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Where you live, or who you are? The role of childhood for adult health, southern Sweden, 1881-2015

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Abstract

Adverse early-life conditions not only cause instant health problems but also have lasting negative effects on both economic well-being and health. Improvements in early life, whether less exposure to disease, better diets, or better upbringing in general have been considered an important driver of the historical mortality decline and economic growth. Consequently, understanding the role of early life factors and how they have changed over time is important for the understanding the long-term improvements in living standards and health over the last two centuries. Our previous studies using longitudinal data from Southern Sweden up to 1968 have shown that individuals born in years with high infant mortality rates experienced lower socioeconomic performance and higher levels of adult and old age mortality. No effects of economic cycles and socioeconomic status of parents at time of birth were found. 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. Following individuals up to 2015, we find that exposure to disease in early life affects mortality later in life, particularly at higher ages, also for more recent births cohorts. We also find some evidence that parental socioeconomic status and economic cycles in early life influenced later life health for these later born cohorts though the effects found vary by age, sex and cohort of birth a great deal.

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

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Introduction

Earlier research has identified three main factors in early life 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 mortality in later life (Barker, D. J., 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. Because of improvements in diets, humans got healthier and stronger. This way, better diets has not only expanded life but also contributed to an increase in labour productivity and economic growth, making room for the next generation to become even better nourished and healthier, more productive, and so on (Floud, Fogel, Harris, & Hong, 2011; Fogel & Costa, 1997).

Second, exposure to infectious disease in the first years of life may cause damage that influences adult health. Exposure to airborne infectious diseases at a young age is associated with cough, phlegm, and impaired lung function later in life (Barker, David J. P. et al., 1991; Shaheen et al., 1994). One possible mechanism is inflammations caused by infections such as smallpox and whooping cough, which then damages the artery system and can lead to chronic disease in later life (Bengtsson, T. & Lindström, 2003; Finch & Crimmins, 2004; Quaranta, 2013). It has also been found from studies of the long-term effects of the Spanish flu in 1918 that in utero exposure to infectious diseases have similar effects (Almond & Mazumder, 2005). In fact, in utero exposure to the 1918 pandemic was associated not only with worsening educational and with job-market outcomes, but with higher cardiovascular disease prevalence and mortality as well (Almond, 2006; Helgertz, J. & Bengtsson, 2019; Mazumder, Almond, Park, Crimmins, & Finch, 2010; Myrskylä, Mehta, & Chang, 2013; Richter & Robling, 2015). It has been argued that reductions in exposure to disease in the first years of life is an important factor behind the overall expansion of life expectancy (Finch & Crimmins, 2004; Lindström & Davey Smith, 2019). The effects, both demographic and economic, are proposed to be similar to the ones caused by improvements in diets.

Third, early-life family characteristics and socioeconomic conditions are important predictors of later life 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). Parental factors might influence child health and therefore health later in life in different ways. First, it may influence diets and exposure to disease, the latter through the choice of location of the household. Since the socioeconomic position, and income, of parents might be a result of their own health, the influence on child health could be biological. It could also be that parents, depending on their resources, might mediate the effects of diseases in first years of life, given that the knowledge and the treatment exists. For example, they might use better-trained midwives or be keener

on vaccinating their children and providing them with antibiotics when it became available in the 1940s. Yet another external factor of potential importance for the health of children is the general economic situation. For example, a recession could influence the time and resources spent on children negatively. It may influence the foetus as well since it might influence the health of pregnant mothers. In fact, this is what has been found for the Netherlands, where recessions during foetal stage increases all-cause, and in particular cardiovascular disease, mortality late in life (van de Berg, Lindeboom, & Portrait, 2006). Again, this factor might depend on the socioeconomic position of the parents.

Analyses of a rural/semi-urban area in southern Sweden have shown that exposure to disease, not diets, in the first years of life have a strong effect on mortality at older ages, 55-85 years, in the period 1760 to 1894 (Bengtsson, T. & Lindström, 2000; 2003). Being born in years with outbreaks of smallpox and whooping cough lead to increased mortality in airborne infectious diseases, congenital heart disease, stomach, and kidney diseases and shortened life after 55 by about three years. The effects was direct, thus infections caused scarring, which showed up in later life, and due to ability to reach socioeconomic positions (Bengtsson, T. & Broström, 2009). Similar effects were found when expanding the analysis to 1968 (Quaranta, 2013). The effects was stronger for certain disease, like whooping cough, than others were, as measles (Quaranta, 2013). Analysis of ages from one year and above shows that scarring and selection equals out up until adulthood, where after the effects of scarring dominates to grow larger and larger by age (Quaranta, 2014).

