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ABSTRACT

Economic Conditions at Birth and Cardiovascular Disease Risk in Adulthood: Evidence from New Cohorts*

Most of the literature that exploits business cycle variation at birth to study long-run effects of economic conditions on health later in life is based on pre-1940 birth cohorts. They were born in times where social safety nets were largely absent and they grew up in societies with relatively low female labor force participation. We complement the evidence from this literature by exploiting post-1950 regional business cycle variations in the Netherlands to study effects on cardiovascular disease (CVD) risk in adulthood, by gender. We operationalize CVD risk by constructing the Systematic COronary Risk Evaluation (SCORE) index from an extensive set of biomarkers. The data are from a large cohort study covering socio-economic, biological and health data from over 75k individuals aged between 18 and 63. We conclude that women born in adverse economic conditions experience higher CVD risk.

JEL Classification: I10, I15, J11

Keywords: early-life conditions, developmental origins, recession, health, unemployment, long-run effects, biomarkers

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1. Introduction

An expanding body of literature has documented the negative effects of adverse economic conditions around the time of birth on a range of high-age mortality and morbidity outcomes. To avoid endogeneity problems with the use of measures of economic conditions in the household into which the individual is born, many studies exploit exogenous variation in transitory contextual conditions instead. Notably, the business cycle at birth has been used as an instrumental variable in reduced-form analyses (see, for instance, van den Berg *et al.*, 2006, 2009, 2011, 2013, Angelini and Mierau, 2014, Cutler *et al.*, 2016, and the overviews in Almond and Currie, 2011 and van den Berg and Lindeboom, 2014). The literature focuses predominantly on over-all or cause-specific mortality as outcome measures. Since mortality is an end-state phenomenon, the analyses typically examine cohorts born before World War II. This gives rise to a number of issues.

First of all, the extent to which a recession leads to adverse economic conditions within households may be smaller for cohorts born after World War II. Western European countries have established social safety nets that include relatively generous unemployment benefits and welfare payments for those without work. In econometric terms this would mean that the business cycle as an instrumental variable for economic conditions in the household has become weaker. Note that this does not imply that the causal effect of the conditions at the individual or household level has diminished in size.

Another difference between pre-war birth cohorts and modern cohorts is that among the former, female labor force participation in adulthood was less common. Somewhat speculatively, this might imply that women in modern cohorts encounter more stress, adverse life events and shocks in adulthood, increasing the extent to which early-life conditions are expressed in adult health. In econometric terms, this would mean that on average the causal long-run effect of conditions at the individual level has increased among women. If at the same time the role of the

business cycle at birth has diminished then it is an open question whether the net association between the cycle at birth and adult health among women has increased or not.

This gender issue is important in the light of the fact that studies with cohorts born a long time ago tend to find smaller over-all effects among women than among men, at least in terms of mortality outcomes (see the above references; some studies focus mainly or solely on men). Biological evidence documents gender-differences in fetal sensitivity (Catalano *et al.*, 2005) which may be driven by different intra-uterine growth strategies between male and female fetuses (see, Eriksson *et al.*, 2010).

A third difference between more distant and more recent cohorts relates to past improvements in healthcare, prenatal care and health education. These allow certain potentially adverse health outcomes to be detected early or to be prevented in modern cohorts. This could lead to a reduction of the actual causal long-run effect of adverse conditions.

The current paper deals with the above issues by examining data from post-World War II birth cohorts. We are particularly interested in cardiovascular diseases (CVD) as health outcome. There are a number of reasons for this. First, CVD stands out as the leading cause of death in Europe and around the world (Nichols *et al.*, 2014). Secondly, CVD mortality has been in the mainstay of the early-life conditions literature since its inception (see the above references). Indeed, the Barker hypothesis (see Section 2) was formulated specifically in terms of CVD (Barker, 1995). Thirdly, we are restricted to examine outcome measures that are observable among cohorts born after World War II. For cohorts born in, say, the 1970s, mortality outcomes are not suitable. Indeed, even actual CVD is not very common among prime-aged individuals. To proceed, we operationalize CVD *risk*, using biomarker data. Biomarkers enable the observation of a quantitative outcome variable at prime ages. The fact that reliable biomarkers exist for CVD is an additional argument for focusing on CVD rather than outcomes that are harder to predict, such as many types of cancers. Notice, however, that the literature on the

developmental origins of late-life health puts CVD in the same range of outcomes as type-2 diabetes, mental health and cognitive impairments (see again the above references and, e.g., Lumey *et al.*, 2011), suggesting that CVD biomarkers are also informative on the risks of those health outcomes.

