than the females between ages 20-49, with an interaction term that is statistically significant. No large or statistically significant differences across sex are seen in old age, although the effect for males is very slightly smaller.

*Current findings*

Figure 1 shows the hazard curves measuring the risk of death after age 20 by IMR at birth and SES at birth and for males and females separately. When considering the levels of IMR at birth, among females

higher hazards of death are observed in old age for those born in years with high IMR, while no strong differences can be observed among males. When considering SES at birth, both men and women show lower risks of death if they were born into families with high SES. The onset of such effect appears at an earlier age for men than for women.

Table 3 shows the results of Cox proportional hazards models for men and women born between 1900 and 1950. Among females, the year of birth is statistically significant in all models, showing a decline in the risk of death over time. In ages 20-50 no other variable among those considered is statistically significant. In ages 50-75 women who were born into a high SES family experienced an 11% lower risk of death relative to those who were born into a low-medium SES family, an effect which is statistically significant at the 5% level. An effect of the same magnitude, although with a stronger statistical significance, is observed for the SES variable among females aged 75-95. For such age group, relative to women born in years with low-medium IMR, those born in years with high IMR show an 11% higher risk of dying, a result which is statistically significant at the 5% level. Moreover, women born outside of Scania have a 9% lower risk of death than those born within such county.

When conducting tests based on Shoenfeld residuals, a violation of the proportional hazards assumption is observed for the IMR variable. For this reason a model for ages 78-95 is also estimated (results not shown), which presents no violations of the proportional hazard assumptions. In such age group women born in years with high IMR show a 15% higher risk of dying relative to women born in years with low-medium IMR, an effect which is statistically significant at the 1% level. Women born into families with high SES have a 12% lower risk of dying than those born into families of low-medium SES. Violations of the proportional hazards assumption are also observed in the models for ages 50-50 and 50-75 for the year of birth variable, although since such variable is only used as a control in the models such violation is considered non-problematic.

For males the year of birth variable also shows a decline in the risk of death over time, although such effect was not statistically significant among those aged 20-50 (Table 3). For men aged 50-75, being born into a high-SES family reduced the risk of death, relative to being born into a low-medium SES family, by 10%. The effect for such variable is slightly lower (reduction in the risk of death by 8%) in ages 75-95. In the same age group men born outside of Scania show 8% lower risk of death than those born within the county. No statistically significant effects are observed for the effect of being born in a year with high IMR in any of the age groups considered. None of the variables except for year of birth present violations in the proportional hazards assumption.

For males additional models were tested to try to identify until when the variable measuring IMR in the year of birth significantly affected the risk of death. Many of the models showed fluctuations between a dominance of selection and scarring across age. When restricting the estimations to those born before

1830, relative to men born in years with low-medium IMR, those born in years with high IMR had a 19% higher risk of dying in ages 55 to 70, a result that was statistically significant at the 5% level.

**Conclusions**

In previous research it has been shown that for historical populations the IMR of the year of birth is a good indicator of early life exposures, and that such exposures affected mortality later in life. This effect was observed in Scania for individuals born during the 19th century as well as in other populations. The current paper aimed to analyse whether such effects could also be observed for individuals born in the first half of the 20th century.

Using official statistics we have calculated county level IMR for all counties in Sweden for the period 1900 to 1950. The series for each county was detrended and deviations for the trend were calculated. IMRs of their county and year of birth were linked to all individuals. Using Cox proportional hazards models we have tested whether men and women born in years with high IMR experienced different risks of death than those born in years with low-medium IMR. As an additional indicator of early life exposures we have considered socioeconomic status at birth, classifying it into high and low-medium, based on the SOCPO scale. Models were estimated for different age groups: 20-50, 50-75 and 75-95.

The results showed that, among individuals born between 1900 and 1950, for females being born into a family of high SES reduced the risk of death in ages 50-75 and 75-95, while being born in a year with high IMR increased the risk of death in ages 75-95. For males, being born into a family of high SES reduced the risk of death in ages 20-50, 50-75 and 75-95, while no significant effects where observed for the variable measuring IMR levels at birth. Additional models were estimated to try to identify when such variable ceased to have an effect for males. Significantly higher risks of death were observed among those born in years with high IMR and before 1930, but in ages 55-70.

