# Essays in Empirical Health Economics

Inauguraldissertation
zur Erlangung des akademischen Grades
eines Doktors der Wirtschaftswissenschaften
der Universität Mannheim

vorgelegt von
Kai Eberhard Kruk

Mannheim, 2011

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|---|---|
| Dekan: | Prof. Dr. Martin Peitz |
| Referent: | Prof. Dr. Hendrik Jürges |
| Korreferent: | Prof. Axel Börsch-Supan, Ph.D. |
| Datum der mündlichen Prüfung: | 25. Oktober 2011 |

# Acknowledgements

Completing this dissertation would not have been possible without the support from numerous persons. Foremost, I want to thank my advisors Hendrik Jürges, Steffen Reinhold and Axel Börsch-Supan for their constant encouragement and mentoring during my Ph.D. studies. I learned very much from their experience and knowledge and this dissertation benefitted greatly from their comments and suggestions. Some of my research is inspired by their former projects and ideas.

Furthermore, I thank my colleagues Stefan Listl, Edgar Vogel, Tabea Bucher-Koenen and Karsten Hank for helpful comments on earlier versions of chapters of this dissertation.

Many people I had the opportunity to meet at conferences and workshops helped to considerably improve the quality of my papers through their feedback. Therefore, I want to express my gratitude to all participants of the conferences and workshops I attended during the last two years. In particular, I thank Maarten Lindeboom, Andrew Jones, Stefanie von Hinke Kessler Scholder, and Patrick Heuveline for constructive discussions.

My family was a constant source of support, in particular in periods when instruments were weak, standard errors too large or theories hard to digest. I would have had a hard time without you!

Lastly, I want to express my gratitude to my colleagues at MEA. The SHARE team provided excellent support when I struggled with the data. I also want to thank all those people that provided the infrastructure needed to concentrate on research whilst working at MEA.

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# Chapter 1

# General Introduction

Abating health inequalities is high on the political agenda. In 2005, the WHO invoked an expert commission on the social determinants of health. Its final recently published report recommends reducing societal risk factors in order to alleviate health inequalities worldwide (WHO (2008)). Moreover, it states that reducing inequalities in health is a moral imperative. The design of adequate policies, however, requires an accurate understanding of promotive and protective factors for the incidence and prevalence of diseases.

A number of widespread diseases nowadays are not caused by the microbiological environment but by the social and economic context individuals are embedded in. The more recent research literature accounts for this development by putting a focus on identifying causes of diseases rooted in the individual social and economic living conditions.

Despite the public interest and enormous efforts made by a large number of researchers, many facets of the relationships between the individual social and economic living conditions and health are still poorly understood.

This dissertation investigates assorted aspects of this relationship. It is structured in four self-contained papers, each dealing with one specific research question that is part of this broader research agenda. Chapters 2 and 3 focus on the effect of markers of socioeconomic status on health, whereas chapters 4 and 5 focus on the effect of the social environment on mental health. It embarks on an interdisciplinary journey by touching topics previously investigated by psychologists, public health researchers, sociologists and economists.

Since the approach taken reflects the view and methodological grounding of an economist, I termed it "Essays in Empirical Health Economics", although some of the topics covered are rather new to the economics profession.

It is the main ambition of this dissertation to make a small contribution to the knowledge base on the interrelationship between the social and economic environment and health. My primary motivation for pursuing this line of research is to further our understanding of prevalence patterns in our society. By applying new methods and data in part to old but also to new questions, I hope to shed light on a number of recent debates, such as the following:

- Can we take the correlation of education and health as evidence for a causal effect of education? Therefore, does increasing the level of education of disadvantaged groups increase equality in terms of health?
- Children from poor families suffer more often from critical health conditions. Can we conclude that redistributing resources towards the lower end of the income distribution protects children? Can a universal health insurance system provide children with equal chances of recovering from periods of poor health?
- Features of the social environment, such as household composition, strongly correlate with the probability to suffer from mental disorders. Can we conclude that our environment shapes our mental well being or is it rather the other way around?

The questions listed above are discussed controversially in both academia as well as popular press. I hope that the findings presented in this dissertation contribute to a well informed debate and thus, possibly, to the design of sound policies.

The secondary motivation for the single chapters is to highlight and investigate problems of populations that only recently moved into the spotlight of health economic research. The research in this dissertation is mainly concerned with two specific populations: children and elderly individuals.
Chapters 2 and 4 deal with health in old age, which in the light of population aging and the fact that health care systems already spend a considerable

amount of resources for the elderly, seems a fertile line of research, which will likely gain importance in the near future.[1]
Chapters 3 and 5 are concerned with child health, an area of research that only in the last decade became an established part of the research agenda in economics. Given the substantial long-term costs of poor child health that a number of recent studies highlighted, I consider the socioeconomic determinants of poor child health a highly promising line of research that deserves more attention.[2]

