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Don't Stress: Early Life Conditions, Hypertension, and Selection into Associated Risk Factors*

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Abstract

Early life conditions have been linked to various domains of later life health, including cardiovascular outcomes. Using life history data from 13 European countries, I find that childhood socioeconomic status and measures of childhood health are related to hypertension, although there is cross country heterogeneity in these effects. I account for potential omitted variable bias by using aggregate measures of public health at birth, which are plausibly exogenous to the individual. I find that infant mortality at birth is positively related to hypertension, even allowing for cohort effects, and controlling for GDP at birth. Results imply that improvements in early life conditions in Europe led to an overall reduction in the hypertension rate of between 3 and 6 percentage points, for the cohort born 1931-1935, relative to the cohort born 1956-1960. An alternative strand of literature in epidemiology links contemporaneous factors, such as work place environment, to heart disease. However, theories of life cycle decision making suggest that individuals may be selected into these adverse environments and behaviours on the basis of their initial conditions. I demonstrate a strong association between early environment and these risk factors. Results imply that these should therefore be viewed as outcomes which lie on the causal pathway between initial conditions and later outcomes, in which case ignoring this selection will misattribute at least part of the effects of early life environment to current circumstance. This has important policy implications for targeting hypertension as it indicates that emphasis should also be placed on combatting disadvantage across the life course, rather than just factors which only manifest themselves in adulthood.

JEL Classification: *I12, I14, N34, J11*

Keywords: *Early Life Conditions, Hypertension, Work Stress, Infant Mortality, Health Behaviour*

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1 Introduction

High blood pressure is an important marker for cardiovascular diseases (CVD), which are now responsible for a third of all deaths. Associated costs to the US economy are in the order of hundreds of billions of dollars (Heidenreich et al., 2011). The magnitude of the burden of hypertension in both developed and developing countries, contributes to predictions of a worldwide epidemic of cardiovascular disease (Kearney et al. 2005). Understanding patterns in the prevalence of high blood pressure is therefore a key concern for policy makers. In developed countries those at the top of the socioeconomic distribution tend to have lower rates of hypertension than those at the bottom. In Ireland, circulatory diseases mortality is 120% greater in the lowest occupational classes compared to the highest (Battel-Kirk and Purdy, 2007). Even small differences in blood pressure between socioeconomic groups could have a substantial impact on cardiovascular disease mortality (Colhoun et al., 1998).

The motivation for this paper relates to the potential policy implications of considering a life course perspective on the determinants of hypertension. If it is the case that contemporaneous factors (such as body mass index) are the major influence on high blood pressure, then components of adult lifestyle should be the main target. However, if hypertension is also determined by experience in early life, this implies that the focus should also be on reducing disadvantage across the life cycle. In addition, ignoring the problem of selection into a variety of adult outcomes on the basis of initial conditions could overstate the importance of what are considered to be established risk factors. The role of early life conditions are important in this context as even in high income countries such as the US, there remains a substantial degree of inequality in infant health (Currie, 2011). Developing countries are currently undergoing the type of improvements in public health environment that this paper documents for Europe. These nations are expected to experience increasing rates of population ageing, and are also expected to experience significant rises in hypertension prevalence (Kearney et al., 2005). Therefore, this paper argues that the role of early life environment in affecting blood pressure deserves a greater focus than has previously been provided to it.

A mounting body of evidence suggests that initial environment is an important determinant of outcomes in later life (Almond and Currie, 2011a). Individuals born with poor infant health, such as reduced birth weight, have been shown to have a higher risk of heart disease and hypertension as they age (Barker, 1997). Cohorts who experience reductions in early life mortality go on to experience reductions in late life mortality (Finch and Crimmins, 2004). If initial health endowments are an important determinant of health, then inequality in adult outcomes is at least partly determined by inequality in childhood (Case et al., 2002). Measured effects of childhood health may suffer from omitted variable bias; however, the best evidence supports a causal interpretation of this relationship (Almond and Currie, 2011b). Previous research has examined the effects of macroeconomic conditions at birth (Portrait et al., 2010; van den Berg et al., 2006; van den Berg et al., 2009a; van den Berg et al., 2009b; Doblhammer et al., 2011; van den Berg et al., 2011a). Each of these find an important impact of initial conditions. This paper adds to the literature by evaluating the long run effects of public health environment.

Body mass index (BMI), smoking, and lack of exercise have all been shown to be risk factors for high blood pressure. In addition, a substantial literature examines the correlation between work place environment and hypertension. However, as behavioural factors play an important role in cardiovascular disease, some understanding of how individuals make health related choices across the life cycle is required to understand how the risk of high blood pressure evolves as we age. Particularly in relation to health, there

is now a widespread acceptance across the social sciences that an individual's well-being is a stock, which evolves according to a series of endogenous and exogenous events in each time period. There are likely to be both direct and indirect effects of early life conditions (van den Berg et al., 2009b). Direct effects operate through the permanent weakening of the immune or other regulatory biological systems (Barker, 1997). Indirect pathways operate through the consequences of subsequent experience and behaviour, such as educational or disease environment, which are also likely to be affected by initial endowments.

An implication is that individuals may be selected into adverse environments and lifestyles on the basis of their early life conditions. In section 4, I demonstrate that this prediction is consistent with life cycle models of behaviour. If this is the case, then work environment (for example) lies on the causal pathway running from early life health to adult outcomes. Ignoring this potential selection problem in empirical research has the potential to be severely misleading. Simultaneously controlling for early life conditions along with these risk factors is not sufficient to account for this;[1] rather a structural approach which formally models this selection is required. This problem of adding inappropriate control variables without an identification strategy is common in the literature. For example, in a prominent review article on the Barker Hypothesis (Huxley et al., 2002), the authors find that of the 55 studies which report regression coefficients, 49 adjust for adult weight.[2]

I address the direct and indirect pathways linking childhood conditions to hypertension by using data from the Survey of Health, Ageing and Retirement in Europe (SHARE). The data are a representative sample of more than 40,000 adults over 50 years of age in 13 European countries. The most recent wave contains information on retrospective life histories which I make use of in order to measure initial conditions. I find that childhood circumstance is related to adult hypertension, as are measures of public health at birth which are not affected by individual level characteristics. A unit in increase in country level infant mortality at birth is associated with an increase in the risk of hypertension of between .06 and .11 percentage points for that individual. In addition, the data show that early life conditions are also related to risk factors for cardiovascular disease, such as BMI and work place environment.

The rest of this paper is structured as follows. Section 2 discusses the literature and data. Section 3 demonstrates a direct relationship between early life conditions and high blood pressure, both at the individual level and using aggregate measures of population health at the time of birth. In section 4, I expand on the theoretical motivation (based on Ehrlich and Chuma, 1990) for considering selection into adverse environments on the basis of initial conditions. I illustrate that there is empirical support for a gradient in these risk factors according to early life environment. Section 5 concludes by summarising the implications of these findings.

[1] At a minimum this is likely to reduce the significance of the early life environment variables due to their collinearity.
[2] Only 7 account for parental socioeconomic status.

2 Hypertension

2.1 Literature and Associated Risk Factors

According to Kearney et al. (2005), 26.4% of the global adult population had hypertension in 2000, with this estimated to rise to 29.2% in 2025. A common finding is that relative position, or alternative measures of psychosocial stress, are strongly related to heart disease. This line of inquiry was first established in studies of British civil servants (Whitehall I and II, e.g. Marmot et al., 1984; Marmot et al., 1997). This research documented that even within this relatively well-off cohort, rank plays an important part in determining health, both in terms of mortality and morbidity. The ratio of subsequent heart disease related deaths in the highest occupation grades (compared to the lowest) was in the region of 3-1 (Pickering, 1999). The hypothesis that a lower relative position in society places demands on an individual's circulatory system, through increases in stress responses and feelings of a lack of control over one's life, has been supported in animal studies (Steptoe, 2000). Marmot et al. (1997) find that much of the socioeconomic gradient in cardiovascular outcomes can be attributed to differences in psychosocial work environment. Additional contributions are made by coronary risk factors (mainly smoking) and height (taken as a proxy for early life conditions). According to Pickering (1999), the bulk of evidence favours environmental factors. About half of the prevalence of CVD can be explained by three risk factors, namely cholesterol, blood pressure, and smoking, and the Whitehall studies show that about a quarter of the SES gradient can be explained by these factors. Behavioural inputs are therefore a key component of cardiovascular disease (Stringhini et al., 2011), and these are clearly not randomly distributed in the population. Cutler and Lleras-Muney (2010) find that income, health insurance and family background account for a third of the education gradient in health behaviours, with knowledge and cognitive ability another third, and social networks a further 10%. However, education is also an outcome of early environment. Conti et al. (2010), and results in section 4 of this paper demonstrate that part of the effects of early life conditions on behaviour are orthogonal to education.

2.2 Data

SHARE (the Survey of Health, Ageing and Retirement in Europe) is a pan-European study, which is nationally representative of the over 50s in each of the participating countries. SHARE adopts a similar approach to the English Longitudinal Study of Ageing, and the Health and Retirement Study. The third wave (SHARELIFE) focuses on the life histories of respondents in 13 countries. I use two sub-samples of the data. I take measures of childhood circumstance (including health and socioeconomic status) from wave 3, combined with adult health and demographic information from wave 2. In order to account for potential omitted variable bias and include cohort effects in the analysis, I use a second sample of the individuals present in waves 1 and 2, combined with a database on infant mortality. Further details are discussed in sections 3.1 and 3.2. The total sample size for each of these groups is outlined in table 1, along with descriptive statistics for the main variables in the analysis.

There are a number of advantages to these data. The recent collection of life histories enables allows me to measure early life conditions directly. Given the theoretical issues discussed above in relation to selection (and expanded on in section 4), this allows me to determine whether initial endowments are associated with risk factors for cardiovascular disease. While not as detailed as birth cohort studies,

SHARE has the advantage of covering individuals late on in their life course. The earliest nationally representative birth cohort study began in the UK in 1946, therefore those respondents are all in their 60s (unlike SHARE, which is designed to be nationally representative of all individuals over 50). SHARE also facilitates cross country comparisons, a feature I make use of by examining the effects of aggregate measures of health care access around the time of birth. This feature of the data also allows me to consider whether the relationships I examine are "universal", namely the extent to which they hold across diverse populations.