Analyses for later born cohorts show, however, mixed results for Sweden. 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, J., 2010; Helgertz, Jonas & 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 the effects was only very small (Helgertz & Bengtsson, 2019). Since exposure to infectious disease were so important in the 19th century, public health preventions were taken when they became available, which was in the 1880s. Midwives were trained to use antiseptics and isolation hospitals were build, leading to declining mortality both instantly and over the life course confirming the importance of exposure to disease for health in childhood and later in life (Lazuka, Quaranta, & Bengtsson, 2016; Lazuka, 2018a). While such factors might interact with parental socioeconomic status, this is, however, not what was found. The same goes for the introduction of sulpha in the 1940s (Lazuka, 2018b). Meanwhile, infant mortality declined at an almost constant rate, which is why we expect a shift in its role for health at older ages.

This paper contributes to this rapidly increasing strand of literature by evaluating the role of socioeconomic status, economic cycles, and disease exposure in the first years of life on later life health for individuals born 1881 to 1940 in southern Sweden following them up to 2015. It expands our previous work by analysing whether the role of exposure to infectious diseases have changed over time and whether other early-life factors have become more important. We also expand by adding data for a nearby town, Landskrona to our original sample of five rural/semi-urban parishes.

Data and methods

This work uses data from the Scanian Economic Demographic Database (SEDD) (Bengtsson, T., Dribe, Quaranta, & Svensson, 2018), 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 1922 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). 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 until 2015.

In addition to demographic information, the SEDD also contains detailed data 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. Occupations were coded into HISCODE and later categorized into HISCLASS.

Data for Landskrona is still being digitized and will be expanded back until 1900. SES at birth was collected for all in-migrants to the rural parishes, but for in-migrants to Landskrona it was only collected for those who were ever observed after 1947. Future versions of the data will include SES at birth, as well as income data after 1902, for all individuals.

The sample selected for analysis considers individuals born anywhere in Sweden between 1881 and 1940, who lived in one of the five rural parishes or Landskrona between 1931 and 2015. The long-term impacts of early life exposures on later life mortality is measured separately for men and women by cohort of birth (1881-1899, 1900-1919 and 1920-1940). Separate models are estimated for ages 50-70 and 70-95. For individuals born between 1920 and 1940 no estimation is made of mortality in ages 70-95, given that not all such individuals can be followed until age 95.

The role of early life exposures is evaluated by considering three different indicators. Occupation of the father at the time of birth is used to measure the household conditions in early childhood. Disease exposure is measured by infant mortality rates (IMR) for the county of birth of each individual. Data

from official sources was used (Statistika Centralbyrån, 1940), which was available from 1881 onwards. Economic conditions in early life are measured by the yearly per capita real GDP (in 1910 price levels) for Sweden (Schön & Krantz, 2015). Data on county level GDP is not available on a yearly basis and that is why national series were used instead. The series were transformed by taking the natural logarithms. We use data on IMR and GDP for the years 1881 to 1940. We consider for each individual the IMR for the year of birth and the year after birth and GDP for the year before birth and the year of birth.

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, Gerard J., 2010). The trends in IMRs and GDPs 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, particularly years of epidemics, the level of nutrition, and the business cycle, which are better captured by cyclical fluctuations. The series of IMR and GDP were therefore detrended by applying a Hodrick-Prescott (HP) filter (Hodrick & Prescott, 1997) with a filtering factor of 6.25, which is the recommended value to remove the trend from yearly series (Ravn & Uhlig, 2002).

Years with high IMR 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. Figure 1 shows the relative deviations from the trend in IMR and the threshold for high IMR by county. Years with low GDP were define as years where the relative deviations from the trend were lower than the 20^{th} percentile in the distributions of the relative deviations from the trend of GDP in Sweden. Figure 2 shows the series of the natural logarithm of per capita real GDP, as well as the series of relative deviations from the trends and the threshold for low GDP.