The single papers presented in this dissertation are not only characterized by the common subject but also by two methodological commonalities. For many years, casual relationships, such as the fact that higher educational attainment positively correlates with better health, were oftentimes taken as causal relationships. In this context, this would imply that as we observe that highly educated individuals are on average healthier than those individuals with less education, education must causally affect health.
However, conclusions such as this one can be misleading. One of the most fruitful fields of economic research in the past decades has been the development of econometric methods that allow to identify causal effects. Some authors go as far as terming this development the "credibility revolution in empirical economics" (Angrist and Pischke (2010)). Using these methods often allows to pinpoint causal effects rather than interpreting loose empirical relationships causally. In this dissertation, I put a particular focus on causal interpretations and I try to be very careful with respect to causal statements. I make use of several of these methods such as instrumental variables estimation, regression discontinuity design techniques and panel data methods. A substantial part of each paper is concerned with a discussion of the assumptions needed for causal conclusions and with robustness checks that inspect the validity of those assumptions that are testable.

Moreover, all analyses are based on large and representative micro data sets. The advantages of these surveys compared to the small and selective samples

[1] Projections by the OECD suggest that health care expenditures for seniors will at least double until 2050 in OECD countries (Colombo et al. (2011)).

[2] For evidence on the long-term consequences of poor health in childhood, see for example Case and Paxson (2010) and the references cited therein.

that are often used are obvious: Larger samples allow more robust statistical inference which facilitates causal conclusions.

## General Abstract

For a long while, it has been well known that individuals with higher income and more education have on average a better health status than individuals at the lower end of the distributions of income and education. However, this relationship is still poorly understood. Chapter 2 explores the question whether education exerts a causal effect on health outcomes. This project is joint work with Hendrik Jürges and Steffen Reinhold. The novelty of this paper lies in the assessment of health status based on biomarkers, which are objective measures of health status that have entered standard surveys only recently. The biomarkers we investigate reflect stress levels and therefore allow to test one specific aspect of the relation between education and health status, namely we ask whether more education allows individuals to follow a less stressful lifestyle. The identification strategy is based on two reforms that each increased the years of compulsory schooling by one year in the United Kingdom. Our results suggest that there are at most small positive effects of education on objective measures of health.

Epidemiologists and economists have provided supportive evidence for the hypothesis that health inequality has its origins in early childhood. Even at very young ages, patterns of inequality are observable and evidence from several countries suggests that these patterns become more pronounced as children age. The third chapter of this dissertation deals with the evolution of health inequality in early childhood. In contrast to the focus on the effects of education in the second chapter, chapter 3 is devoted to the effect of parental income as an alternative marker of socioeconomic status. I shed light on the intergenerational transmission of inequality by linking parent's income to the health status of their offspring. In particular, I follow up a panel of children from a British cohort study and assess the relationship between parental income and a child's health status in several ways and at several points in time. The results of this study indicate that the medium

term consequences of certain diseases differ substantially by the socioeconomic status of families. However, there is only weak evidence supporting the hypothesis that children from low income families are generally more susceptive to longstanding health conditions than children from high income families.

Chapters 4 and 5 of this dissertation are devoted to a topic that has attracted considerable attention from both researchers and the popular media in the last years: the rising prevalence of mental diseases. In particular, I focus on the determinants of depression in old age and Attention Deficit Hyperactivity Disorders (ADHD) among children. Both diseases imply high costs to affected individuals and health care systems. Despite these high costs, we still lack evidence about the determinants of mental disorders as well as on protective factors that reduce the risk of incurring these illnesses. So far, most of the literature in social sciences has focused on describing patterns of prevalence rather than pointing out causal mechanisms. Social scientists have only recently started to work on the causal determinants of these disorders.

Chapter 4 deals with the effect of having children on mental health in old age. In a sense, I twist the question raised in chapter 3 by asking for the role of children for their parent's health status in old age. In particular, I shed light on the question whether the positive aspects of child bearing outweigh its cost in terms of mental well being. For a long while, social scientists have conjectured that children protect their parents from depression in old age as they prevent loneliness and provide care. However, attempts to identify a causal effect of additional children on their parents' mental health status have not yet been undertaken. Answering this question is a methodological challenge as people with specific characteristics select into different levels of fertility. Thus, it is difficult to disentangle the causal effect of children from the selection effect into individual levels of fertility. I apply an identification strategy based on instrumental variables to overcome this problem and to calculate estimates for the causal effect of additional children on several measures of mental health in old age. The data set I use for this project comes from the Survey of Health, Aging and Retirement in Europe which has

the advantage of combining an extensive measurement of mental health with complete information on individual fertility histories. Overall, my results do not point to statistically significant causal effects of additional children for men. The birth of children can under certain circumstances even increase the risk of depression in old age for females.