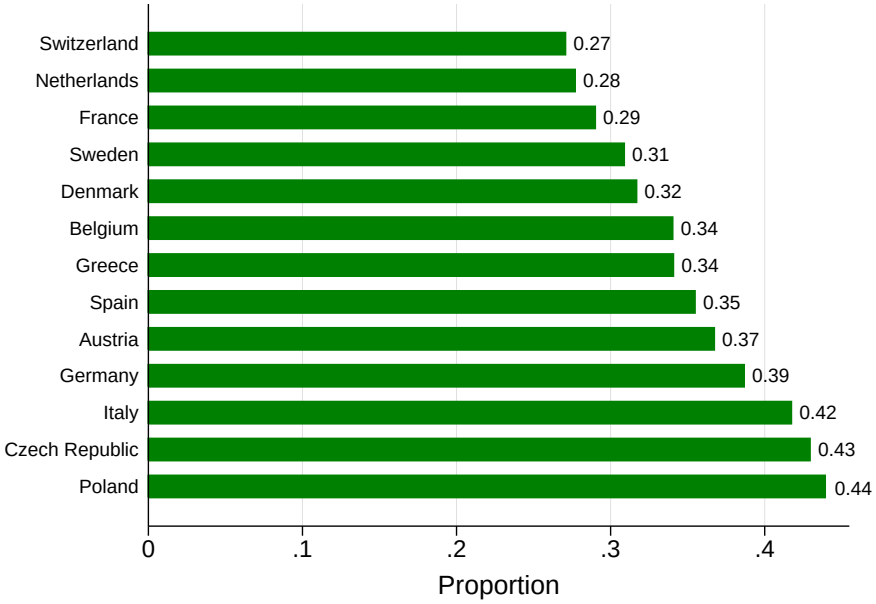
I begin by documenting the prevalence of hypertension. Figure 1 illustrates the proportion of the population in each country who report having been physician diagnosed with high blood pressure. These results are broadly in line with previous studies (Blanchflower and Oswald, 2008). Switzerland has the lowest rate at 27%, while Poland has the highest with 44%. In general, a North-South gradient is apparent, although with some inconsistencies.[3] The fact that the measure of hypertension is self-reported is of concern. The specific question from wave two is: "has your doctor ever told you that you suffer from hypertension/high blood pressure?". Johnston et al. (2009) compare objective and subjective measures of hypertension using the Health Survey for England. They find that self-reported measures underestimate the socioeconomic gradient. However, their subjective measure of hypertension is less robust than that used in SHARE, as in the HSE respondents are asked whether they have any long standing illness. In fact, in terms of prevalence rates, the self-reported measure of hypertension in this data is much closer to the objective measure presented in their study. In addition, in these data a high proportion of individuals (70%) report having regular screening, which indicates that they are likely to have knowledge of their condition. A related concern is that diagnosis may be non-random in the sense that some groups may be less likely to have access to a doctor. However, I have examined whether education is related to the likelihood of having regular blood pressure checks, and there is no evidence that individuals with less education are less likely to undergo these tests. If anything, there is an inverse relationship. There is further evidence that this variable may be reliable as the same question used in SHARE is also asked in the Health and Retirement Study, where self-reports have been found to be accurate (White et al., 2011). Blanchflower and Oswald (2008) find no evidence that cross country differences in hypertension prevalence reflect differences in the number of doctors or hospitals.

Hypertension is related to current socioeconomic status in the data. Figure 2 shows the ratio of hypertension for those with some college education relative those with none, in each country. I define some college education as having left full time education after the age of 18. There is a positive gradient; those with more education have lower hypertension. For example, in France the rate of high blood pressure is 47% higher in the lower education group.[4]

[3] Figure 8 in the appendix documents the average age at which the condition manifested itself. There does not appear to be a clear relationship across countries between age of onset and prevalence (this is confirmed with non-parametric rank order tests).

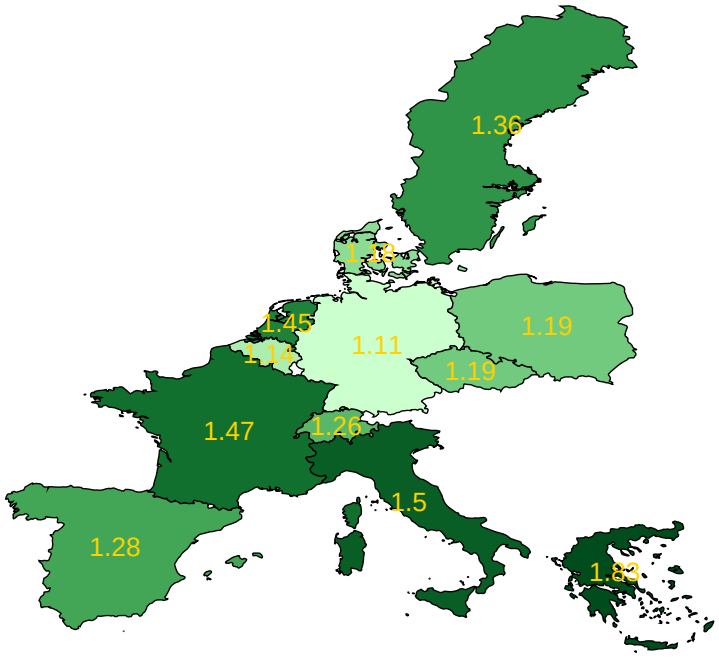
[4] These are raw figures which are not adjusted for other factors.

Figure 1: High Blood Pressure Prevalence Across Europe



Note: Country averages are weighted. The sample refers to those present in wave 2 and 3.

Figure 2: Inequality in High Blood Pressure



Note: Country averages are weighted. The sample refers to those present in wave 2 and 3.

Table 1: Sample Size and Descriptive Statistics

Country Sample Size for W2+W3	No.	%
Austria	709	3.11
Germany	1,674	7.33
Sweden	1,576	6.9
Netherlands	1,854	8.12
Spain	1,662	7.28
Italy	2,328	10.2
France	2,102	9.21
Denmark	1,997	8.75
Greece	2,576	11.28
Switzerland	334	1.46
Belgium	2,534	11.1
Czech Republic	1,789	7.83
Poland	1,699	7.44
Total	22,834	100

Country Sample Size for W1+W2	No.	%
Austria	1,777	4.52
Germany	3,287	8.36
Sweden	3,417	8.69
Netherlands	3,570	9.08
Spain	3,127	7.95
Italy	3,694	9.39
France	3,391	8.62
Denmark	2,953	7.51
Greece	3,730	9.48
Switzerland	1,454	3.7
Belgium	3,839	9.76
Czech Republic	2,695	6.85
Poland	2,392	6.08
Total	39,326	100

Variables for W2+W3 Sample	Median	Mean	SD	N
Wave 2				
Age	63	64.2	9.9	22,834
Male	0	0.4	0.5	22,834
Height(CM)	168	167.8	8.9	22,585
Doctor Said You Had Hypertension	0	0.4	0.5	22,792
Never Smoked	1	0.5	0.5	22,834
Exercise Less than Once a Month	1	0.5	0.5	22,762
BMI	26.2	26.7	4.3	22,371
Drinks 5+ Days a Week	0	0.2	0.4	22,761
Total Amount of Cigarettes (Log)	5.9	5.6	1.3	10,642
Mother Alive	0	0.2	0.4	22,549
Father Alive	0	0.1	0.3	22,715
Mother's Age or Age at Death	79	76.4	12.9	22,223
Father's Age or Age at Death	75	72.1	13.8	22,056
Month of Birth	6	6.3	3.4	22,817
Birth Order: Only Child	0	0.1	0.3	22,719
Birth Order: Eldest	0	0.3	0.5	22,719
Birth Order: In Between	0	0.3	0.5	22,719
Birth Order: Youngest	0	0.3	0.4	22,719
Wave 3				
Childhood Socioeconomic Status	-0.2	0	1	21,863
Work Stress Score	29	29.3	5.6	18,377
Age Finished Fulltime Education	17	16.7	5.5	22,564
Left Fulltime Education Before 19	1	0.7	0.5	22,678
Number of Childhood Illnesses	1	1.2	0.9	22,411
Had Regular Dentist During Childhood	0	0.5	0.5	22,573
Had No GP Between 0-15 Years Old	0	0.1	0.2	22,566
Have Regular Blood Pressure Checks	1	0.7	0.5	22,650
Had Vaccinations During Childhood	1	0.9	0.2	22,374
Macro Variables				
Infant Mortality Rate	71	72.7	34.3	19,819
GDP	4,086	4,111.6	1671	21,314

Note: Data for variables shown refer to those present in wave 2 and 3. The source for infant mortality, measured in deaths per 1,000 live births, is Abouharb and Kimball (2007) and Flora et al., (1987). The source for GDP, measured in millions of 1990 Geary Khamis $, is Maddison (2008).

Hypertension is also associated with various risk factors as previously identified in the literature. The clearest bivariate relationships are with and exercise (whether measured in terms of vigorous or moderate activities) and BMI (body mass index, defined as mass divided by height squared). One drawback of the data is that height and weight are self-reported. SHARE asks respondents directly about stress in various aspects of their lives. Individuals who are retired are asked about the main job of their careers, while those currently employed are asked about their current jobs. There is a clear gradient with respect to work environment.[5] For details, see table 7 in the appendix.

[5]There are 13 available variables that ask about stress in work environment. I avoid picking any one of those particular measures or choosing several, as this approach could result in selection bias or be misleading due to the problems of inference associated with multiple hypothesis testing. I therefore combine all 13 available variables into an index by summing the answer to each. For every answer the value 1 corresponds to "strongly agree", while 4 corresponds to "strongly disagree". The variables have been recoded such that a high score corresponds to an adverse environment; therefore a score of 13 corresponds to the least stressful workplace, while 52 is the most stressful. The distribution of this index is approximately normal. The questions relate to whether work was: physically demanding, involved time pressure, was emotionally demanding, involved conflicts, had freedom to decide, allowed the development of skills, gave recognition, had adequate salary, had adequate support, had good atmosphere, employees were treated fairly, did not increase health risks, and the individual did not sacrifice too much for their job.

3 Hypertension and Early Life Environment

This section considers the empirical relationship between early life conditions and hypertension in greater depth. I begin by taking the data on life cycle events from SHARE wave 3, and combining it with the behavioural and health information from wave 2. A simple conceptual framework for considering an individual's health at time t (in discrete time) can be formulated as follows.

$$\text{Health}_{j,t} = X_{kj}\beta_{1k} + \sum_{i=1,t>1}^{t} Health_{j,t-i}\beta_{2(t-i)} + \sum_{i=1}^{t} Investment_{j,t-i}\beta_{3(t-i)} + \sum_{i=1}^{t} Environment_{j,t-i}\beta_{4(t-i)} + \epsilon_j, \forall t > 0 \quad (1)$$

An indicator of health at time t, for individual j, is firstly given as a function of a vector of individual characteristics (a matrix X, with K columns), which are fixed at birth, such as gender, place of birth and time of birth. Secondly, current health is taken to be determined by the sum of health in previous periods.[6] Thirdly, health is a function of the sum of investments, or behaviours, up to time $t-1$, which either augment, or depreciate, the current stock of health in time t. Investments at time $t = 0$ are assumed to be made by parents. Fourthly, health is a given as a function of the sum of environmental influences, which could either be exogenous changes in policy environment, or the result of individual choices, such as place of residence. Finally, ϵ_j represents individual level frailty, which is unobserved. I distinguish between investments (which affect the individual only), and environment, which affects a particular cohort. This is particularly important to account for when dealing with cross country data, as is the case here. For ease of illustration, I approximate a more complicated health production function with a linear model, however as every past period is weighted ($\sum_{i=1,t>1}^{t} \beta_{2(t-i)}$, $\sum_{i=1}^{t} \beta_{3(t-i)}$, and $\sum_{i=1}^{t} \beta_{4(t-i)}$), this allows for some periods to have a more influential effect on current health than others. I do not attempt to specify complementarities (as in Cunha and Heckman, 2007), as the focus of this paper is on estimating the empirical effects of initial health, and I do not have the data to adopt a more complex framework. See Almond and Currie (2011a) for further discussion.