Cox proportional hazard models are estimated, including three main types of explanatory variables. The first is IMR, categorized as high and medium-low. Two different lags are included in the models, IMR for the year of birth and for the year after birth. The second variable is GDP, categorized as medium-high and low. Two different lags are included in the models, GDP for the year before birth and for the year of birth. The third variable is SES at birth, categorized as high (HISCLASS 1-4: managers, professionals, medium skilled workers and farmers), low (HISCLASS 5-6: lower skilled and unskilled workers) and unknown. The models also control for year of birth and for area of current residence, categorized as Landskrona and rural parishes, and consider fixed effects for the county of birth.

Descriptive statistics are shown in tables 1 and 2. The values represent the percentage of total exposure time in each category. Between 15 and 21% of the sample was exposed to high IMR and between 14 and 25% was exposed to a low GDP in early life. Somewhat large differences are seen between the

proportion of individuals for whom GDP was low in the year before birth or the year of birth. Given that each period of study is about 20 years long, such differences are not alarming. Between 42 and 46% of the sample had low SES at birth. It is important to note that the proportion with unknown SES at birth was higher for individuals born in 1881-1899 than for those born in later years, something due to the current data availability, which was described above. Table 2 also shows that between 75 and 80% of the sample lived in Landskrona. This means that the population being considered in this study is substantially different from the population considered in our previous works analysing the long-term effects of early life exposures.

Results

Table 3 shows the results of Cox proportional hazard models measuring the impact of early life exposures on mortality in ages 50-70. The effect exposures to high IMR in the year of birth or the year after birth was not statistically significant for women born in 1881-1899 or 1900-1919. Women born in 1920-1940 in a year with high IMR show a 38% higher risk of dying than those born in a year with low-medium IMR, a result which was statistically significant, while no significant effects of IMR of the year after birth are seen for the same cohort. When it comes to GDP for the year before birth or the year of birth, no statistically significant effects are observed for any of the three cohorts for women. For cohorts 1881-1899, women with unknown SES at birth had a 115% higher likelihood of death than those with high SES. No statistically significant results in relation to SES at birth are observed for those born between 1900 and 1919, while for women born between 1920 and 1940 the likelihood of death was 26% higher for those with low SES and 44% higher for those with unknown SES.

For men aged 50-70, no statistically significant effects of IMR of the year of birth or the year after birth are observed for those born in 1881-1899 or 1920-1940. Men born in 1900-1919 in a year of high IMR show an 18% higher likelihood of death than those born in a year with low-medium IMR, a result which was statistically significant, while no significant effects of IMR of the year after birth are seen for the same cohort. Low GDP in the year before birth results in statistically significant higher risks of death for men born in 1881-1899 and 1920-1940, while no significant effects are seen for the same variable for those born in 1900-1919. No statistically significant effects of GDP of the year of birth are observed for any of the three cohorts. Having a low SES at birth does not lead to statistically significant increases in mortality for men aged 50-70 born in any of the three periods, while those with unknown SES at birth born in 1881-1899 and 1920-1940 have higher risks of death relative to those with high SES.

Table 4 shows the results of Cox proportional hazard models measuring the impact of early life exposures on mortality in ages 70-95. For women born in 1881-1899, 17% and 16% higher risks of death are seen, respectively, for those exposed to a high IMR in the year of birth or the year after birth,

while no statistically significant effects are seen in relation to IMR for those born in 1900-1919. No statistically significant effects are found in relation to GDP of the year before birth or the year of birth for women born in any of the two periods. For both cohorts, women born in families with low SES show higher risks of death than those born in families with high SES (9% and 10% higher risks for cohorts 1881-1899 and 1900-1919, respectively). For men aged 70-95 no statistically significant effects are observed in relation to IMR of the year of birth or the year after birth or GDP of the year before birth or the year of birth. For men born in 1900-1919, those born in families with low SES or with unknown SES have, in both cases, 13% higher risks of dying than those born in families with high SES, while no statistically significant effects of SES at birth are seen for men born in 1881-1899.