The use of biomarker data has an additional advantage. Policy interventions aimed at preventing high-age CVD have to rely on predictors of CVD such as the biomarkers we examine. If a relation with economic conditions early in life exists then the collection of the relevant biomarker data among adults can be targeted to those born under adverse conditions.

The biomarker data we use are collected as part of the Lifelines data – a large-scale study covering over 75,000 individuals born between 1950 and 1992 who were alive and residing in the northern part of the Netherlands in 2013 (see Scholtens *et al.*, 2015). The raw variables (such as blood cholesterol levels, systotic blood pressure and BMI) are used to compute the so-called Systematic COronary Risk Evaluation (SCORE) index. The latter was developed and validated by the European Society of Cardiology to estimate gender- and age-specific 10-year absolute risks of a fatal CVD event (see, Conroy *et al.*, 2003, for a detailed description).

We use spatial and temporal fluctuations in the unemployment rate (taken from Statistics Netherlands) as exogenous indicators of economic conditions early in life. One advantage of this measure, as opposed to GDP, is that it is available at the level of a province for our full observation window. At the time, the Netherlands had 11 provinces, and most individuals in the sample were born in the three that constitute the area from which the original sample was drawn in 2013.

Combining the biomarker-based SCORE index with provincial level unemployment data enables us to analyse the relationship between adverse conditions at birth and CVD risk later in life. To understand some of the potential mechanisms at play in the translation of adverse early-

life conditions to later life health outcomes we also analyse other life-course outcomes such as smoking, body mass and physical activity.

The remainder of the paper is set up as follows. The following section highlights some of the key contributions relevant to our current analysis. Section 3 and 4 describe the data and outline our empirical strategy, respectively. Section 5 presents and discusses our results and the final section concludes.

2. Background Literature

Two complementary frameworks attempt to explain the impact of adverse macroeconomic conditions early in life on health outcomes later in life (see also the overviews cited in Section 1). The *biological programming* framework – alternatively referred to as the critical period, biological imprint, biological embedding or fetal origins hypothesis – states that certain exposures early in life permanently and irreversibly alter the structure and/or function of organs, tissues and systems (see Barker, 1995, Rasmussen, 2001, Kuh and Hardy, 2002, Kuh and Ben-Shlomo, 2004, Case *et al.*, 2005, Wadhwa *et al.*, 2009). The *pathway* framework rests less on the biological imprint, instead stating that adverse early-life circumstances have an indirect influence because they set in motion lifelong trajectories of health-related disadvantages (Kuh and Ben-Shlomo, 2004). While the mechanisms by which adverse conditions early in life translate into health outcomes later in life differ between both frameworks, we may note that they are not mutually exclusive and that they are best seen as complements instead of alternatives.

In addition to these frameworks, selection effects may play a role in the relationship between early-life conditions and health outcomes later in life. Deheija and Lleras-Muney (2004), for instance, document that during periods of high unemployment among black mothers relatively more babies are born to highly educated mothers, while for white women the opposite is true. Such changes to the composition of the birth cohort are then also reflected in the health

outcomes of individuals born during recessions because health is strongly correlated over generations.

As suggested above, early-life conditions may also affect women and men differently. Indeed, Catalano and Bruckner (2005) and Catalano *et al.* (2005) show that there is a larger proportion of male stillbirths and miscarriages during harsh periods. Eriksson *et al.* (2010) argue that while the growth of every foetus is constrained by the placenta, boy's placenta's are more efficient than girls but have less reserve capacity. As a consequence, boys are more susceptible to adverse events during pregnancy. Most of the literature suggests that long-run mortality effects are stronger for men (see references in Section 1; see also Doblhammer *et al.*, 2013). An exception is Yeung *et al.* (2014) who report that effects on CVD mortality are stronger among women than among men.