I mainly consider the relationship between initial conditions and later outcomes, therefore I am primarily concerned with the effects of $Health_{j,t=1}$, $Invest_{j,t=0}$, and $Environment_{j,t=0}$. Seeing as proceeding health behaviours, and at least some component of environmental influence, are likely to be endogenous to the individual (as argued in section 4), I do not control for variables which could potentially represent the outcome of early life conditions. For example, education has been shown to be associated with infant health (Black et al., 2007; McGovern, 2011). Smith (2009) provides evidence for economic outcomes.

In the data, I only observe health as reported up to age 15, therefore I take this as the initial period ($Health_{j,t=1}$). I do not observe initial parental investments directly ($Investment_{j,t=0}$), therefore I control for parental longevity (a measure of genetic endowment and other components of shared family attributes such as parenting behaviour) and childhood socioeconomic status as proxies. Individuals in

[6] Time $t = 0$ could be thought of as the environment in utero, however I do not have the data to disaggregate periods in childhood before age 10-15.

different countries clearly face very different environments throughout their whole lives, and this could be correlated with initial conditions. For example, if less developed countries had poorer early life conditions affecting a particular cohort, but also poorer environments at the time of the second wave of the survey (in 2006), this could bias estimates of the effects of early life conditions. Due to secular trends over time, even within countries earlier cohorts are likely to face less advantageous conditions in later life. Older respondents (who are from earlier birth cohorts) are also clearly at greater risk of hypertension. To proxy for $Environment_{j,t=0}$, I therefore control for region of birth within countries, month of birth, gender and birth order, in addition to a fixed effect for every year of birth cohort in each country. The advantage of this model is that it will account for any factor which is common to the country birth cohort. This includes their current age and any period effects which they encounter in later life, including an event such as World War 2.[7] Estimates of the effects of the variables of interest are now only estimated by within country cohort variation. The addition of fixed effects for place of birth also accounts for the fact that some regions within countries may be more disadvantaged than others. I discuss some limitations of the model below.

Given that I have a single cross section of data with information on early life conditions,[8] for the case of blood pressure this model simplifies to the following (equation 2). It is equivalent to that in equation 1, except I now allow for cross country differences, and use the initial values of each input only (or their proxies, as outlined above). This model is similar to that used in Almond et al. (2012), who examine the influence of disease environment in early life on maternal health.

$$\begin{aligned}\text{HighBloodPressure}_{c,j,t} = X_{kj}\beta_{1k} + SES_{j,t=0}\beta_2 + Health_{j,t=0}\beta_3 \\ + \sum_{p=1}^{2} Endowment_{pj}\beta_{4p} + FE_{c,t-a}\beta_5 + \epsilon_j\end{aligned} \tag{2}$$

The outcome ($HighBloodPressure_{c,j,t}$) for individual j, in country c, at time t, is a function of a set of control variables (again a matrix X with K columns), including place of birth, gender, month of birth and birth order. Hypertension is also given as a function of initial health, initial investments (proxied by SES), and the parental endowment (proxied by parental longevity) of each parent ($p = 1, 2$). As discussed above, I control for environment at the cohort level with a fixed effect for each birth cohort in each country ($FE_{c,t-a}$, where a is the age of the individual at time t), which accounts for any factor which is common to those individuals. The main coefficients of interest are therefore β_2 and β_3.

The measure of childhood socioeconomic status is derived according to the approach adopted in Mazzonna (2011), where a principal components analysis is used to construct an index based on parental occupation, rooms per capita, household facilities, and books in the household. This method has been shown to match up well with patterns in aggregate indicators. It is standardised to have a mean of 0 and standard deviation of 1.

[7] As is well known, with a single cross section of data it is not possible to separately identify the effects of cohort and age. This is not an issue here, as the aim is not separate identification of each, but rather to control for their combined effects, and ensure that they do not bias estimates of the effects of early life conditions. In section 3.2, I use an additional wave of data and control for each simultaneously.

[8] Life histories are collected in wave 3. Physician diagnosed conditions are asked in waves 1 and 2.

There are four main measures of childhood health in the data. I construct the total number of childhood illnesses from a series of questions based on the prevalence of specific diseases. There are small numbers in most categories (making it problematic to identify the effect of one particular illness), which is why I aggregate in this manner. The three other variables are: self-reported health in childhood, whether the individual ever missed school due to health, and height (now commonly used as an indicator of early life conditions, Steckel, 2009). The first three of these are obtained from SHARE wave 3, while height is obtained from the respondent's first interview (in either wave 1 or wave 2). The choice of how to measure childhood health is mainly driven by data availability in the literature. Smith (2009b) uses self-reported childhood health. Case et al. (2005) uses number of illnesses and height in childhood. Case et al. (2002) additionally have information on missing school due to illness along with hospital and bed stays. In the SHARE data these four indicators are not all highly correlated, for example in a regression of height (controlling for country, year of birth and gender) on childhood illnesses the R^2 is .06, .48 for height on bad childhood health, and .52 for height on missing school due to health. Each of these is likely to capture different aspects of health. For example, missing school may be more likely to involve an accident or injury, while differential reference points and diagnosis are a concern for self-reported health status and childhood illnesses. This reinforces the need to control for cohort effects. As discussed above, adding fixed effects implies that regression estimates are only identified by within group comparisons. Without fixed effects, results could be influenced by cross country (and cohort) heterogeneity in reporting thresholds. Apart from height, none of the measures of childhood health show any improvement over time (self-reported childhood health status), and in some cases deteriorate (number of illnesses and missing school due to health). This is perhaps an indication that public health care was getting better at identifying morbidity over time. However, as height is self-reported, this variable is also likely to suffer from measurement error and potential bias due to shrinking, in which case using it as a proxy for early life conditions may underestimate these effects (assuming this measurement error is classical).

Despite these concerns, there is evidence to believe that these retrospective data are reliable (Smith, 2009a; Haas and Bishop, 2010; Havari and Mazzonna, 2011). I present results from each of the four indicators of childhood health in table 2 (using the empirical model outlined in equation 2), and they are consistent, apart from the lack of significance for missing school. This is in line with the view that this variable is more likely to capture temporary injuries, although it is not possible to verify this in these data. I use a linear probability model (the outcome variable is dichotomous), as this approach makes it simpler to incorporate the 600 dummy variables used to control for country year of birth cohorts. However, marginal effects from a probit give similar results. Selective mortality is always a concern with retrospective data, therefore I restrict the sample to those aged 85 and under, due to the fact that cumulative mortality is likely to be substantial above this. Provided mortality occurs first in those individuals who are most affected by poor initial environment, these effects will be an underestimate (van den Berg et al., 2011a).

The number of childhood illnesses, poor self-reported health in childhood, and height, are associated with hypertension. The effect of height is quadratic (the F test for joint significance is 3.01, $p = .050$), with the turning point occurring around 170cm, after this value the marginal effect is positive. Missing school is not significantly associated with high blood pressure. Proxies for health endowments from fathers are significant, for example, an extra year of paternal life is associated with a reduction in the risk of hypertension of around .1 percentage point. Childhood socioeconomic status (SES) is also negatively related to high blood pressure; a unit increase in the index reduces the risk by between 1.2 and 1.3 percentage points. There is little evidence of non-linearity in the effects of SES, as higher order terms

are not significant when included. There is therefore some indication of a direct effect of initial conditions on high blood pressure in later life. For health, this is the case at least as when measured by the number of childhood illnesses, self-rated health, or height. Parental health endowments also appear to have an effect.

In figure 3, I investigate heterogeneity across countries by running the specification in table 2 separately for each nation. I also consider gender differences. I show the coefficient on childhood illnesses and childhood SES for each subgroup. For every country except Sweden, the effect of childhood health is as expected (i.e. worse early life health is related to a greater risk of hypertension as an adult). The effect is largest in Spain, with an extra childhood illness being associated with an increased risk of suffering from hypertension of roughly 3 percentage points. Consistent with the previous literature, men are more likely to be adversely affected by early life health than women. The effect of childhood socioeconomic status is less uniform, and is even positive for some countries.[9] In contrast to the effects of health, women are more affected than men by SES. This is consistent with previous research which finds that men are more vulnerable in a biological sense to poor early life health conditions (Drevenstedt et al., 2008). It is difficult to compare the magnitude of these differences directly with other papers due to differences in the independent variables used, however van den Berg et al. (2011b) also find that men are more adversely affected by early life hunger in terms of hypertension. Although beyond the scope of this paper, future research should identify the factors underlying the country fixed effects.

[9]This could be due to sampling error caused by reduced sample when disaggregating by country. For example, there are only around 300 observations for Switzerland. See table 1 for further details on the sample size.

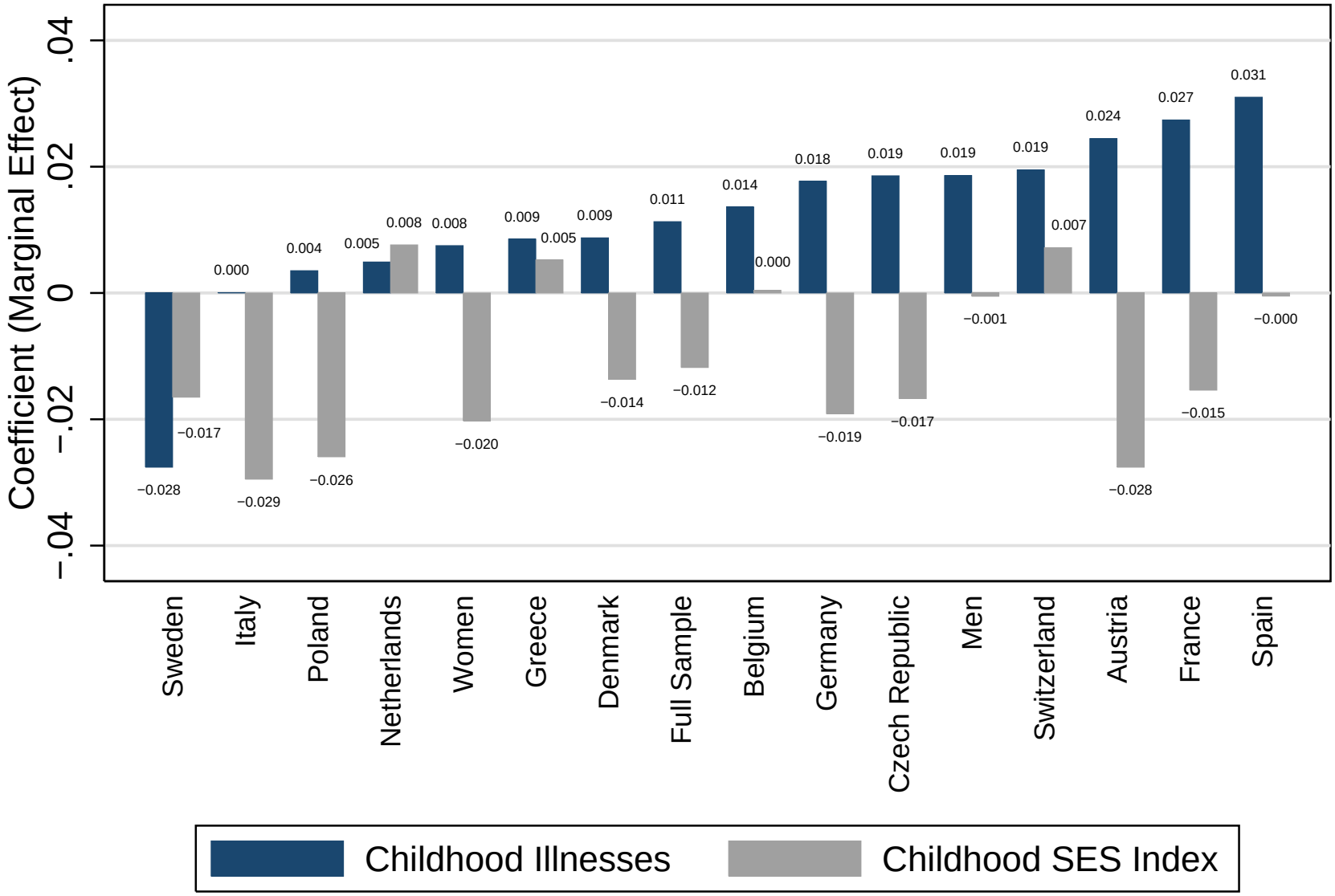
Table 2: Early Life Conditions and Hypertension