The last chapter further explores the nexus between the social environment and mental health. Specifically, Chapter 5 is concerned with the question whether changes in the social environment increase the risk that a child develops symptoms of Attention Deficit Hyperactivity Disorder (ADHD). The motivation to work on ADHD is obvious. ADHD is by now the most common mental disorder in childhood, affecting about 1 out of 15 children (Faraone et al. 2003). Although ADHD is associated with substantial costs, we still know little about causal determinants rooted in the social environment. Most previous research suggests that genetics play an important role in explaining symptoms of ADHD. However, it is suspected that it is not genetics alone that determines behavioral disorders but rather an interplay of genetic disposition and the social environment that shapes the phenotype of ADHD. Despite the considerable public interest that this topic has aroused, it is astonishing that only few methodologically sophisticated papers investigate the social origins of ADHD.

The last chapter of this dissertation elucidates whether the absence of the father in the household and the birth of siblings affect a child's probability of developing mental disorders. A key advantage of the data set I use is that it is possible to follow up children over several years. This feature allows me to separate the effect of changes in household composition from the effect of unobservable factors which are constant over time, such as the genetic disposition or early life exposure to toxic substances. I compare the results for the effect of changes in household composition on symptoms of ADHD to the effect on symptoms of depression and anti-social behavior. My results strongly back the hypothesis that changes in household composition increase the probability for children to develop a symptomatology of ADHD. The estimated effects are quite robust and roughly comparable in their magnitude to the effects on other mental disorders.

# Chapter 2

# The Effect of Compulsory Education on Health – Evidence from Biomarkers

***Joint work with Hendrik Jürges and Steffen Reinhold***

## 2.1 Introduction

In this paper, we aim at contributing to the growing literature on identifying the causal link between education and health. Theoretically, the economic literature has identified causal effects of education on health through at least four plausible channels: (a) just as in the labor market, education raises efficiency in health production (raises the marginal productivity of inputs), i.e. it increases an individual's productive efficiency (Grossman (1972)); (b) education changes inputs into health production (through information) and thereby increases allocative efficiency (Rosenzweig and Schultz (1982)); (c) education itself changes time preference (and thus inputs into health production) because schooling focuses students' attention on the future (Fuchs (1982), Becker and Mulligan (1997)); (d) education has an indirect effect mediated through higher income, occupational status, and access to better housing, or environmental conditions.

Numerous studies have indeed documented a strong positive empirical asso-

ciation between education and health (see the surveys by Cutler and Lleras-Muney (2006) or Grossman (2006)). Interpretation of this correlation as causal is difficult, however, because education is most likely an endogenous variable, for instance because unobserved variables such as time preferences possibly drive both education and health behavior decisions, or because health (at younger ages) affects educational achievement (reverse causation). Recent empirical work addresses causality issues head on using natural experiments such as exogenous changes in compulsory schooling laws for identification.

In this paper, we study the possible causal link between education and health using two nationwide changes in minimum school leaving age in England in 1947 and 1973 as sources of exogenous variation. In those years, minimum school leaving age was raised from 14 to 15 (affecting birth cohorts born in or after April 1933) and from 15 to 16 years (affecting birth cohorts born in or after September 1957), respectively. Both reforms have already been used in previous studies to study causal effects of education on wages (Oreopoulos (2006), Devereux and Hart (2010)), or political participation (Milligan, Moretti, and Oreopoulos (2004)). We are also not the first to exploit this reform for causal analyses of education on health outcomes (see e.g. Oreopoulos (2006), Clark and Royer (2008), Silles (2009), Lindeboom, Llena-Nozal, and Van der Klaauw (2009), Powdthavee (2010)). Oreopoulos (2006) finds positive effects of this reform on self-rated health (and a range of labor market outcomes) in the combined UK General Household Surveys from 1983 to 1998. Clark and Royer (2008) use vital statistics and data from the Health Survey for England and find very small – not always significant – positive effects of the reform on mortality, self-rated health or health behavior. Critique concerning the external validity of such studies and their value for current policy recommendations could come from the fact that cohorts affected by the reform were born some 75 years ago. Education policy today might have a different effect. Silles (2009) also exploits the increase in mandatory school leaving age in 1973 that affected cohorts born in or after September 1957. Comparison of the effects of the two reforms that were 26 years apart gives us some indication whether (causal) education effects on health are stable over time. In fact, using data from the UK General Household Surveys, Silles

(2009) finds significantly positive causal effects of education on self-rated health for the 1947 reform but not for the 1973 reform.