In previous research we had shown that among individuals born during the 19th century IMR in the year of birth had strong impacts on mortality later in life, particularly in ages above 30 years, and that the pattern varied between men and women. In the present work we have shown that IMR remains an important measure of early life exposures also for individuals born during the first half of the 20th century. Among females such pattern was evident for all cohorts considered, while among men the effects diminished for those born after 1930. Among males and females born during the 20th century the relative increases in mortality for individuals born in years with high IMR appeared for older age-groups than among individuals born during the 19th century. Such pattern is not surprising, given the change in causes of death that has been experienced across time, with declines in the importance of infectious diseases and an increase in the importance of chronic diseases. Chronic diseases have been shown to be associated with adverse early life exposures, and they primarily develop during old-age.

In future versions of this work we will expand the analysis in various directions. First of all, besides focusing on all-cause mortality, cause-specific mortality and hospitalizations will be considered as outcomes. Secondly, alternative identifications of early life exposures will be considered. In the present study the same indicator of IMR is used for all individuals born in the same year, regardless of their date of birth. This means that for men and women born during the latter part of the year the indicator primarily captures the conditions experienced in utero, while for those born in the beginning of the year it captures primarily the conditions experienced in first year of life. To overcome this limitation we will consider IMR of the year after birth (Helgertz & Persson, 2014) or individually defined measures (Quaranta, 2014). Given that aggregate data is used, the latter will be calculated by using the weighted average of the IMR of two consecutive years, with weights that depend on the proportion of infancy that occurs in each year. In future versions of this work we will also collect county level infant mortality rates for all counties in Sweden also for the 19$^{th}$ century, which will allow us to study the change over time in the impact of IMR in early life using indicators defined in a homogenous way for all cohorts. Finally, we will link the SEDD to the Swedish Death Index, a digital dataset of all deaths occurring in Sweden between 1901 and 2009. Such addition will allow us to follow all individuals present in SEDD until their death prior to 1968 and to therefore consider individuals living anywhere in Sweden instead of just focusing on those living in the five rural parishes and Landskrona, as is currently done.

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Table 1: Descriptive statistics

| | Females | | | Males | | |
|---|---|---|---|---|---|---|
| | 20-50 | 50-75 | 75-95 | 20-50 | 50-75 | 75-95 |
| *IMR year of birth (%)* | | | | | | |
| Low-medium (ref.) | 77.93 | 81.1 | 85.4 | 77.39 | 80.79 | 86.53 |
| High | 22.07 | 18.9 | 14.6 | 22.61 | 19.21 | 13.47 |
| Year of birth (average) | 1927.63 | 1922.75 | 1915.29 | 1927.88 | 1923.20 | 1916.19 |
| *SES at birth (%)* | | | | | | |
| Low-medium (ref.) | 61.75 | 63.81 | 63.68 | 61.61 | 80.79 | 86.53 |
| High | 38.25 | 36.19 | 36.32 | 38.39 | 19.21 | 13.47 |
| *County of birth (%)* | | | | | | |
| Scania (ref.) | 23.04 | 24.44 | 22.87 | 22.67 | 24.54 | 24.38 |
| Other | 76.96 | 75.56 | 77.13 | 77.33 | 75.46 | 75.62 |

Table 2: Previous findings - Hazard ratios in relation to the impact of exposure to a high IMR in infancy and to SES at birth, cohorts 1813-1898 (Quaranta, 2013)

| | Females | | | |
|---|---|---|---|---|
| | 1-4 | 5-19 | 20-49 | 50-70 |
| *IMR year of birth* | | | | |
| Low-medium | ref. | ref. | ref. | ref. |
| High | 0.89 | 1.00 | 0.94 | 1.24** |
| | [0.69,1.15] | [0.76,1.30] | [0.74,1.20] | [1.02,1.50] |
| *SES at birth* | | | | |
| Medium-high | ref. | ref. | ref. | ref. |
| Low | 1.20 | 1.16 | 1.10 | 0.94 |
| | [0.96,1.50] | [0.89,1.52] | [0.87,1.39] | [0.78,1.13] |
| Number of individuals | 5678 | 9062 | 12420 | 3351 |
| Number of deaths | 427 | 377 | 457 | 648 |