Variables	Hypertension	Hypertension	Hypertension	Hypertension
Number of Childhood Illnesses	0.0113**			
	(0.004)			
Missed School for a Month Due to Health		0.0011		
		(0.012)		
Fair Or Poor Childhood Health			0.0277**	
			(0.014)	
Height (CM)				-0.0263**
				(0.011)
Height Squared				0.0001**
				(0.000)
Childhood Socioeconomic Status	-0.0118***	-0.0108***	-0.0111***	-0.0113***
	(0.004)	(0.004)	(0.004)	(0.004)
Female	0.0370***	0.0384***	0.0375***	0.0336***
	(0.007)	(0.007)	(0.007)	(0.010)
Mother's Age or Age at Death	-0.0005*	-0.0005	-0.0005*	-0.0005
	(0.000)	(0.000)	(0.000)	(0.000)
Father's Age or Age at Death	-0.0017***	-0.0017***	-0.0017***	-0.0016***
	(0.000)	(0.000)	(0.000)	(0.000)
Constant	0.4917	0.4637	0.3565	2.4844*
	(1.025)	(1.025)	(1.028)	(1.419)
Cohort Effects:	Country Birth Year FE	Country Birth Year FE	Country Birth Year FE	Country Birth Year FE
Sample	W2+W3	W2+W4	W2+W5	W2+W6
Observations	19,064	19,186	19,226	19,118
R-squared	0.099	0.098	0.098	0.099

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Note: Standard errors are clustered at the country/year of birth level. Hypertension is a binary variable indicating diagnosis by a physician. Dummy variables for each year of birth cohort in each country are also included in the regression, but omitted from the table, as are place of birth fixed effects, controls for whether either of a respondent's parents are still alive, month of birth, and birth order (comprising four categories: only child, eldest, in between, or youngest). Childhood health variables refer to the period up to age 15. The childhood SES index is a standardized variable which is constructed as per Mazzonna (2011). All columns are linear probability estimates. The sample used is those present in wave 2 and 3.

Figure 3: Cross Country Heterogeneity in the Effects of Childhood Circumstance on Hypertension



Note: The sample refers to those present in wave 2 and 3. The specification used is that in table 2 and outlined in equation 2.

3.1 Hypertension and Public Health at Birth

It is important to note that this simple model is not sufficient to identify the causal effect of initial health. As in equation 3, suppose that early life health is determined by a vector of variables (Z), which may be equal to the variables on the right hand side of equation 2 (call them X), include a subset of these such as cohort and gender, or contain additional components. In order to identify the causal effect of initial health (β_3) in equation 2, the covariance between the error terms in the two equations must be 0 (i.e. $Cov(\epsilon 1_j, \epsilon 2_j) = 0$). This is equivalent to the condition that there is no determinant of health which in reality enters into both X and Z, but is not observable in the data. For example, if there is some aspect of childhood socioeconomic status which is not adequately measured, then this will be a source of bias. In this situation, any association between childhood health and adult health in the data could be spurious.

A solution to the identification problem would be to use some factor (Z_k) which is present in Z, but not in X in equation 2 above, which exogenously (i.e. is uncorrelated with $\epsilon 1_j$) shifts initial health. Geographic variation is a one potential source (Almond and Currie, 2011a), and candidates for suitable sources of exogeneity include aspects of public health which are external to the individual.

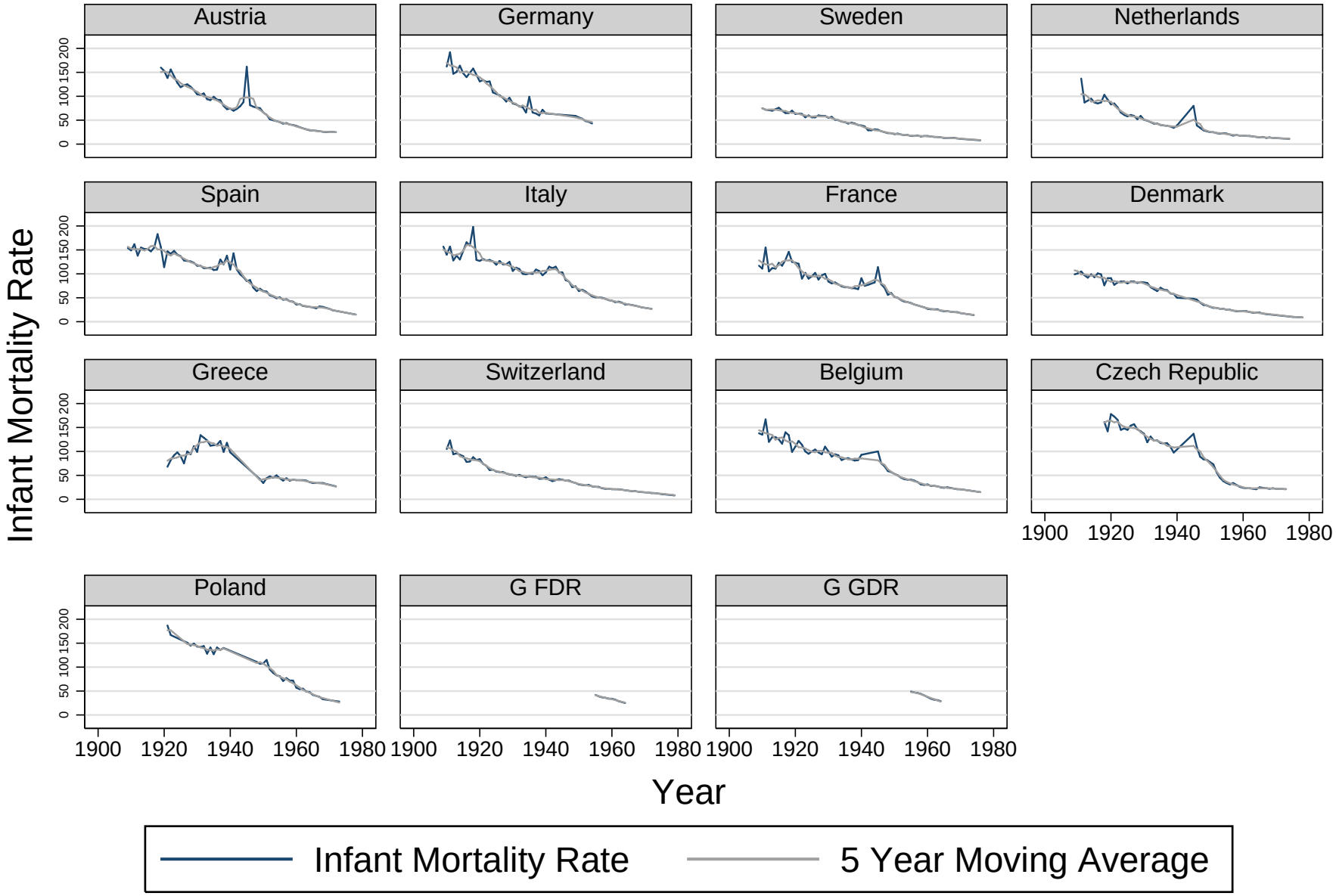
$$\text{Health}_{j,t=0} = Z_i\beta_6 + SES_{j,t=0}\beta_7 + \epsilon 2_j \quad (3)$$

A previous set of papers has used aggregate measures of economic conditions at birth (Portrait et al., 2010; van den Berg et al., 2006; van den Berg et al., 2009a; van den Berg et al., 2009b; Doblhammer et al., 2011; van den Berg et al., 2011a). Here I use country level infant mortality, and other indicators of health care access. This is similar to the approach in Almond et al. (2012). These are plausibly exogenous in the sense that they are unaffected by individual characteristics (i.e. are the same for everyone in a country in a particular year, regardless of SES, or genetic endowment). Infant mortality is likely to be correlated with cohort level characteristics, however. It is of concern, for example, that individuals who experience higher infant mortality are from earlier birth cohorts. Due to secular improvements in (for example) education or economic development, a high level of infant deaths is likely to be associated with other cohort level factors which adversely affect hypertension. It is not possible to follow the strategy adopted in table 2, and include fixed effects for each year of birth cohort in each country, as infant mortality only varies at the country year level. In addition, age and cohort are equivalent with a single wave of data. I therefore add individuals who were interviewed in wave 1 (and not subsequent waves) to the wave 2 sample, which allows me to control for both age and cohort. For this sample, I can no longer control for variables in wave 3 (i.e. place of birth and socioeconomic status), however in table 8 in the appendix I conduct a robustness check where I evaluate the effect of infant mortality in the wave 2 and wave 3 sample used in table 2. Omitting these variables does not alter the effect of infant mortality on hypertension.

I obtain country level infant mortality data from historical records (Abouharb and Kimball, 2007; Flora et al., 1987), and information on access to vaccinations, dental care, and a general practitioner during childhood from wave 3 of the SHARE data. For the latter 3, I aggregate for each country year of birth cohort, and take a 5 year moving average, which is necessary due to sample size in some country year of birth cells. For certain countries which were most affected by World War 2 (such as Germany and Poland), data on infant mortality is missing for these years, therefore individuals born in these countries during the war are omitted from the analysis. I exclude them as these respondents are likely to have suffered exceptional hardship in a manner not captured by the infant mortality rate. In addition, several papers already focus on the effects of World War 2 with SHARE data (Kesternich et al., 2012; Havari and Peracchi, 2011; van den Berg et al., 2011b).