Additional estimations were also made only considering individuals residing in Landskrona (results not shown), given that, as was observed in Table 1 and Table 2, Landskrona constitutes between 75 and 80% of the study sample. The results are very similar to those presented in Tables 4 and 5 both, in terms of the magnitude, direction and statistical significance for the main explanatory variables. The only difference is that for males born in 1881-1899, statistically significant higher risks of death were observed in ages 70-95 for those exposed to a high IMR in the year after birth. Additional models were also estimated for the entire sample introducing an interaction between IMR in the year of birth and SES at birth and GDP in the year before birth and SES at birth, to test whether the impact of exposure to disease and of the economic conditions in early life varied by SES. None of the interaction terms were statistically significant in any of the models (results not shown). However, such estimations are limited by the fact that in the current version of the data SES at birth is not available for all individuals in Landskrona.

Conclusions

In previous studies we analysed the impact of adverse early life exposures for individuals born during the latter parts of the 18th century and during the 19th century in five rural/semi-urban parishes in Southern Sweden following them until they died, emigrated or to 1968. We found increased mortality in old age for those born in years with a high disease load, but no significant effects of exposure to high food prices nor to low SES. The current study expanded such analyses by evaluating the impact of disease exposure, economic fluctuations and SES in early life on mortality in late adulthood and old age for individuals born anywhere in Sweden between 1881 and 1940 who resided in the five rural/semi-urban parishes or in the city of Landskrona between 1931 and 2015. Disease exposure in early life measured by using infant mortality rates of the county of birth of each individual, while economic fluctuations were measured using national series of GDP. SES at birth was measured using occupation of fathers, also for in-migrants, although in the current version of the data such information is not available for all individuals.

We found that adverse early life exposures had some negative effects on mortality in late adulthood and old age for individuals born between 1881 and 1940. Such effects varied depending on sex, cohort of birth and type of early life exposure considered. High disease load in early life only showed scarring effects in late adulthood (ages 50 to 70) for women born in 1920-1940 and for men born in 1900-1919, and in old-age (ages 70 to 95) for women born in 1881-1899. Being exposed to an economic downturn in early life resulted in scarring effects for men in late adulthood, but not in old age, while no significant effects were observed for women. Being born in a family with low SES resulted in increased mortality in late adulthood for women born in 1920-1940, but not for men, and in old age for women born between 1881 and 1919 and for men born between 1900 and 1919.

The current work expands our previous studies both temporarily, by considering later born cohorts, and geographically, by considering rural and urban areas. Compared to our earlier findings, in this study we therefore found a somewhat weaker role of disease exposure in early life and a somewhat stronger role of economic fluctuations and socioeconomic status. However, several studies find no social differences in adult mortality until the latter part of the 20th century, which one would anticipate if socioeconomic conditions in childhood were important for later life health (Bengtsson, Tommy & van Poppel, 2011; Bengtsson, Tommy & Dribe, 2011). Although the results of this study are preliminary, partly given to the fact that digitization is still ongoing, we find that the role of exposure to disease in first year of life does not vanish entirely, which is what we expected. Furthermore, economic factors, both at family and community level, seems to become more important for later birth cohorts.

Future version will expand the analysis in various directions. First of all, having further data on Landskrona and complete information on SES at birth would allow to improve the measure of such variable as well as to consider earlier age groups. Secondly, cause-specific mortality will be considered both as covariates and as outcomes in addition to all-cause mortality. Thirdly, instead of taking into account two different lags of IMR and GDP in early life and assigning the same value to all individuals regardless of their date of birth, we will calculate weighted averages of IMR and GDP of two consecutive years in order to better identify more distinct windows of exposure (i.e. the foetal stage and infancy). We will also look into interactions between socioeconomic status and economic variations in GDP per capita. Finally, we will use data from the Swedish Death Index, a digital dataset of all deaths occurring in Sweden between 1860 and 2016. Such addition will allow us to follow all individuals present in SEDD until their death 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.

Figure 1: Series of relative deviations from the trend in IMR by county, 1881-1940