3. Data

For the purpose of our analysis we combine individual health data from Lifelines with data on regional unemployment from Statistics Netherlands. In what follows we describe each in turn and explain the construction of our key variables of interest.

3.1 Lifelines

Lifelines is a large population-based cohort study and biobank carried out in the northern part of the Netherlands that was established as a resource for research on complex interactions between environment, phenotypic and genomic factors in the development of chronic diseases and healthy ageing (see Klijs *et al.*, 2015, and Scholtens *et al.*, 2015, for a detailed description of the study). In total, Lifelines supplies us with a sample of 95,422 individuals born between 1950 and 1992.[1] For our analysis, we select only individuals born in the Netherlands, which reduces

[1] Lifelines also contains respondents born before 1950 and after 1992. For the former, however, we do not have regional unemployment data, while the latter were administered a different

the sample size to 80,820. Further, we exclude any observations that do not contain all of the information necessary to calculate our main outcome variable (the CVD risk SCORE) such as smoking status, age, gender, total cholesterol and blood pressure. Our final sample consists of 76,566 individuals.

By linking the Lifelines data to birth certificate data from the Municipal Personal Records Database (in Dutch: *Gemeentelijk Basisadministratie*), we obtain information on the province of birth of each sample member. While Lifelines contains individuals born all over the Netherlands, births from the three northern provinces are naturally overrepresented. Table 1 presents the sample sizes per province.

[TABLE 1 ABOUT HERE]

For our purpose, the most important feature of the Lifelines cohort study is that it includes biomarkers concerning cardiovascular disease (CVD) risk, which we can use to construct the Systematic COronary Risk Evaluation (SCORE) index. The SCORE index was developed and validated by the European Society of Cardiology to estimate the 10-year risk of a fatal CVD event. In contrast to an actual fatal CVD event (e.g., a major heart attack), the advantage of a surrogate endpoint such as the SCORE index is that we are able to consider relatively younger individuals who are more representative of current cohorts.

The SCORE index is constructed according to an algorithm (see Appendix A of Conroy *et al.*, 2003) that uses age, gender, smoking status, blood cholesterol levels and blood pressure as inputs to estimate the 10-year absolute risk of a fatal CVD event. We can draw all the constituent parts of the SCORE index from the Lifelines data and are, therefore, able to associate the CVD risk to each individual in our sample. A detailed overview of the CVD risk and in its distribution

survey due to their age. We are using the Lifelines Baseline sample for ages 18 to 63 (release 201303, made available in 2014).

is contained in Table 2 as well as Figure 1. Both the table and the figure highlight that CVD risk exhibits a strong gender specific pattern. Moreover, we note that CVD risk is strongly skewed, with most individuals displaying limited CVD risk – an issue that we address in the analysis of our results.

[TABLE 2 ABOUT HERE]

[FIGURE 1 ABOUT HERE]

In addition to the biomarkers required to construct the SCORE index, Lifelines also includes health, lifestyle, family and socioeconomic information about the respondents, which we use to explore some of the potential mechanisms by which early-life conditions translate into later life health outcomes. Indeed, the *pathways* framework suggests that adverse early-life conditions set in motion a series of health-related advantages which in turn lead to adverse health outcomes later in life. The potential pathways we explore are the following. The health and lifestyle factors are the body mass index (i.e., weight in kilograms divided by the square of height in meters), whether or not an individual smokes and the number of days with physical activity per week. The family indicators cover marital status and the number of children. The socioeconomic indicators are employment status and the highest completed education. Moreover, to tentatively assess *selection* effects we exploit the fact that Lifelines contains some socioeconomic indicators of the family into which the individual was born, albeit the information is limited. In this respect we use the age of the respondent's mother when he/she was born and whether or not she was smoking during the pregnancy. Both indicators have been shown to have a strong relationship with a family's socioeconomic status – with family's from lower

socioeconomic groups tending to have children earlier and being more likely to smoke.[2] The summary statistics of these and our other variables of interest are provided in Table 3.

[TABLE 3 ABOUT HERE]

3.2 Unemployment Data

We proxy the early life conditions by using provincial unemployment data from Statistics Netherlands. The unemployment rate provides us with a contextual variable that serves as a proxy of the socioeconomic conditions under which the individual was born without suffering from the endogeneity of individual level socioeconomic indicators. Provincial level unemployment data are available from 1950 onward, which creates the lower bound for the birth year in the data. We display the development of unemployment between 1950 and 1992 in Figure 2.