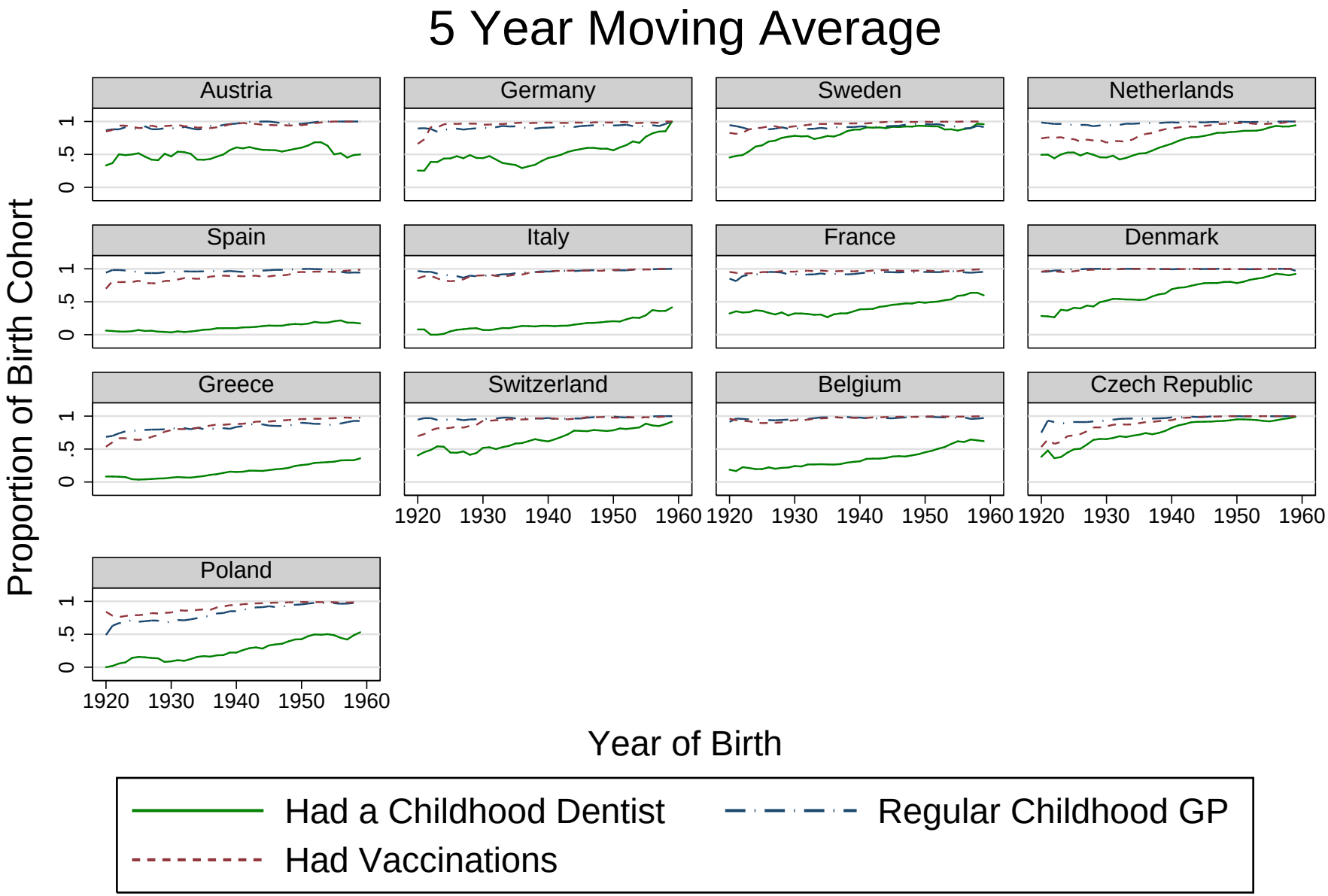
Figures 4 and 5 demonstrate the significant improvements in European public health over time. A great deal of variation across countries is also apparent. Most show a consistent decline in infant mortality; however there are important differences in levels. There is a strong relationship at the aggregate level between infant mortality at time of birth and hypertension prevalence now. It is strongest for the 1936-1940 cohort (figure 6), but is also present for other year of birth groups (figure 9 in the appendix). The average infant mortality rate in the Netherlands for that particular cohort was around 40 deaths per 1,000 live births, and the current rate of high blood pressure is less than 40%. Alternatively, for Poland the infant mortality rate was close to 140, and the rate of high blood pressure is nearly 60%. There is a similar relationship for height (figure 7). These height effects are more convincing, in the sense that maximum height is fixed early on in life, and arguably not affected by subsequent country level confounders (such as the level of economic development).

Figure 4: European Infant Mortality



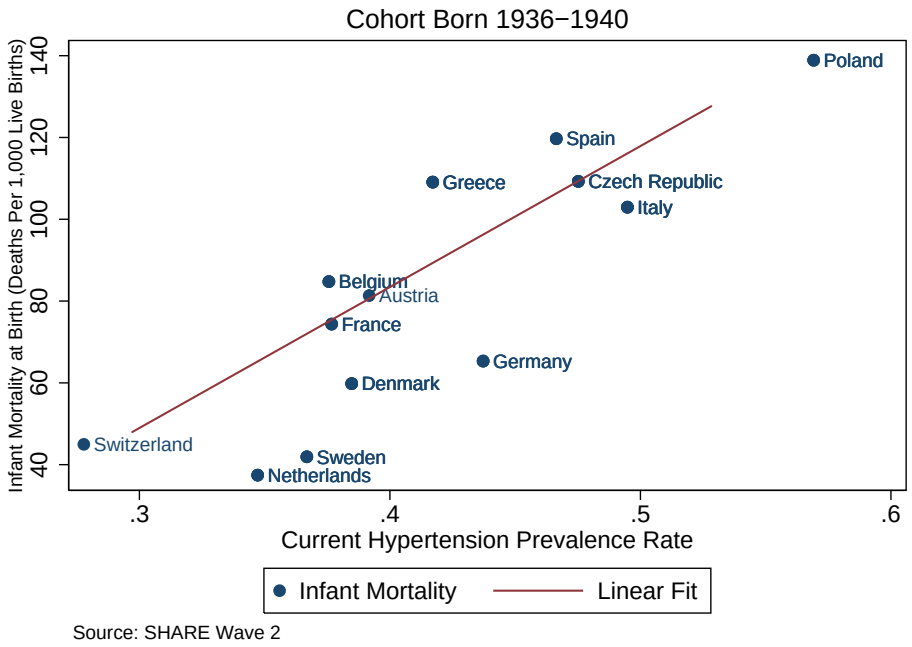
Source: Abouharb and Kimball (2007) and Flora et al. (1987). G GDR and G FDR refer to East and West Germany.

Figure 5: European Public Health Care Access



Source: SHARE Wave 3

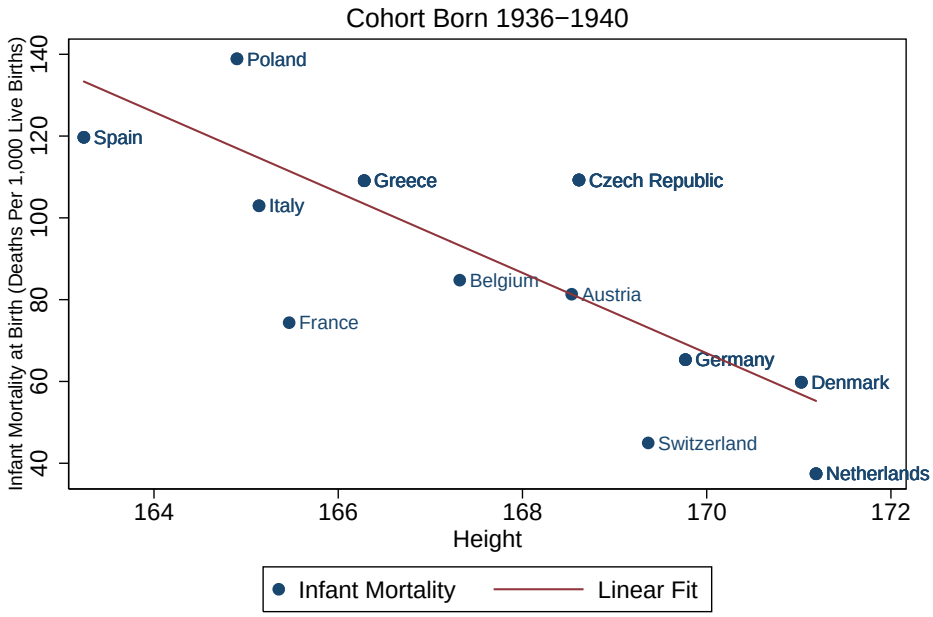
Figure 6: The Long Run Association Between Infant Mortality and High Blood Pressure



Source: SHARE Wave 2

Note: Country averages are weighted. The sample refers to individuals present in waves 2 and 3 of SHARE.

Figure 7: The Long Run Association Between Infant Mortality and Height



Source: SHARE Wave 2

Note: Country averages are weighted. The sample refers to individuals present in waves 2 and 3 of SHARE.

Table 3: Infant Mortality and Hypertension (Without GDP)

Variables	Hypertension	Hypertension	Hypertension
Infant Mortality at Birth	0.0011***	0.0010***	0.0006***
	(0.0003)	(0.0003)	(0.0002)
Cohort Effects:	Country 5 Year Age Group	Country 5 Year Age Group + Country 5 Year Birth Group	Country Age^2 Trend
Sample	W1+W2	W1+W2	W1+W2
Observations	32,115	32,115	32,115
R-squared	0.067	0.070	0.065

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table 4: Infant Mortality and Hypertension (With GDP)

Variables	Hypertension	Hypertension	Hypertension
Infant Mortality at Birth	0.00078**	0.00082**	0.00076***
	(0.00030)	(0.00033)	(0.00020)
GDP	-0.00002***	-0.00001	0.00001**
	(0.00001)	(0.00001)	(0.00001)
Cohort Effects:	Country 5 Year Age Group	Country 5 Year Age Group + Country 5 Year Birth Group	Country Age^2 Trend
Sample	W1+W2	W1+W2	W1+W2
Observations	31,313	31,313	31,313
R-squared	0.067	0.069	0.065

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Note: Standard errors are clustered at the country/year of birth level. In addition to cohort effects, control variables are gender, parents' age or age at death, parent is alive, month of birth, birth order (comprising four categories: only child, eldest, in between, or youngest) and survey year. Infant mortality data (number of deaths per 1,000 live births in a respondent's country of birth) is taken from Abouharb and Kimball (2007) and Flora et al. (1987). GDP data is taken from Maddison (2008). All columns are linear probability model estimates. The sample refers to those present in wave 1 or wave 2.

Table 3 presents results where infant mortality at birth is included as the childhood health measure. Adding individuals interviewed in wave 1, to those interviewed in wave 2, which allows me to control for both age and cohort. I also control for survey year.[10] The data now contain individuals who experienced the same birth conditions, but were different ages at the time of survey. I only use one observation per individual (the latest interview), as panel data does not help with identification in this case (there being

[10]These coefficients are identified for two reasons. Firstly, year effects are not country specific, and secondly, age effects are in 5 year groups (as in Doblhammer et al., 2011).

no within person variation in early life conditions). I am unable to control for the variables from wave 3 (namely childhood SES and place of birth), however as outlined above, this does not seem to affect estimates (table 8 in the appendix). Other control variables are the same as table 2 (gender, month of birth and birth order, controls for whether either of a respondent's parents are still alive, and parents' age or age at death). The sample is also restricted to those born in the country of interview.