While we analyze the effect of the same reforms as others, partly using the same data, we deviate from these papers in two important ways. First, in contrast to most earlier studies, all of our estimations will be sex-specific. As we show below, the education reforms have affected education decisions of men and women differently, and this can have a crucial effect on causal estimates of education effects based on these reforms. The second innovation of our paper is to complement the earlier analyses – that have mainly relied on self-reported health measures – by using biomarkers as health outcomes. One important recent development in survey research is the integration of biomarkers. Biomarkers are often associated with genetic information, i.e., DNA samples. However, the vast majority of biomarkers currently collected and analyzed are non-genetic: anthropometric measurements (height, weight, waist circumference, lung capacity, grip strength, balance), blood pressure, and blood and saliva samples. The scientific value of collecting such biomarkers in large surveys is promising (National Research Council (2008)). First, biomarkers improve the measurement of health. Self-reports of health are subject to considerable under-, over-, or misreporting, depending on the circumstances and dimensions at hand (e.g. Jürges (2007), Jürges (2008), Bago, O'Donnell, and van Doorslaer (2008)). Objective information can be used to validate respondents' reports and to study the amount and determinants of under-, over-, or misreporting in population surveys. Self-ratings of health may be subject to reporting bias that is correlated with important determinants of health. Self-reports of health have their own distinct scientific value. For instance, it has been shown that they contain information on health status even after conditioning on objective measures of health (Idler and Benyamini (1997)). Thus, biomarkers should be seen as complementary measurements rather than substitutes. However, the value of self-assessments alone as policy outcome measures is less clear. It would be hard to evaluate the benefits of a health care reform, say, that improves self-assessed health but leaves more objective measures of health unchanged.

Second, biomarkers allow studying physiological pathways in the complex

relationship between social status and health, providing information on important links that can be used to identify causal relationships. Below, we analyze markers that are known to be risk factors for cardiovascular disease. Thus our analyses allow identifying whether education has a causal effect not only on manifest conditions but also on the risk of developing a disease.

Third, biomarkers provide direct information on pre-disease pathways, in particular by measuring physiological processes that are below the individual's threshold of perception. This could be important for finding causal effects of education on the health at younger ages when diseases have not yet become manifest. More generally, combined with longitudinal data on individuals, biomarkers help to identify the role of the environment in turning health risks into manifest diseases. The latter points are especially important if education has important indirect effects on health through occupational status and work-related stress (Brunner et al. (1996)).

In our analyses, we concentrate on two biomarkers for inflammatory processes: blood fibrinogen, a blood-clotting factor, and blood C-reactive protein (CRP), a protein released into the bloodstream when there is active inflammation in the body. Both have recently gained much interest in the medical literature as predictors of incident cardiovascular disease (for reviews of the literature see e.g. Kamath and Lip (2003) and Hirschfield and Pepys (2003)). Elevated levels of fibrinogen and CRP have been shown to be strong, independent predictors of weight gain (Duncan et al. (2006)), incident diabetes (Pradhan et al. (2001)), or incident cardiovascular disease (Ridker et al. (2002), Ridker et al. 2003). Whether these associations are causal is still unknown. Still, for the medical practitioner such findings suggest that patients who would benefit most from interventions targeting blood pressure and cholesterol lowering, smoking cessation or exercise promotion, could be identified by blood fibrinogen and CRP levels.

Besides genetic variation, fibrinogen levels have been shown to be positively associated with age, being female, and being a smoker, obese, or physically inactive. Fibrinogen concentration has also been shown to be associated with childhood environmental conditions (measured by adult height and parental socioeconomic status), education level (Brunner et al. (1996)), and subjec-

tive social status (Demakakos et al. (2008)). Higher CRP levels have been shown to be associated with higher age, being female, and also with subjective social status (Demakakos et al. (2008)).

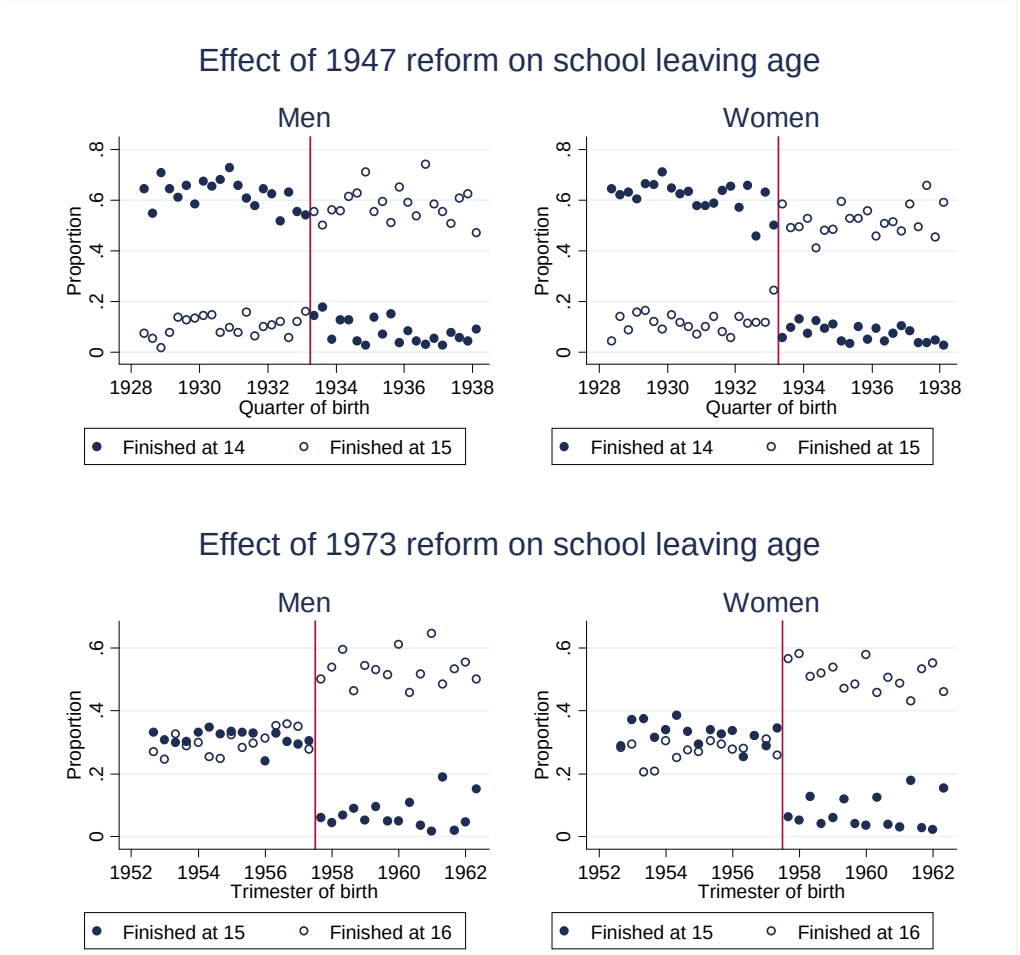
Our study proceeds as follows. In the next section, we will briefly describe the school reforms analyzed in this paper and their effect on educational attainment. In section 2.3, we explain the identification strategy (fuzzy discontinuity design) which we use to exploit these reforms. Section 2.4 describes the data and shows some descriptive results on the correlation between education and self-rated health, blood fibrinogen and blood CRP levels. Section 2.5 contains the causal estimates and robustness checks. We discuss our results and give conclusions in section 2.7.