[FIGURE 2 ABOUT HERE]

During our sample period, the Netherlands went through all phases of the business cycle multiple times. After World War II, the Netherlands enjoyed a period of substantial economic growth with low associated unemployment rates. At the end of the 1970s and for much of the early 1980s, the Netherlands were hit by a strong recession due to the second oil crisis. This recession was particularly strong in the northern Netherlands where unemployment peaked at well over 10% at the height of the recession. In the early 1990s alongside the world-wide economic boom, unemployment rates dropped significantly all over the country. While the data

[2] Naturally, the impact of these indicators need not only run through the socioeconomic status of the parents but may also have direct effects in their own right. Smoking during pregnancy in particular has been associated to a plethora of adverse health outcomes for babies.

display a clear common trend among the provinces, we also observe substantial differences in both levels and trends of unemployment between the provinces. Implying that province-level unemployment data provides us with additional variation from which to identify our relationship of interest.

4. Methods

Our main interest lies in the relationship between unemployment in the birth year and CVD risk later in life. To this end, we start with a simple linear specification with CVD risk as the outcome variable and the provincial unemployment level in the birth year as main explanatory variable. To account for the structure of the data, we also include a birth year and a province fixed effect. We allow the effect of unemployment at birth to be gender specific to account for potential gender differences in the relationship between early-life conditions and health later in life.

Beyond a direct impact on the SCORE index, age can act as a risk factor or as modifier of the other risk factors – the levels of cholesterol and blood pressure increase with age and increase earlier in life in men than in women. We control for age in the model by including a linear spline with knots at 30, 40, 50 and 60 years. Since we also account for birth year fixed effects in the model, there is a risk of multicollinearity. However, since the Lifelines data were gathered over a 7-year period (2006-2013), sample members with identical ages can have a variety of different birth years.

To sum up, we estimate the following specification:

$$CVD_{ipt} = \alpha + \beta_1 u_{pt} * m_i + \beta_2 u_{pt} * f_i + \beta_3 m_i + \sum_{k=1}^{K} \beta_4 s_{ipt} + \theta_t + \rho_p + \varepsilon_{ipt} \quad (1)$$

where CVD_{ipt} denotes ten-year fatal CVD event risk for individual *i* born in province *p* in year *t*; u_{pt} is the unemployment rate in province *p* and birth year *t*, m_i is a dummy variable taking value 1

if male and 0 if female, while f_i takes the opposite value; s_{ipt} stands for a linear spline with K knots in age of the individual i born in province p and year t; ρ_p is a province fixed effect, and θ_t is a year-of-birth fixed effect. We estimate the specification in (1) by ordinary least squares (OLS).

Since our main explanatory variable – the unemployment level – is measured at the province level, the error terms are likely to be correlated within provinces. Therefore, cluster-robust standard errors are required for statistical inference. The province fixed effects partially control for the within-province correlation, but perhaps not completely. In addition, since there are only 12 provinces[3] in the Netherlands, the number of clusters is small which means that the estimated variance matrix of the OLS estimator is likely to be downwards biased (Cameron and Miller, 2015). Therefore, we apply the bias-correction of Bell and McCaffrey (2002), which was named CR2VE by Cameron and Miller (2015),[4] to the standard cluster-robust variance estimates. CR2VE correction implies scaling the province specific vector of residuals $\hat{u}_p$ so that $\tilde{u}_p = \left(I_{Np} - H_{pp}\right)^{-0.5} \hat{u}_p$, where $H_{pp} = X_p(X'X)^{-1}X'_p$ with X_p being an $N_p \times K$ matrix, where K is the number of variables included in the model, and N_p is the size of the sample in province p. Stacking X_p over P provinces yields X. In addition, since the number of observations varies considerably across provinces, the effective number of clusters is reduced even further (Imbens and Kolesar, 2016). We therefore base the Wald tests on a $t(\nu^*)$-distribution where the degrees of freedom $\nu^*$are determined by the data as proposed by Imbens and Kolesar (2016). According to a Monte Carlo study performed by Cameron and Miller (2015), the null hypothesis is rejected too often if we use the "standard formula" for cluster robust standard errors when the number of clusters is small. However, the use of CR2VE residual and of the data-determined degrees of freedom leads to a considerable improvement in inference. That is, the actual size of the t-test

[3] Due to the small sample sizes, the three provinces with smallest number of observations (Limburg, Flevoland and Zeeland) were grouped together so effectively we have 10 clusters.
[4] See Cameron and Miller (2015) section VI.B. on page 342 and section VI.D. on page 346.