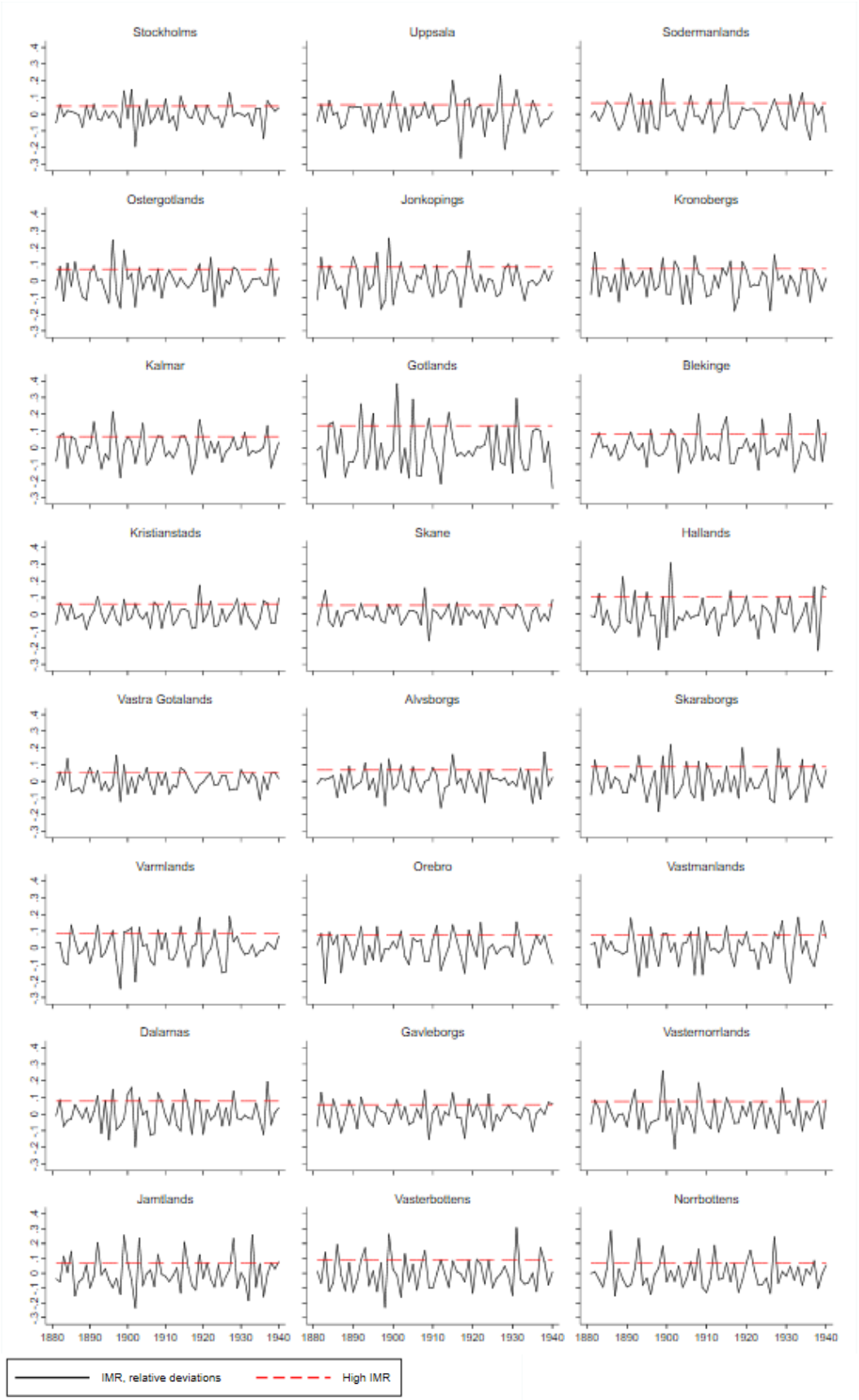

Figure 2: Series of the natural logarithm of per capita real GDP and relative deviations from the trend, Sweden 1881-1940