I begin by controlling for each 5 year age group in each country. In addition to accounting for average differences in hypertension prevalence across countries, this approach allows for differential age effects by country. A unit increase in infant mortality at birth is associated with an increase in the risk of hypertension of .11 percentage points. I add controls for the 5 year country birth cohort, which accounts for their shared environment. This includes, for example, the education system for that particular cohort, both at birth and in later life. The estimated effect reduces to .1 percentage point but is still significant. Given that infant mortality in Europe fell from an average of 90 deaths per 1,000 live births in 1931-1935 to 35 deaths per 1,000 live births in 1956-1960, these are important effects. The improvements in early life conditions over the period corresponds to a total reduction in the rate of high blood pressure of around 6 percentage points. Delaney et al. (2011) examine the consequences of a natural experiment in Ireland, and find that changes in infant mortality similarly affected disability rates 50 years later. In the final column, I adopt the approach used in that paper, by allowing for a quadratic age term for each country. The effect size is reduced to .06 percentage points, but is still significant. This estimate corresponds to a reduction of around 3 percentage points in the hypertension rate over the same time period. As a further robustness check, I add GDP at birth to the model in table 4. The estimates of the effects of infant mortality are very similar. This is not surprising, as improvements in childhood conditions in the early part of the 20th century have been linked to changes in sanitation and clean water rather than income (Cutler and Meara, 2004; Cutler and Miller, 2005; Delaney et al., 2011). Furthermore, van den Berg et al. (2009a), and van den Berg et al. (2009b), find no relationship between infant mortality and the business cycle. When 5 year cohort fixed effects are used in the model, the variation in infant mortality is restricted to deviations from the average for that birth cohort in a particular country. In contrast, for the final model, the coefficients are identified only using deviations from a quadratic trend for that particular country. There is little difference in the results when GDP is added, however they do become slightly more uniform across the three models. As GDP contains a trend component, its addition may have a similar effect to adding the country trend in model 3. Controlling for a country specific trend in this manner is likely to be beneficial as it provides an additional check for omitted variables which could be improving health outcomes over time.

Table 9 in the appendix presents equivalent models for the other public health measures. In this case results are generally not significant for women, therefore I restrict attention to men. Results are also not significant for the quadratic specification, or for GP access. Seeing as these variables are estimated from the data, and also require averaging, they may be subject to measurement error. It is likely that infant mortality more accurately reflects the effects of early life conditions because of this. I present bootstrapped standard errors to account for the uncertainty involved in the estimation of these averages as analytic standard errors are incorrect in these circumstances (Murphy and Topel, 1985). Adding GDP has little effect on these estimates.

There are potential limitations to this approach of using aggregate level indicators of early life health, which while exogenous to the individual, may not be exogenous to the cohort. A drawback of using data from multiple countries is that it is more difficult to conclusively tie changes in infant mortality to exogenous events, as in Delaney et al. (2011). Infant mortality could be correlated with other factors at the time of birth, for example economic development. Infant mortality could also be correlated with environment in later life, although adding fixed effects for 5 year cohorts in each country means that these shared experiences will be controlled for. The omitted factor would have to vary with infant mortality within these 5 year cohorts to bias results, and additionally be unrelated to GDP, which I also control for. Alternatively, the composition of cohorts may differ according to initial environment. For example, a potential threat to identification is that families may anticipate the infant death rate. If this is the case, then cohorts born during times of high mortality may differ from cohorts born during times of low mortality, due to selective fertility. However, as is clear from figure 4, apart from World War 2 there is a strong downward trend in infant mortality in every country. In addition, many of the advances in early life mortality in developed countries have been linked to exogenous changes in medical technology or public health policies (Costa and Kahn, 2006; Cutler and Meara, 2004; Cutler and Miller, 2005; Delaney et al., 2011), which seem unlikely to have been anticipated. Previous literature which has used aggregate indicators has not found any evidence for selective fertility (van den Berg et al., 2006; van den Berg et al., 2011a). The specification where I control for country age trends has the benefit of only estimating the impact of infant mortality using deviations from this quadratic trend. Even if it were possible to extrapolate infant mortality in period $t+1$ based on previous trends, it seems even less likely that these deviations could have been anticipated. In table 12 in the appendix, I regress childhood SES on the infant mortality rate, and find no evidence for selection in these data. However, by definition it is not possible to definitively prove that there is no selection on unobservables.

In summary, I conclude that there is evidence in favour of an association between early life environment and hypertension, even accounting for omitted variable bias by using aggregate measures of population morality at birth, controlling for cohort effects, and accounting for GDP. For males, there is some evidence of an effect of public health care access around the time of birth. Having established the presence of a direct relationship between initial conditions and high blood pressure, I now turn to a more indirect pathway, namely the issue of risk factors and selection.

4 Initial Endowments and Selection

4.1 Theoretical Framework

Grossman's seminal work (e.g. Grossman, 1972) on defining health as an investment good provides an important tool for understanding health behaviour, and an analytical framework for describing the processes which lead individuals to either build up or wear down their stock of health. In this setup, health is not assumed to be some exogenous external force, but rather a form of capital which evolves endogenously according to the decisions of the individual. Consumption decisions, such as smoking or choice of diet, are examples of processes which have the potential to adversely affect health, while exercise or preventative actions can been seen as a form of health investment.

A standard way of analysing behaviour across the life cycle is the permanent income/life cycle hypothesis. Originally developed to augment our understanding of saving and consumption patterns, the basic premise is simple. Individuals are assumed to maximise total lifetime utility, subject to their lifetime budget constraint. In the case of continuous time, this translates into a constrained optimisation problem, namely the maximisation of:

$$\text{Lifetime Utility}=\int_0^T U(Z(t),H(t))dt \quad (4)$$

Lifetime utility corresponds to the sum of utility in each period (some function U(.), where utility is discounted to take account of the fact that utility in the future is likely to be less highly valued than utility today). Welfare depends on consumption ($Z(t)$, some aggregate good), and a person's stock of health, $H(t)$. This maximisation problem covers the period from birth ($t = 0$) until death ($t = T$). This model can be extended to incorporate health as an endogenous individual characteristic (i.e. a stock which can either be run down through the purchase of consumption goods, or augmented through investments in health capital). The original formulation by Grossman (1972) viewed longevity as an indirect outcome of the stock of health capital, but did not develop the terminal conditions required to analyse this problem in discrete time. These transversality conditions are required in order to ensure that consistent optimal paths for stock variables exist. Ehrlich and Chuma (1990) provide a framework whereby death occurs once health drops below a certain minimum level. A conclusion of their model is that demand for health depends on the initial and terminal conditions, and that optimal levels of health and choice of longevity depend on initial endowments, rather than just current circumstance.[11] These findings are summarised in table 5. One implication is that only considering an individual's current environment could provide misleading results, given that agents' optimal path of health investment may already have been set in childhood or before. This analysis provides a theoretical basis for expecting that individuals with poor early life conditions may be selected into behaviours and environments which have adverse health consequences. It is important to test this prediction empirically in order to be able to assess the validity of the literature which finds a relationship between work place stress (for example) and health outcomes.

[11] A complimentary way of framing this issue is in terms of non-cognitive abilities, as in Conti and Heckman (2010). For example, these attributes could be incorporated into the model via the discount rate.

According to this model of rational decision making, longevity, the stock of health capital, and health investment in any given periods are all expected to be positively correlated with initial health and initial wealth. In other words, individuals who experienced poor early life health or childhood SES are expected to have a lower level of demand for health in later life than those who experienced good early life health or SES.[12]

Table 5: Theoretical Predictions for Selection and Initial Conditions (Ehrlich and Chuma (1990))

	Variable	
	H0 (Initial Health)	A0 (Initial Assets)
Parameter		
T (Longevity)	+	+
H (Health Capital)	+	+
I (Health Investment)	+	+

In the following section I consider the relationship between initial conditions and cardiovascular risk factors from an empirical point of view.

4.2 Early Life Conditions and Cardiovascular Risk Factors

I examine the main risk factors for cardiovascular disease which have been identified in the literature, namely alcohol and tobacco consumption, BMI and exercise, and work place stress. I use the following variables in the analysis. "Drinking to excess" takes the value 1 if individuals report drinking 5 or more days a week, and zero otherwise. "No regular exercise" takes the value one if individuals report exercising less frequently than once a month, and zero otherwise. BMI is constructed from self-reported weight and height (and is therefore likely to be measured with error). The amount of cigarettes an individual has consumed is in logs, and is constructed from information on how long the person smoked, and the quantity. I adopt a model to account for both participation and intensity. An alternative would be to estimate a selection model, however this would require a valid exclusion restriction (a variable which affects participation but not intensity), and hurdle models have been shown to provide reliable estimates (Madden, 2008). Hurdle models are implemented with the participation equation in the first stage and a truncated regression model in the second (McDowell, 2003). Work stress is measured using 13 questions on work place environment, where respondents are asked to rate their careers overall (if they are retired), and the main job of their career if they are still employed. I again do not implement a selection model for work stress (those who never worked are excluded from the regression) due to the difficulties associated with finding an appropriate exclusion restriction. As childhood health adversely affects labour force participation (Smith, 2009), these results can therefore be seen as an underestimate. I use the sample

[12]One other interesting feature of the model is the prediction that reductions in the cost of health investments may in fact accentuate differences between initial health endowments and longevity. It has been established as an empirical reality that gaps in life expectancy by socioeconomic status are rising over time, at least in the US (Meara et al., 2008; Cutler et al., 2010; Montez et al., 2011). This is at odds with declining costs of interventions, and higher rates of preventable disease amongst the less educated. See Cutler and Lleras-Muney (2010) for a discussion. One explanation for this puzzle, which is consistent with this framework, is the potential for these groups to have different demand for health. Whereas an improvement in medical treatment could result in lower mortality rates for a high SES group, for the low SES group these advances could be used to "finance" higher levels of consumption. The variance in life expectancy across population groups over time and geography suggests that there may be some element of demand involved, and all that is required to view longevity as endogenous is the existence of some technology which converts economic resources into health capital (Ehrlich and Chuma, 1990). Individuals constantly face choices which affect health, and these often involve some sort of trade off, generally between consumption today and health at a later date. Eating high calorie food provides utility, but has a negative effect on health; exercise augments an individual's stock of health capital, but may involve sacrificing consumption opportunities. It therefore seems reasonable to consider the theoretical background to these choices.

from wave 2 and wave 3 of the data, so as to be able to determine the effects of childhood socioeconomic status. I use the same specification as outlined in section 3, namely I include a fixed effect for each cohort in every country. This analysis is presented in table 6. As before, I use a linear probability model due to the inclusion of the multitude of cohort controls.

Childhood illnesses are associated with more regular exercise and a more stressful work environment. The effect on exercise is non-linear, after two illnesses the marginal effect becomes positive. The F value for the joint test of significance of the quadratic term is 4.30 ($p = .014$). Childhood SES is associated with lower BMI, fewer total cigarettes consumed, and a less stressful work environment. The effect here is also quadratic ($F = 109.76$, $p = .000$), although as the turning point occurs around 4 the marginal effect is negative for the vast majority of the sample. Childhood SES is also (perhaps surprisingly) associated with an increased risk of drinking to excess and ever smoking. This is perhaps indicative of an income effect. Table 13 in the appendix presents results for height instead of childhood illnesses. The effects of height are more complementary to SES. Overall, there is clear evidence for a relationship between initial environment and cardiovascular risk factors. However, the association appears to vary according to the indicator used.