## 2.2 Institutional Background

In this section, we will briefly describe the most salient aspects of the changes in schooling laws in Britain that we use for identification. The first change in minimum school leaving age analyzed in our paper was part of the 1944 Education Act and took effect on April 1st, 1947. Individuals who were born before April 1933 and who turned 14 before the law change could leave school at the end of the term in which they turned 14 (the UK school year is divided into three terms). Individuals who were born in April 1933 or later and who turned 14 after the law change had to stay in school until the end of the term in which they turned 15, i.e. at least until Summer 1948. This law change had a dramatic effect on the average age at which British pupils left school (see below). In 1973, minimum school leaving age was again raised, from 15 to 16, by the Raising of the School Leaving Age (ROSLA) Order of 1972. This reform affected pupils born on or after September 1st 1957.

Figure 2.1 illustrates the effect of the 1947 and 1973 changes in compulsory school leaving age on educational attainment (these data are from the combined HSE samples described below). For both reforms, we show the percentage of pupils who have finished school at age 14, 15 and 16, respectively, for birth cohorts born 5 years before to 5 years after the first cohort that was affected by the reform. Among pre-1947 reform cohorts, roughly

Figure 2.1
EFFECT OF THE TWO REFORMS ON SCHOOLING



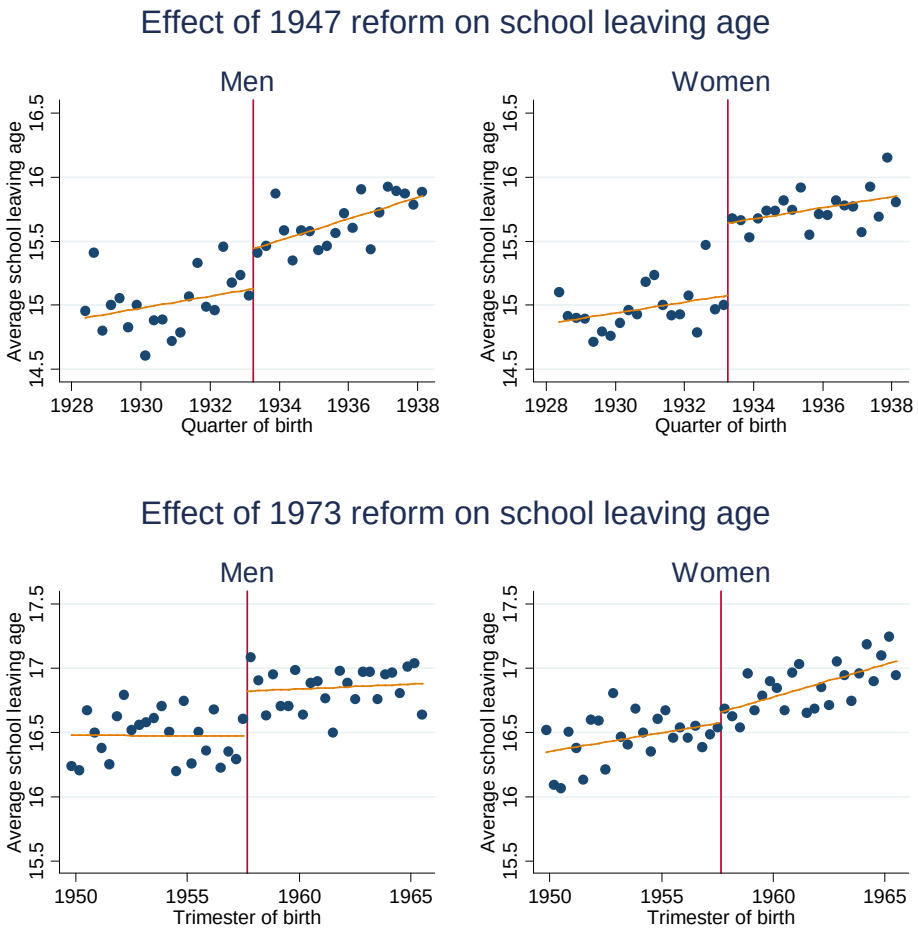

Effect of the 1947 and 1973 changes in compulsory school leaving age on educational attainment, measured by the proportion of respondents who left school at ages 14, 15 or 16, respectively.

60 percent left school at the age of 14, and 10 percent left school at age 15. The relationship between the two proportions practically reverses after the reform. About 55 percent of each cohort left school at age 15. 7 percent of those immediately affected by the reform still left school at age 14. In principle, nobody born in or after April should report a school leaving age of 14. This is not the case however, which might be due to misreporting, individual non-compliance, or districts failing to provide sufficient school places

immediately after the reform – as pointed out by Clark and Royer (2008). Over the years, this proportion decreased to 4 percent for the cohorts born after the first quarter of 1933. It is interesting to note the effect of the reform on the average number of years in school (see Figure 2.2).

Education has been on a secular increase for men and women. The 1947 reform has boosted this increase further but the increase at the discontinuity was much larger for women than men. Average school leaving age has

Figure 2.2
Effect of the reforms on Average School Leaving Age




Effect of the 1947 and 1973 changes in compulsory school leaving age on educational attainment, measured by the average school leaving age.