(the probability of Type I error given the sample size) is close to the nominal size of the test (the desired significance level α).[5]

5. Results

We present our main estimation results in Table 4. Column (1) contains the OLS estimate of the model specified in Equation (1) with the cluster robust standard errors determined as outlined above. The results suggest that while the impact of unemployment on women's CVD risk is significant at the 1% level – even after taking into account the CR2VE standard errors – the impact on men does not differ significantly from zero.[6] More precisely, for women a 1 percentage point increase in the provincial unemployment level leads to a 0.02 percentage point increase in the risk of experiencing a fatal CVD event in the coming 10 years. While this effect may seem relatively small, comparing it to the effect of ageing indicates that, for instance, for a 45 year old woman born when the unemployment rate was elevated by 1 percentage point, the CVD risk is equivalent to that of an identical woman who is 6 months older but born when the unemployment rate was not elevated.

[TABLE 4 ABOUT HERE]

In addition to the *biological programming* and the *pathways* frameworks – to which we return below – any impact of early-life conditions on later life outcomes may be due to selection effects. While Lifelines does not include information about the individual socioeconomic conditions into which a child was born, we do have knowledge of the age of the respondent's mother when he/she was born and whether or not she was smoking. Both indicators have been

[5] See page 362 of Cameron and Miller (2015).

[6] Regardless of the early-life conditions, men do have a higher CVD risk than women, as is reflected by the substantial point estimate of the gender dummy.

shown to have a strong relationship with a family's socioeconomic status – with family's from lower socioeconomic groups tending to have children earlier and being more likely to smoke. To account for potential selection effects we add the two indicators to our main specification and report the estimation results in column (2) of Table 4. The results indicate that while age of the mother is not associated with elevated CVD risk, being born to a mother who smoked is strongly associated to heightened CVD risk later in life. However, after accounting for these two variables, the magnitude of the impact of unemployment at birth on CVD risk later in life is essentially unaffected. This result leads us to believe that selection effects do not rationalize our results.

The pathways framework implies that adverse early-life conditions set in motion lifelong trajectories of health-related disadvantages, which in turn influence health outcomes. To consider the implications of this framework further, Table 5 presents the relationships between unemployment levels at birth and various life-style and socioeconomic status indicators using the specification of Equation (1). We caution the reader that, in the absence of instrumental variables for events after birth, our data do not allow for a full-fledged mediation analysis. As such the following results should be considered in somewhat tentative fashion. The lifestyle factors – smoking, body mass index and physical activity levels – reveal that women born when unemployment was high are less likely to smoke but also less likely to engage in physical activity. Employment and education outcomes are not unaffected. Men, whose health was found to be unaffected by adverse employment conditions around birth, are found to be less likely to work and less likely to have completed higher education. In sum, it seems that the life-cycle of men and women are differently affected by adverse conditions early in life.

[TABLE 5 ABOUT HERE]

We have performed a range of sensitivity analyses to verify whether the results are robust with respect to aspects of the model specification (results available upon request). In general the key results are not qualitatively affected by modifications of the specification; that is, sign and significance remain preserved. For example, the results are robust to adding other biomarkers from blood samples to the SCORE index (such as glucose levels and inflammation markers). One exception to the general robustness is found when birth-year fixed effects in the model specification are replaced by a second-degree polynomial in the year of birth. Apparently, the polynomial is not able to capture the effects of the major restructuring of the economy in the early 1980s, or the combination of a low-degree polynomial and an age spline is not sufficiently flexible to fit secular time and age patterns in the data. Also, low-degree polynomials may be less suitable than birth-year fixed effects if the operationalization of the definition of unemployment changes over calendar time or if there are institutional changes in the ease with which transitions into out-of-the-labor-force states such as disability and early retirement can be made. As a final sensitivity analysis, we estimated models for actual CVD occurrence. As expected, due to the low occurrence of CVD, none of the estimated effects is significantly different from zero. The signs of the effects are in line with our results.