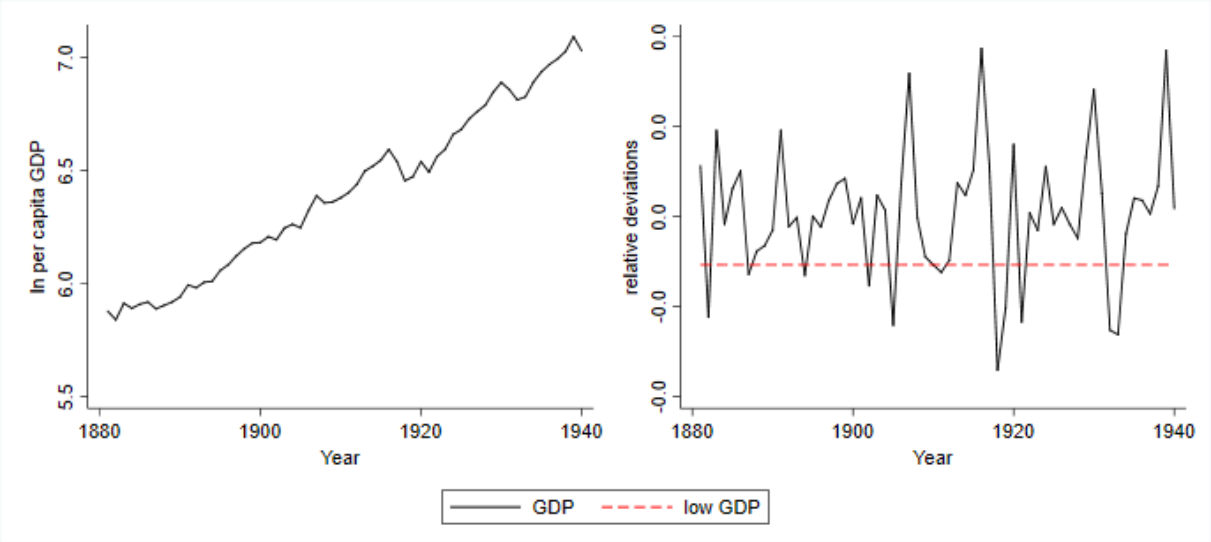

Table 1: Descriptive statistics, ages 50-70

	Women			Men		
	1881-1899	1900-1919	1920-1940	1881-1899	1900-1919	1920-1940
IMR year of birth						
Medium-low	83.42	79.32	88.86	83.68	80.81	87.70
High	16.58	20.68	11.14	16.32	19.19	12.30
IMR year after birth						
Medium-low	84.38	79.37	85.57	83.86	78.75	84.99
High	15.62	20.63	14.43	16.14	21.25	15.01
GDP year before birth						
High-medium	85.02	80.78	79.61	84.71	80.69	79.73
Low	14.98	19.22	20.39	15.29	19.31	20.27
GDP year of birth						
High-medium	85.34	75.28	85.96	83.44	75.75	85.23
Low	14.66	24.72	14.04	16.56	24.25	14.77
SES at birth						
High	34.13	36.28	38.66	32.30	35.00	37.44
Low	40.89	45.12	46.94	43.98	46.56	48.46
Unknown	24.97	18.60	14.41	23.72	18.45	14.10
Place of residence						
Rural parishes	20.98	19.59	22.69	23.06	21.11	24.66
Landskrona	79.02	80.41	77.31	76.94	78.89	75.34
Year of birth	1890.477	1909.612	1929.028	1890.539	1909.659	1929.238

Note: values calculated as percentage of time at risk

Table 2: Descriptive statistics, ages 70-95

	Women		Men	
	1881-1899	1900-1919	1881-1899	1900-1919
IMR year of birth				
Medium-low	83.90	79.05	82.52	81.19
High	16.10	20.95	17.48	18.81
IMR year after birth				
Medium-low	84.38	79.31	84.23	78.85
High	15.62	20.69	15.77	21.15
GDP year before birth				
High-medium	86.12	80.67	85.62	80.65
Low	13.88	19.33	14.38	19.35
GDP year of birth				
High-medium	85.19	75.24	83.46	75.41
Low	14.81	24.76	16.54	24.59
SES at birth				
High	35.70	36.69	33.55	36.28
Low	42.11	45.39	44.85	45.57
Unknown	22.18	17.92	21.61	18.15
Place of residence				
Rural parishes	20.91	21.51	24.16	24.36
Landskrona	79.09	78.49	75.84	75.64
Year of birth	1890.816	1909.759	1890.591	1909.915

Note: values calculated as percentage of time at risk

Table 3: Results of Cox proportional hazard models measuring the likelihood of death in adulthood and old-age based on early life exposures by sex and cohort of birth, ages 50-70