| | Males | | | |
|---|---|---|---|---|
| | 1-4 | 5-19 | 20-49 | 50-70 |
| *IMR year of birth* | | | | |
| Low-medium | ref. | ref. | ref. | ref. |
| High | 0.75** | 0.91 | 1.29** | 1.14 |
| | [0.58,0.95] | [0.69,1.19] | [1.04,1.61] | [0.95,1.38] |
| *SES at birth* | | | | |
| Medium-high | ref. | ref. | ref. | ref. |
| Low | 1.27** | 1.16 | 1.10 | 1.03 |
| | [1.03,1.56] | [0.88,1.53] | [0.85,1.41] | [0.85,1.25] |
| Number of individuals | 5964 | 8962 | 11637 | 3285 |
| Number of deaths | 497 | 355 | 431 | 612 |

| | All individuals | | | |
|---|---|---|---|---|
| | 1-4 | 5-19 | 20-49 | 50-70 |
| *IMR year of birth* | | | | |
| Low-medium | ref. | ref. | ref. | ref. |
| High | 0.89 | 0.99 | 0.93 | 1.22** |
| | [0.69,1.16] | [0.76,1.30] | [0.73,1.19] | [1.01,1.48] |
| *SES at birth* | | | | |
| Medium-high | ref. | ref. | ref. | ref. |
| Low | 1.24*** | 1.16 | 1.10 | 0.98 |
| | [1.06,1.44] | [0.96,1.41] | [0.93,1.30] | [0.86,1.12] |
| *Sex* | | | | |
| Females | ref. | ref. | ref. | ref. |
| Males | 1.15* | 0.93 | 0.93 | 1.05 |
| | [1.00,1.32] | [0.79,1.09] | [0.80,1.08] | [0.92,1.19] |
| *IMR year of birth & sex* | | | | |
| High & males | 0.83 | 0.92 | 1.40** | 0.94 |
| | [0.58,1.18] | [0.63,1.34] | [1.02,1.94] | [0.72,1.23] |
| Number of individuals | 11642 | 18024 | 24057 | 6636 |
| Number of deaths | 924 | 732 | 888 | 1260 |

Notes: The models control for year of birth (continuous variable), parish, and an indicator of whether the individual was born in one of the studied parishes or migrated to them. 95% confidence intervals in brackets. $^{*}$ $p < 0.10$, $^{**}$ $p < 0.05$, $^{***}$ $p < 0.01$.

Table 3 –Hazard ratios in relation to the impact of exposure to a high IMR in infancy and to SES at birth, cohorts 1900-1950

| | Females | | | Males | | |
|---|---|---|---|---|---|---|
| | 20-50 | 50-75 | 75-95 | 20-50 | 50-75 | 75-95 |
| *IMR year of birth* | | | | | | |
| Low-medium (ref.) | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 |
| High | 1.06 | 1.05 | 1.11** | 1.02 | 1.01 | 0.96 |
| | [0.78,1.43] | [0.93,1.20] | [1.01,1.22] | [0.82,1.27] | [0.92,1.12] | [0.86,1.07] |
| Year of birth | 0.98*** | 0.99*** | 0.98*** | 0.99 | 0.99*** | 0.97*** |
| | [0.97,0.99] | [0.98,0.99] | [0.97,0.98] | [0.99,1.00] | [0.98,0.99] | [0.97,0.97] |
| *SES at birth* | | | | | | |
| Low-medium (ref.) | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 |
| High | 0.95 | 0.89** | 0.89*** | 0.85* | 0.90*** | 0.92** |
| | [0.75,1.21] | [0.81,0.99] | [0.83,0.95] | [0.71,1.02] | [0.83,0.97] | [0.86,1.00] |
| *County of birth* | | | | | | |
| Scania (ref.) | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 |
| Other | 0.84 | 1.00 | 0.91** | 1.02 | 1.03 | 0.92* |
| | [0.62,1.14] | [0.89,1.12] | [0.84,0.99] | [0.83,1.26] | [0.94,1.12] | [0.84,1.01] |
| Individuals | 20799 | 10586 | 5899 | 21941 | 10254 | 4285 |
| Deaths | 284 | 1754 | 3508 | 540 | 3013 | 2908 |

Note: 95% confidence intervals in brackets. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Figure 1: Hazard of death by IMR and SES at birth for males and females, cohorts 1900-1950