6. Conclusions

We analyse the effect of provincial unemployment level at the time of birth on a biomarker-based measure of CVD risk, using data on birth cohorts from 1950-1992. We pay particular attention to gender differences in this effect. A key result is that women exposed to unfavorable business-cycle conditions at birth are at an increased risk for fatal CVD events in adult life. We interpret this as evidence that unfavorable economic conditions in the household at birth cause an elevated CVD risk in prime and late adulthood among women. The fact that studies using data from earlier birth cohorts did not unambiguously find strong evidence for such an effect among

women may reflect a gradual increase in the size of this causal effect over the past century. This in turn could be due to increases in stress levels among women in their adulthood, increasing the scope for dynamic complementarities between early-life conditions and shocks in adulthood. However, this requires further research.

For men, CVD risk is unaffected by early-life exposure to recessions. As explained in the paper, this does not necessarily entail that causal effects of adverse economic conditions at the individual level are absent. Instead, the business cycle as an instrumental variable for those conditions may have weakened due to improving social safety nets over the 20th century.

In the end, relatively recent birth cohorts are potentially more representative of current and future cohorts. And the usage of biomarkers allows us to detect elevated health risks well before health events occur. Taken together, this means that the results point at increased risks of actual CVD in the near future.

Extended acknowledgments regarding the Lifelines data: The authors wish to acknowledge the services of the Lifelines Cohort Study, the contributing research centres delivering data to Lifelines, and all the study participants. Lifelines is a multi-disciplinary prospective population-based cohort study examining in a unique three-generation design the health and health-related behaviours of 167,729 persons living in the North East region of The Netherlands. It employs a broad range of investigative procedures in assessing the biomedical, socio-demographic, behavioural, physical and psychological factors which contribute to the health and disease of the general population, with a special focus on multimorbidity. In addition, the Lifelines project comprises a number of cross-sectional sub-studies which investigate specific age-related conditions. These include investigations into metabolic and hormonal diseases, including obesity, cardiovascular and renal diseases, pulmonary diseases and allergy, cognitive function and depression, and musculoskeletal conditions.

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Figure 1: Histograms of absolute 10-year risk of fatal cardiovascular disease event for men and women.