One interpretation of these results is that there may be no direct association between childhood conditions and these variables, and rather that these effects are operating entirely through circumstance in adulthood, such as health or adult socioeconomic status. I have also estimated models where I control for education, household income, the ability to make ends meet, and self-rated health, and the results for the childhood variables are not very much affected by their addition.[13] As I have argued throughout this paper that caution must be taken when controlling for variables which partly represent the outcomes of early environment, these estimates could be biased due to endogeneity. Nevertheless, they provide some indication that the effects in table 6 are not operating solely through adult circumstance. They also provide confirmation that the work stress variable is not simply picking up the effects of adult income or SES. Finally, these results are consistent with Conti and Heckman (2010), who find that half of the education gradient in a number of adult health outcomes can be explained by selection on early life conditions.

5 Conclusions

Early life conditions have been shown to have an effect on cardiovascular outcomes such as hypertension, although the effects of individual level characteristics may be affected by omitted variable bias. Relatedly, an established literature links factors such as BMI and work place environment to the risk of heart disease. However, theory suggests that individuals may be selecting into adverse environments and behaviours on the basis of their initial conditions. This has implications for our understanding of the causal relationship between the current environment and current outcomes, as both may be affected by early life conditions. Despite this, controlling for variables which could lie on the causal pathway from initial conditions to adult outcomes is common in the literature (Huxley et al., 2002).

I use data from the Survey of Health, Ageing and Retirement in Europe (SHARE) to examine the effect of early life conditions on high blood pressure in 13 countries. I find a significant relationship between childhood health and childhood SES on the presence of high blood pressure as an adult. To account

[13] These results are available on request.

for potential omitted variable bias, I use aggregate measures of population health which are not affected by individual level characteristics. I find no evidence of selective fertility, at least as when measured by socioeconomic status. Results suggest that a unit in increase in the infant mortality rate at birth increases the risk of hypertension by between .06 and .11 percentage points. When I control for cohort fixed effects and GDP at birth, there is little change in these estimates. This is consistent with the view that improvements in early life conditions were driven by changes in sanitation and public health practice, rather than income. These are important effects given the scale of the declines in infant mortality in Europe over the past century, and imply an overall reduction in the hypertension rate of between 3 and 6 percentage points, for the cohort born 1931-1935, relative to the cohort born 1956-1960. Childhood background is also related to adverse lifestyle outcomes, particularly BMI and work place stress. As there is support in the data in favour of selection on the basis of initial conditions, caution should be exercised when controlling for contemporaneous variables such as work place environment. This paper demonstrates that participation in certain adult outcomes, which have been shown to be risk factors for cardiovascular disease, can be predicted from a person's early life environment. Selection may therefore be an important source of bias in studies which incorporate these factors alongside measures of initial conditions, as these should also be interpreted as outcomes of early life disadvantage.

Results in this paper have potentially important policy implications in that the data imply that efforts aimed at reducing health inequalities across the life cycle may have the added benefit of contributing to the reduction of hypertension prevalence, both through direct biological and indirect behavioural pathways. In terms of future research, more attention should be paid to mediating behaviours, and the development of structural models which take account of the complex pathways which emerge from adverse early life conditions. Future research should also continue to investigate the causal relationship between childhood health and adult outcomes. The use of country level infant mortality in this paper facilitates cross country estimates of the effects of early life conditions using standardised data, and nationally representative samples. However, identification in this paper relies on cohort fixed effects, and controlling for the level of economic development. An ideal alternative would be to use public health measures at smaller geographic units (i.e. within countries), linked to changes in policy or infrastructure, which would be exogenous to the cohort, and not just the individual. Currently this is not possible on a Europe wide basis due to data limitations; however efforts drawing on historical records should continue, in order to catalogue the life cycle experiences of Europeans across the continent.

Table 6: Early Life Conditions and Cardiovascular Risk Factors

Variables	Drink to Excess	No Regular Exercise	BMI	Amount of Cigarettes	Never Smoked	Work Quality Score
Number of Childhood Illnesses	0.0059	-0.0296***	0.0333	-0.0059	-0.0067	0.3001***
	(0.018)	(0.010)	(0.039)	(0.017)	(0.004)	(0.054)
Childhood Illnesses Squared		0.0078***				
		(0.003)				
Childhood Socioeconomic Status	0.2010***	-0.0077*	-0.4689***	-0.0708***	-0.0289***	-0.8561***
	(0.019)	(0.004)	(0.037)	(0.015)	(0.004)	(0.061)
Childhood Socioeconomic SQ						0.1154***
						(0.033)
Female	-1.3282***	0.0766***	-0.2729***	-0.4876***	0.2830***	0.0918
	(0.043)	(0.008)	(0.070)	(0.032)	(0.010)	(0.089)
Mother's Age or Age at Death	0.0044***	-0.0008***	-0.0083***	-0.0018	0.0001	-0.0111***
	(0.001)	(0.000)	(0.003)	(0.001)	(0.000)	(0.004)
Father's Age or Age at Death	0.0015	-0.0005*	-0.0079***	-0.0031***	0.0014***	-0.0011
	(0.001)	(0.000)	(0.002)	(0.001)	(0.000)	(0.004)
Constant	-1.5004	0.6787***	29.1572***	2.2778	-0.1066	36.3540***
	(4.686)	(0.049)	(10.321)	(4.603)	(1.149)	(13.035)
Cohort Effects:	Country Birth Year FE	Country Birth Year FE	Country Birth Year FE	Country Birth Year FE	Country Birth Year FE	Country Birth Year FE
Sample	W2+W3	W2+W3	W2+W3	W2+W3	W2+W3	W2+W3
Observations	19,043	19,040	18,819	8,998	19,083	15,617
R-squared	0.262	0.150	0.095		0.172	0.211

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Note: Standard errors are clustered at the country/year of birth level. Dummy variables for each year of birth cohort in each country are also included in the regression, but omitted from the table, as are place of birth fixed effects, controls for whether a either of a respondent's parents are still alive, month of birth, and birth order (comprising four categories: only child, eldest, in between, or youngest). Childhood health variables refer to the period up to age 15. The childhood SES index is a standardized variable which is constructed as per Mazzonna (2011). Drink to excess (binary) represents drinking 5 or more days a week. No regular exercise (binary) entails moderate exercise less than once a month. Amount of cigarettes is measured in logs, and excludes those who never smoked. The work quality score combines 13 questions on work environment, with 13 representing the least stressful and 52 the most stressful. It excludes those who never worked. The sample refers to individuals present in waves 2 and 3. All columns are linear probability estimates apart from amount of cigarettes which is a truncated regression.

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Appendix

Table 7: Hypertension and Risk Factors

BMI	Hypertension Rate
Standard	0.24
Overweight	0.37
Obese	0.52
Total	0.35

Years Smoking	
None	0.37
1-10	0.33
11-20	0.34
21-30	0.32
31-40	0.31
40+	0.35
Total	0.35

Days a week consumed alcohol last 3 months	
Almost every day	0.41
Five or six days a week	0.37
Three or four days a week	0.34
Once or twice a week	0.3
Once or twice a month	0.29
Less than once a month	0.32
Not at all in the last 6 months	0.33
Total	0.35

Activities requiring a moderate level of energy	Hypertension Rate
More than once a week	0.33
Once a week	0.36
One to three times a month	0.41
Hardly ever, or never	0.44
Total	0.35

Work Stress Score Quintile	
Lowest Stress Quintile	0.31
Second Stress Quintile	0.33
Third Stress Quintile	0.34
Highest Stress Quintile	0.37
Total	0.34

Note: The sample refers to individuals present in waves 2 and 3 of SHARE.

Table 8: The Effects of Infant Mortality in Different Samples

Variables	Hypertension	Hypertension	Hypertension
Infant Mortality at Birth	0.0011***	0.0012***	0.0011***
	(0.0004)	(0.0004)	(0.0003)
Cohort Effects:	Country 5 Year Age Group	Country 5 Year Age Group	Country 5 Year Age Group
Controls:	W2+ W3	W1+ W2	W1+ W2
Sample	W2+W3	W2+W3	W1+W2
Observations	16,661	16,661	32,115
R-squared	0.084	0.072	0.067

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Note: Standard errors are clustered at the country/year of birth level. In addition to cohort effects, control variables are gender, parents' age or age at death, parent is alive, month of birth and birth order (comprising four categories: only child, eldest, in between, or youngest), and survey year. Column one additionally controls for childhood SES and place of birth. Infant mortality data is taken from Abouharb and Kimball (2007) and Flora et al. (1987). The variable is the number of deaths per 1,000 live births in a respondent's country of birth. All columns are linear probability model estimates. Columns 1 and 2 refer to the sample of those present in wave 2 and wave 3. Column 3 refers to those present in waves 1 or 2.

Table 9: Childhood Vaccinations and Hypertension

Variables	Hypertension	Hypertension	Hypertension
Vaccinations	-0.4284*	-0.6612**	-0.3141
	(0.2208)	(0.282)	(0.1939)
Cohort Effects:	Country 5 Year Age Group	Country 5 Year Age Group + Country 5 Year Birth Group	Country Age Trend
Gender:	Male	Male	Male
Sample	W1+W2	W1+W2	W1+W2
Observations	16,270	16,270	16,270
R-squared	0.047	0.054	0.046

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Note: Standard errors are clustered at the country/year of birth level. Regressions shown are for males only. In addition to cohort effects, control variables are parents' age or age at death, parent is alive, month of birth and birth order (comprising four categories: only child, eldest, in between, or youngest), and survey year. Vaccination refers to the proportion of the respondent's country birth cohort who had vaccinations (5 year moving average). It is derived from SHARE wave 3. All columns are linear probability model estimates. The sample refers to those present in waves 1 and wave 2.

Table 10: Childhood Dentist and Hypertension

Variables	Hypertension	Hypertension	Hypertension
Childhood Dentist	-0.3287***	-0.3587**	-0.1104
	(0.1101)	(0.1495)	(0.096)
Cohort Effects:	Country 5 Year Age Group	Country 5 Year Age Group + Country 5 Year Birth Group	Country Age Trend
Gender:	Male	Male	Male
Sample	W1+W2	W1+W2	W1+W2
Observations	16,270	16,270	16,270
R-squared	0.048	0.054	0.046

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Note: Standard errors are clustered at the country/year of birth level. Regressions shown are for males only. In addition to cohort effects, control variables are parents' age or age at death, parent is alive, month of birth and birth order (comprising four categories: only child, eldest, in between, or youngest), and survey year. Childhood dentist refers to the proportion of the respondent's country birth cohort who had a regular childhood dentist (5 year moving average). It is derived from SHARE wave 3. All columns are linear probability model estimates. The sample refers to those present in waves 1 and wave 2.