jumped by about 0.4 years for men and 0.6 years for women. The pattern of change we find for the 1973 reform is different (see lower panel of Figure 2.1). Of the pre-1973 reform cohorts, 32 percent on average left school at the age of 15. A similar proportion, 30 percent, left school at the age of 16. After the reform, the percentage of pupils leaving at age 16 increased to about 52 percent whereas the proportion of those leaving at age 15 became negligible (roughly 7 percent). During the observation period, the average number of years in school (Figure 2.2) was fairly stable for men, except for the jump of about 0.35 years induced by the 1973 reform. Education of women was still on the increase and the 1973 reform apparently only had a fairly small effect on average years in school.

Based on the described reforms, we aim at identifying the effect of schooling on health by comparing health outcomes of individuals born until March 1933 to those born in or after April 1933 and of those born in or after September 1957 to those born until August 1957. The assumption underlying our empirical approach that allows identifying a causal effect, and which is described in the next section, is that there are no unobserved cohort-level determinants of health that have changed at the time of the reform.

## 2.3 Econometric Method

The nature of the two reforms analyzed in this paper clearly makes them a candidate for a regression discontinuity design (RD). The idea of the RD approach is that the probability of receiving a particular treatment (here: an additional year of education) is a discontinuous function of a continuous treatment-determining variable (here: day of birth). This allows to estimate causal effects of the treatment by comparing outcomes (here: health) for individuals just below and just above the treatment threshold (for an overview of recent econometric developments concerning the RD design see Imbens and Lemieux (2008) and Lee and Lemieux (2009)). As documented in the preceding section, the treatment in our application is not purely assigned on the basis of the birth date (i.e. the treatment is under partial control of the individuals). After both reforms, some individuals left school at younger

ages than the legal school leaving age (at least so they said in the HSE and ELSA). They thus did not receive the treatment after the threshold date, i.e. the probability of treatment did not jump to 1. In such cases, a so-called "fuzzy" RD (FRD) design becomes appropriate. In case of a binary treatment, the FRD design is basically a Wald estimator. To see this, let Y be the health outcome, X be the date of birth as treatment-determining variable, W be the treatment received, and value c be the threshold value of the treatment-determining variable, then the FRD estimator can be written as (Imbens and Lemieux (2008)):

$$\tau_{FRD} = \frac{\lim_{x \downarrow c} E(Y|X = x) - \lim_{x \uparrow c} E(Y|X = x)}{\lim_{x \downarrow c} E(W|X = x) - \lim_{x \uparrow c} E(W|X = x)} \tag{2.1}$$

Under certain assumptions (monotonicity or no defiers, i.e. individuals do not leave school earlier *because* of the reform), and by taking limits from above and below the threshold value c, $\tau_{FRD}$ identifies the average treatment effect on the treated (averaged across all compliers at the threshold c). Take the 1947 reform as an example. If sample size was no problem, equation 2.1 would tell us to just compare the average health of all individuals born on April 1st 1933 with outcomes of all individuals born on March 31st 1933 and divide the difference (the numerator) by the difference in average school leaving ages of those two groups of individuals (the denominator).