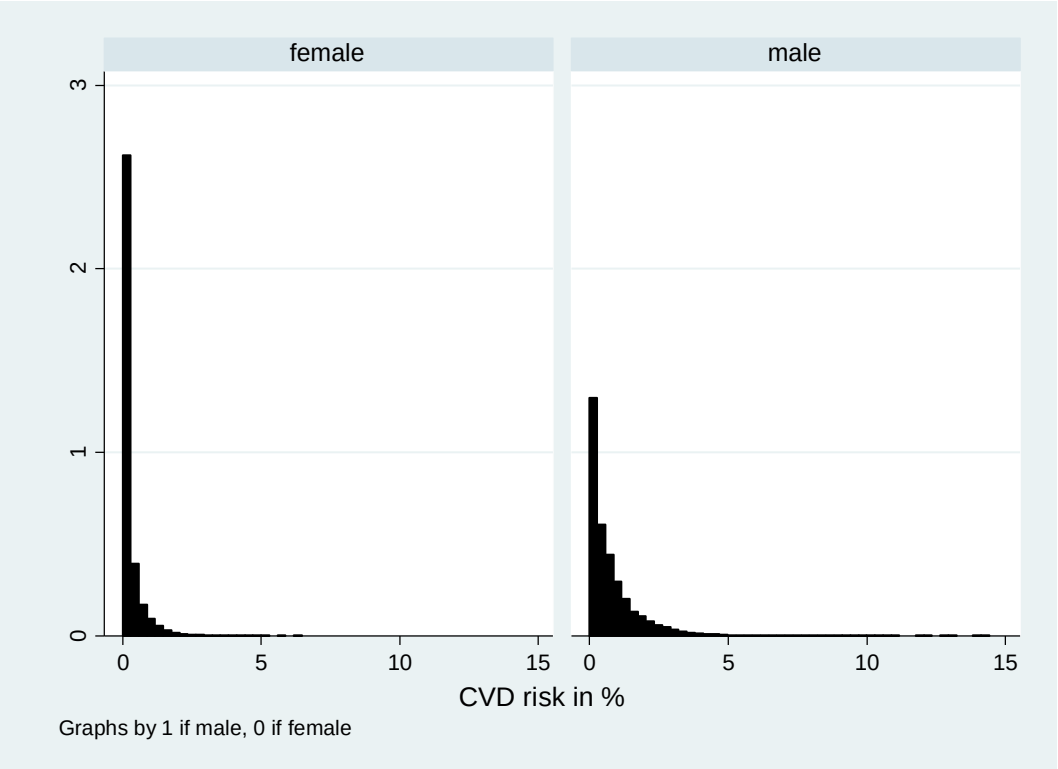
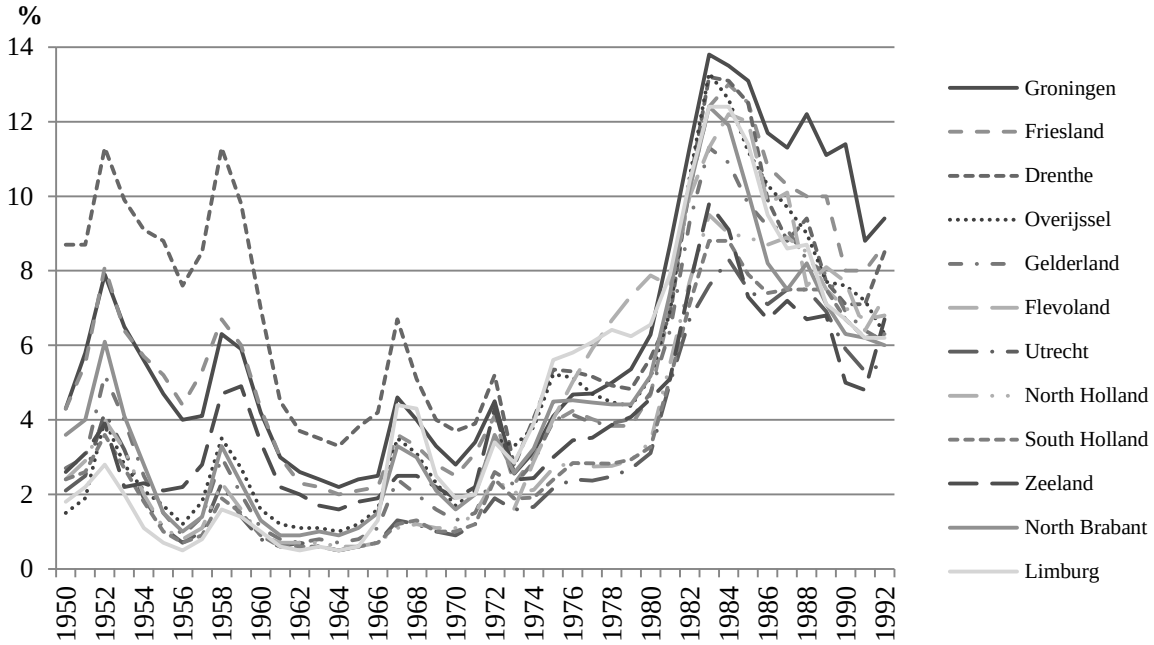


Figure 2: Provincial unemployment level in the Netherlands, 1950-1992



Source: Statistics Netherlands (www.cbs.nl)

Table 1: Sample size per province

Province	Frequency	Percent
Friesland	27,864	36.39
Groningen	22,112	28.88
Drenthe	13,757	17.97
Zuid-Holland	2,955	3.86
Noord-Holland	2,788	3.64
Overijssel	2,684	3.51
Gelderland	1,775	2.32
Utrecht	1,045	1.36
Noord-Brabant	899	1.17
Limburg	350	0.46
Zeeland	182	0.24
Flevoland	155	0.2
Total	76,566	100