Table 11: Childhood GP and Hypertension

Variables	Hypertension	Hypertension	Hypertension
Childhood GP	-0.2449	-0.3906*	-0.1038
	(0.2948)	(0.3972)	(0.2464)
Cohort Effects:	Country 5 Year Age Group	Country 5 Year Age Group + Country 5 Year Birth Group	Country Age Trend
Gender:	Male	Male	Male
Sample	W1+W2	W1+W2	W1+W2
Observations	16,270	16,270	16,270
R-squared	0.047	0.054	0.046

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Note: Standard errors are clustered at the country/year of birth level. Regressions shown are for males only. In addition to cohort effects, control variables are parents' age or age at death, parent is alive, month of birth and birth order (comprising four categories: only child, eldest, in between, or youngest), and survey year. Childhood GP refers to the proportion of the respondent's country birth cohort who had a regular childhood GP (5 year moving average). It is derived from SHARE wave 3. All columns are linear probability model estimates. The sample refers to those present in waves 1 and wave 2.

Table 12: Infant Mortality and Selection

Variables	Childhood SES
Infant Mortality at Birth	-0.0012
	(0.001)
Female	-0.0031
	(0.015)
Mother's Age or Age at Death	0.0035***
	(0.001)
Father's Age or Age at Death	0.0011**
	(0.001)
Constant	0.2326
	(0.208)
Cohort Effects:	Country 5 Year Age Group
Sample	W2+W3
Observations	16,678
R-squared	0.183

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Note: Standard errors are clustered at the country/year of birth level. In addition to cohort effects, control variables are the same as table 3. Infant mortality data is taken from Abouharb and Kimball (2007) and Flora et al. (1987). The variable is the number of deaths per 1,000 live births in a respondent's country of birth. The childhood SES index is a standardized variable which is constructed as per Mazzonna (2011). All columns are linear probability model estimates. The sample refers to those present in wave 2 or wave 3.

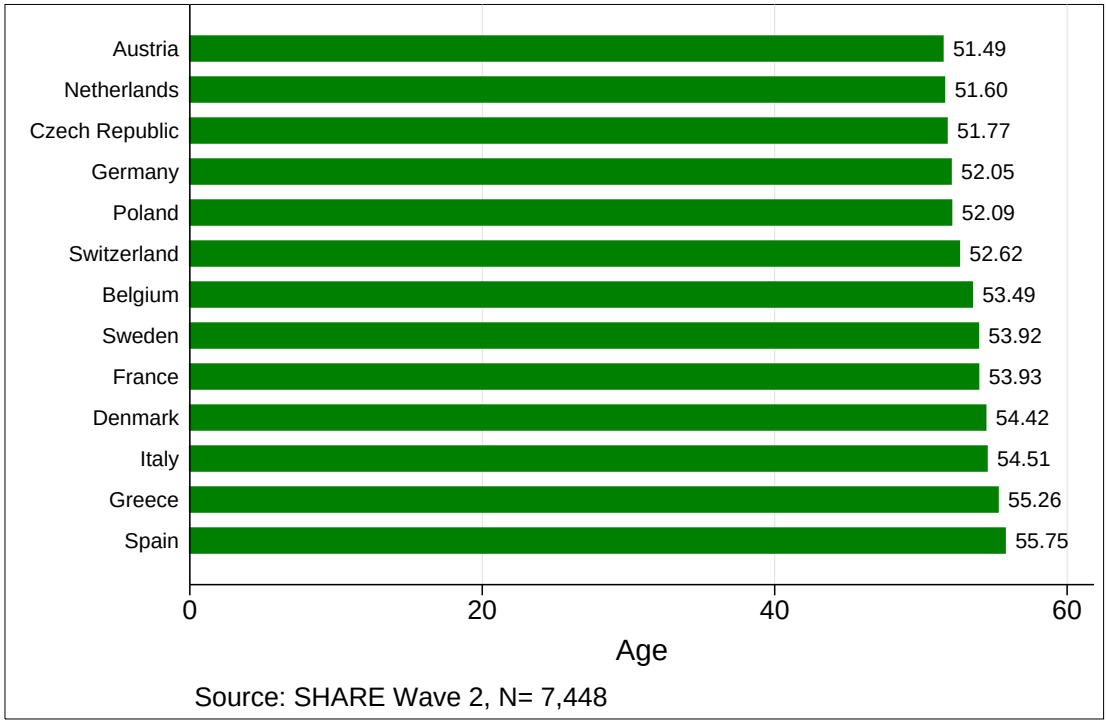
Table 13: Early Life Conditions (Height) and Cardiovascular Risk Factors

Variables	Drink to Excess	No Regular Exercise	BMI	Amount of Cigarettes	Never Smoked	Work Quality Score
Height (CM)	0.0276***	-0.0012*	-0.6697***	0.1313***	-0.0776***	-0.0222***
	(0.010)	(0.001)	(0.116)	(0.044)	(0.012)	(0.007)
Height Squared	-0.0001***		0.0018***	-0.0004***	0.0002***	
	(0.000)		(0.000)	(0.000)	(0.000)	
Childhood Socioeconomic Status	0.0258***	-0.0070*	-0.4133***	-0.0682***	-0.0303***	-0.8244***
	(0.004)	(0.004)	(0.037)	(0.014)	(0.004)	(0.062)
Childhood Socioeconomic Status SQ						0.1193***
						(0.032)
Female	-0.2043***	0.0619***	-1.0410***	-0.5269***	0.2954***	-0.1557
	(0.010)	(0.010)	(0.094)	(0.040)	(0.012)	(0.121)
Mother's Age or Age at Death	0.0003	-0.0008***	-0.0074***	-0.0016	0.0002	-0.0119***
	(0.000)	(0.000)	(0.003)	(0.001)	(0.000)	(0.004)
Father's Age or Age at Death	-0.0001	-0.0006**	-0.0074***	-0.0032***	0.0015***	-0.0015
	(0.000)	(0.000)	(0.002)	(0.001)	(0.000)	(0.004)
Constant	-2.8708**	0.1850	87.5932***	-10.2854*	5.9862***	38.0566***
	(1.191)	(1.063)	(13.840)	(5.879)	(1.490)	(13.297)
Cohort Effects:	Country Birth Year FE	Country Birth Year FE	Country Birth Year FE	Country Birth Year FE	Country Birth Year FE	Country Birth Year FE
Sample	W2+W3	W2+W3	W2+W3	W2+W3	W2+W3	W2+W3
Observations	19,097	19,095	18,976	9,033	19,136	15,662
R-squared	0.186	0.150	0.105		0.173	0.210

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

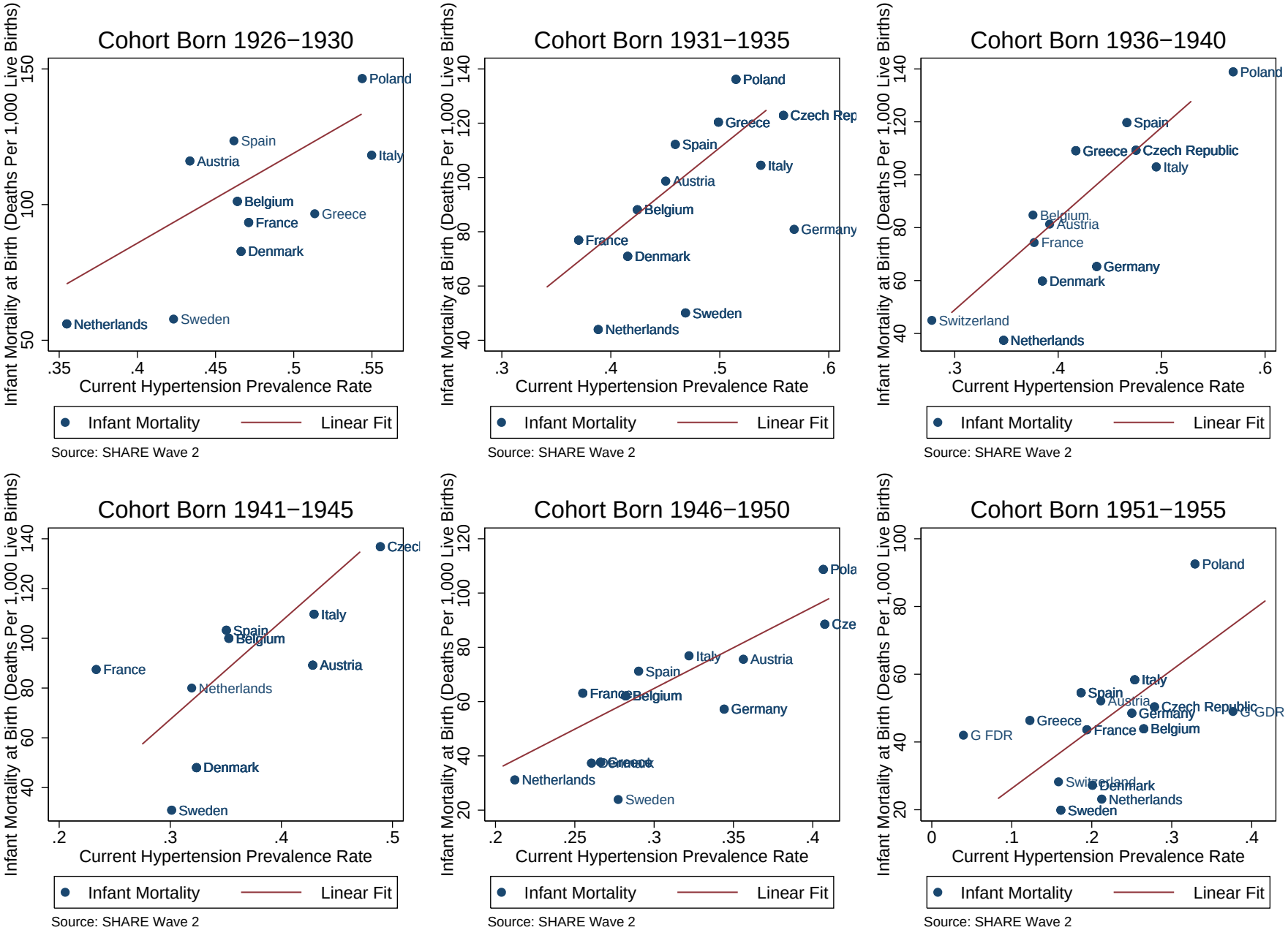
Note: Standard errors are clustered at the country/year of birth level. Dummy variables for each year of birth cohort in each country are also included in the regression but omitted from the table, as are place of birth fixed effects, controls for whether a either of a respondent's parents are still alive, month of birth, and birth order (comprising four categories: only child, eldest, in between, or youngest). The childhood SES index is a standardized variable which is constructed as per Mazzonna (2011). Drink to excess (binary) represents drinking 5 or more days a week. No regular exercise (binary) entails moderate exercise less than once a month. Amount of cigarettes is measured in logs, and excludes those who never smoked. The work quality score combines 13 questions on work environment, with 13 representing the least stressful and 52 the most stressful. It excludes those who never worked. The sample refers to those present in waves 2 and 3. All columns are linear probability estimates apart from amount of cigarettes which is a truncated regression.

Figure 8: Age of Onset of Hypertension



Note: Country averages are weighted. The sample refers to individuals present in waves 2 and 3 of SHARE.

Figure 9: European Infant Mortality and Hypertension (All Cohorts)



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