However, sample size at the discontinuity almost always is a problem. For instance, in our pooled sample described below, we have 54 individuals each born in March and April 1933 with valid fibrinogen values. Finding significant health effects for such small samples is virtually impossible. The task is thus to appropriately estimate average outcomes and treatments *at the discontinuity* using observations that are *further away from the discontinuity*, for instance using all observations that are born four years before and after the threshold. The key issue here is how to model long-run relationships between the treatment-determining variable and the outcomes. Imbens and Lemieux (2008) suggest local linear regression, i.e. linear regressions of Y on x separately for individuals below and above the threshold (within some bandwidth h) and to predict Y at the threshold value of the treatment-

determining variable. Analogous regressions are done for W. Alternatively, one can choose some parametric form, such as a fourth-order polynomial. Lee and Lemieux (2009) recommend not to rely on a single specification. Alternative specifications, using local-linear regression and global approaches, that yield similar results lend credibility to the RD approach. One practical issue is to choose the appropriate bandwidth for the local or global regression. When we show our results we arbitrarily choose one bandwidth (4 years) and estimate local linear regressions. We present results using alternative bandwidths and alternative parametric specifications in Section 2.6 as part of our robustness checks. Another issue, recently discussed in Lee and Card (2008), is the fact that with month of birth data, i.e. with a discrete treatment-determining variable, the limits shown in equation 2.1 do not exist. As suggested in Lee and Card (2008), we account for this fact by computing cluster-corrected standard errors, where clusters are given by each value of the treatment-determining variable (month of birth).

## 2.4 Data and Descriptive Results

We use data from the Health Surveys for England (HSE) 1993, 1994, 1998 to 2000, and 2003 to 2006 and the English Longitudinal Study on Ageing (ELSA) 2006. The Health Survey for England is an annual health interview survey of around 15,000 to 20,000 respondents in England conducted by the National Centre for Social Research (separate surveys are available for Scotland and Wales). The English Longitudinal Study on Ageing is an ongoing multi-disciplinary panel survey of the older population covering around 12,000 respondents in England. It was started in 2004 based on a sample that was derived from three waves of the Health Surveys for England 1998, 1999 and 2001. Part of our ELSA sample consists of respondents already present in the HSE 1998 and 1999, i.e. some individuals are represented twice in our data. We are, however, not able to identify these respondents present in both data sources. In fact, the data use contract explicitly forbids re-identification of such respondents. The data are distributed by the Economic and Social Data Service (ESDS). We restrict our analyses to the survey years listed above because only data from these years contain infor-

mation on blood fibrinogen and CRP levels. Biomarkers are collected during nurse visits after the actual health interview and include not only blood samples but also anthropometric measurements, blood pressure measurements, and saliva samples.

We further restrict our analytical samples in two ways. First, for most of our analyses we use only birth cohorts that are born at most 4 years before and after the two relevant thresholds April 1933 and September 1957 (we lift this sample restriction when we try different bandwidth in our regression discontinuity approach). Second, we eliminate from our sample all respondents who were not born in England, Wales, or Scotland, i.e. respondents for whom it is unclear if they have been in a British school at the time of the reform.

We use two main health outcome measures: blood fibrinogen levels and blood C-reactive protein levels: For comparison with earlier studies we also analyze effects of education on self-rated general health (dichotomized to good/poor health). The blood fibrinogen level is measured in grams per liter and the blood C-reactive protein level is measured in mg per liter. One difficulty with combining biomarkers spanning more than 10 years of data collection is that measurements are not necessarily comparable across years due to changes in collection methods, assays, and laboratories. Indeed, the HSE user guide explicitly warns against comparing biomarker levels over time. In order to make our data compatible for use in a pooled data set, we have standardized all measurements to have the means and standard deviations of the 1998 measurement. Moreover, all analyses conducted include survey year fixed effects to account for differences in data collection methods over time.

As discussed in the introduction, higher levels of fibrinogen and CRP indicate the presence of inflammatory processes and have been shown to be associated with higher risks of obesity, diabetes and cardiovascular disease. In accordance with other studies analyzing the relationship of socioeconomic status and CRP levels, we exclude cases with a CRP level of over 10 mg/L from further analysis. In cases of acute inflammation CRP values can increase by as much as 10,000-fold. High CRP values might thus relate to acute inflammation and not be informative of chronic pathogenic processes (Pearson et

al. (2003)). Including these cases in the data potentially biases our results.