	Women			Men		
	1881-1899	1900-1919	1920-1940	1881-1899	1900-1919	1920-1940
IMR year of birth						
Medium-low (ref.)	1.00	1.00	1.00	1.00	1.00	1.00
High	0.90	0.94	1.38**	0.98	1.18**	0.92
	[0.72,1.11]	[0.76,1.15]	[1.02,1.86]	[0.81,1.19]	[1.01,1.37]	[0.71,1.20]
IMR year after birth						
Medium-low (ref.)	1.00	1.00	1.00	1.00	1.00	1.00
High	1.12	0.89	1.13	0.93	1.02	0.94
	[0.92,1.37]	[0.72,1.10]	[0.85,1.51]	[0.76,1.13]	[0.87,1.19]	[0.74,1.20]
GDP year before birth						
High-medium (ref.)	1.00	1.00	1.00	1.00	1.00	1.00
Low	1.08	0.89	1.01	1.18*	1.03	1.21**
	[0.88,1.33]	[0.72,1.09]	[0.79,1.28]	[0.98,1.42]	[0.88,1.19]	[1.01,1.45]
GDP year of birth						
High-medium (ref.)	1.00	1.00	1.00	1.00	1.00	1.00
Low	1.10	0.92	1.09	1.03	1.01	1.00
	[0.89,1.35]	[0.76,1.11]	[0.84,1.42]	[0.85,1.25]	[0.87,1.16]	[0.81,1.23]
SES at birth						
High (ref.)	1.00	1.00	1.00	1.00	1.00	1.00
Low	0.94	1.05	1.26**	1.02	1.06	1.04
	[0.77,1.14]	[0.88,1.24]	[1.02,1.56]	[0.85,1.21]	[0.93,1.20]	[0.88,1.22]
Unknown	2.15***	1.15	1.44**	2.20***	0.98	1.26**
	[1.80,2.58]	[0.93,1.43]	[1.09,1.90]	[1.85,2.62]	[0.83,1.16]	[1.01,1.57]
Year of birth	0.98**	0.99	0.99	1.00	1.00	1.00
	[0.97,1.00]	[0.98,1.00]	[0.97,1.01]	[0.98,1.01]	[0.99,1.01]	[0.98,1.01]
Place of residence						
Rural parishes (ref.)	1.00	1.00	1.00	1.00	1.00	1.00
Landskrona	1.15	1.09	1.31**	1.60***	1.41***	1.11
	[0.95,1.39]	[0.89,1.34]	[1.02,1.70]	[1.33,1.93]	[1.21,1.64]	[0.92,1.35]
Individuals	4019.00	5296.00	3751.00	3706.00	5256.00	3434.00
Deaths	724.00	673.00	452.00	836.00	1247.00	699.00

Table 4: Results of Cox proportional hazard models measuring the likelihood of death in adulthood and old-age based on early life exposures by sex and cohort of birth, ages 70-95

	Women		Men	
	1881-1899	1900-1919	1881-1899	1900-1919
IMR year of birth				
Medium-low (ref.)	1.00	1.00	1.00	1.00
High	1.17***	1.04	0.93	0.97
	[1.04,1.31]	[0.95,1.15]	[0.82,1.05]	[0.87,1.08]
IMR year after birth				
Medium-low (ref.)	1.00	1.00	1.00	1.00
High	1.16**	1.00	1.10	1.00
	[1.03,1.30]	[0.91,1.10]	[0.97,1.25]	[0.91,1.11]
GDP year before birth				
High-medium (ref.)	1.00	1.00	1.00	1.00
Low	1.00	0.99	1.01	1.00
	[0.88,1.13]	[0.90,1.09]	[0.89,1.15]	[0.90,1.10]
GDP year of birth				
High-medium (ref.)	1.00	1.00	1.00	1.00
Low	0.96	1.02	0.94	1.00
	[0.85,1.08]	[0.94,1.12]	[0.83,1.07]	[0.91,1.10]
SES at birth				
High (ref.)	1.00	1.00	1.00	1.00
Low	1.09*	1.10**	1.01	1.13***
	[0.99,1.20]	[1.02,1.19]	[0.91,1.11]	[1.04,1.23]
Unknown	1.07	1.10*	1.05	1.13**
	[0.95,1.19]	[0.99,1.21]	[0.93,1.19]	[1.02,1.26]
Year of birth	0.99**	0.99***	1.00	0.98***
	[0.98,1.00]	[0.98,0.99]	[0.99,1.01]	[0.97,0.99]
Place of residence				
Rural parishes (ref.)	1.00	1.00	1.00	1.00
Landskrona	1.14**	1.05	1.23***	1.11**
	[1.03,1.27]	[0.96,1.15]	[1.10,1.37]	[1.01,1.22]
Individuals	2747.00	3936.00	2298.00	3204.00
Deaths	2308.00	3255.00	1982.00	2912.00

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