Table 2: Descriptive statistics

Variable	Observations	Mean	Std. Dev.	Min	Max
Provincial unemployment rate (%)	76566	4.620	3.031	0.500	13.80
Male	76566	0.410	0.492	0	1
Age at the first visit	76566	42.42	9.526	20.00	63.04
Birth year	76566	1968	9.562	1950	1992
CVD risk score (%)	76566	0.470	0.778	0	14.12
Body mass index (kg/m2)	76543	26.00	4.349	13.88	66.26
Smoking (Yes/no)	76566	0.234	0.423	0	1
Physically active (Nr. days per week)	73218	4.200	2.200	0	7
Number of children	76566	1.619	1.257	0	10
Married	76443	0.814	0.389	0	1
Working	76566	0.698	0.459	0	1
Not-employed	76566	0.123	0.328	0	1
Disabled	76566	0.028	0.164	0	1
Primary education	76363	0.016	0.127	0	1
Secondary/ vocational education	76363	0.663	0.473	0	1
Higher education	76363	0.305	0.460	0	1

Table 3: Detailed descriptive statistics for CVD risk score (%)

	All	Males	Females
Percentiles			
1%	$1.07*10^{-4}$	0.003	$7.15*10^{-5}$
25%	0.035	0.376	0.015
50%	0.174	0.478	0.085
75%	0.582	1.075	0.268
99%	3.604	4.765	1.908
Observations	76566	31411	45155
Mean	0.470	0.809	0.235
Std. Dev.	0.778	1.025	0.399
Skewness	3.995	3.073	3.758
Kurtosis	30.29	19.13	24.70

Table 4: CVD risk and unemployment level at birth by gender.

	(1) SCORE CVD death risk %	(2) SCORE CVD death risk %
female x unemp	0.021***	0.021***
	0.002	*0.002*
male x unemp	-0.006	-0.006
	0.013	*0.013*
Male	0.679***	0.681***
	0.058	*0.057*
Age of mother at birth		$-3.5*10^{-4}$
		0.001
Mother smoked during pregnancy		0.034***
		0.006
Linear spline in age	*YES*	*YES*
Province fixed effects	*YES*	*YES*
Birth year fixed effects	*YES*	*YES*
Observations	*76,566*	*76,566*
IK degrees of freedom:		
female x unemp	6.8	6.8
male x unemp	6.9	6.9
Test male*unemp - female*unemp	***	***

Note: OLS regression results. CR2VE standard errors clustered at the province level are reported in italics under the coefficients. The Imbens-Kolesar degrees of freedom used in the t-tests for the key variables are reported at the bottom of the table (*** signifies p<0.01, ** signifies p<0.05, and * signifies p<0.1).

Table 5: Life-course outcomes and unemployment level at birth by gender.

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
		Lifestyle		Family			
VARIABLES	Smoking	BMI	Physical activity	Number of children	Married	Working	Higher education
female x unemp	-0.004*	0.010	-0.038**	-0.010	0.007*	0.002	0.002
	0.002	*0.022*	*0.015*	*0.015*	*0.003*	*0.002*	*0.003*
male x unemp	-0.004	-0.046	0.006	-0.009	-0.005	-0.011***	-0.010*
	0.003	*0.027*	*0.015*	*0.014*	*0.004*	*0.002*	*0.004*
male	0.033***	0.842***	-0.313***	-0.166***	0.055***	0.207**	0.073***
	0.006	*0.065*	*0.064*	*0.019*	*0.006*	*0.011*	*0.010*
Linear spline in age	*YES*	*YES*	*YES*	*YES*	*YES*	*YES*	*YES*
Province fixed effects	*YES*	*YES*	*YES*	*YES*	*YES*	*YES*	*YES*
Birth year fixed effects	*YES*	*YES*	*YES*	*YES*	*YES*	*YES*	*YES*
Observations	76566	76543	73218	76566	76443	75684	76363
Imbens-Kolesar degrees of freedom:							
female x unemp	6.8	6.8	6.9	6.9	6.9	6.9	6.9
male x unemp	6.9	6.9	7.0	7.0	7.0	7.0	7.0

Note: OLS regression results. CR2VE standard errors clustered at the province level are reported in italics under the coefficients. The Imbens-Kolesar degrees of freedom used in the t-tests for the key variables are reported at the bottom of the table (*** p<0.01, **p<0.05, * p<0.1).