In our regression analyses shown below, we will use information on adult height to control for both the economic and disease environment in childhood, which can have long-lasting effects on adult health. Adult height reflects the accumulated nutritional experience during childhood including the fetal period, and is shown to have considerable predictive power both for morbidity and mortality (see Fogel (1997), Deaton (2007)) and also educational outcomes (Magnusson, Rasmussen, and Gyllensten (2006)). Controlling for height hence serves two purposes. First, in the descriptive (OLS) regressions, inclusion of height captures the effect of a potentially important third factor (childhood conditions) driving both adult health and educational outcomes. We should again stress at this point that adult height is practically determined before schooling decisions are made, either by its genetic component or by early childhood environment. Second, in the instrumental variables regressions, inclusion of height also helps controlling for unobserved cohort effects that cannot readily be captured by (local) polynomial cohort trends. Note that the first cohort affected by the first reform was born in 1933, i.e. in the immediate aftermath of the great depression, and it is a priori unclear if and how the depression has affected childhood environment (and thus adult health and education) of the cohorts in our analytical sample. For instance, we find some indication in our data that, also after controlling for cohort trends, children born after March 1933 are slightly taller than older cohorts. Table 2.1 briefly describes the analytical samples, separately for the 1947 reform cohorts (born between 1929 and 1937) and the 1973 reform cohorts (born between 1953 and 1961). Columns (1) to (4) show descriptive statistics for the full samples. The average age at survey in older cohorts is 66 years for men and 67 years for women. In the younger sample it is 41 years for both sexes. The average age at which respondents left school has increased substantially from 15.4 years for the older cohorts to 16.7 years for the younger cohorts. The proportion of respondents who reported to be in poor health is 36 percent (men) and 34 percent (women) among the 1947 reform cohorts and 17 percent (men) and 19 percent (women) among the 1973 reform cohorts. Log fibrinogen and log CRP levels are slightly higher among women and lower in the younger cohorts. Table 2.1 also shows that

Table 2.1
DESCRIPTIVE STATISTICS

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
|---|---|---|---|---|---|---|---|---|
| | Full HSE/ELSA sample | | | | With valid blood sample | | | |
| Reform Cohorts | 1947 | | 1973 | | 1947 | | 1973 | |
| Sample: | Men | Women | Men | Women | Men | Women | Men | Women |
| Age at survey | 66 | 67 | 41 | 41 | 66 | 67 | 41 | 42 |
| Age left school | 15.4 | 15.4 | 16.7 | 16.7 | 15.4 | 15.4 | 16.7 | 16.7 |
| Poor health (%) | 36 | 34 | 17 | 19 | 37 | 35 | 18 | 19 |
| Height (cm) | 172 | 159 | 176 | 163 | 172 | 159 | 176 | 163 |
| Ln(fibrinogen) | | | | | 1.00 | 1.04 | 0.86 | 0.93 |
| Ln(CRP) | | | | | 0.63 | 0.66 | 0.12 | 0.14 |
| N | 4787 | 5280 | 5925 | 7252 | 2135 | 2240 | 3074 | 3409 |

both men and women in the 1947 reform cohorts are on average 4 cm shorter than men and women in the 1973 reform cohorts.

The number of observations with valid information on fibrinogen and CRP levels is substantially lower than the full samples. Not all HSE respondents have given consent to be visited by a nurse or to have blood samples taken. Sometimes, respondents are not eligible for blood testing because of medical or other reasons. Further, it is sometimes not possible to identify blood values from samples taken from respondents and finally, some results are invalid for analysis because respondents take medication that affects blood fibrinogen or blood CRP levels. Especially non-compliance on the part of the respondents or medical ineligibility might be a cause of worry due to selection effects. Rather than dealing with this issue formally at this stage, we simply look at differences in average sample characteristics between those with valid fibrinogen/CRP levels and the full samples. As it turns out, the full sample and the sample with valid blood test data are very similar as far as observable characteristics are concerned (a more detailed analysis of participation in the nurse visit is presented in the Appendix to this chapter). Still, to get some information on the possible effect of differences between the full sample and the nurse visit sample on our regression results, we also estimated all regressions using self-rated health as outcome but restricting the sample to those also participating in the nurse visit. We find only small

changes in our results, so that we believe that sample selectivity should not be a cause of concern.

### 2.4.1 Relationship Between Self-rated Health and Biomarkers

To illustrate the correlation of traditional health measures such as self-rated health and the biomarkers used in the present study, table 2.2 shows average levels of (log) blood fibrinogen and (log) blood CRP for different levels of self-rated health, separately for the two analytical cohorts. Within each cohort and for both measures, we find a clear gradient with higher levels of fibrinogen and CRP for respondents who self-report worse health (fibrinogen and CRP levels are also highly correlated with each other, r = 0.50). The younger cohorts generally have lower values than the older cohorts even when reporting the same level of self-rated health, reflecting lower risk of cardiovascular disease. Members of the younger cohort who report to be in poor health have higher CRP levels than members of the older cohorts reporting to be in good health. Table 2.2 also documents the correlation between adult height and health measured by biomarkers. Individuals in the top half of the

Table 2.2
Relation between Self-rated Health, Height and Biomarker Levels

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
|---|---|---|---|---|---|---|---|---|
| | 1947 reform cohorts | | | | 1973 reform cohorts | | | |
| Sample: | Men | | Women | | Men | | Women | |
| | Fib | CRP | Fib | CRP | Fib | CRP | Fib | CRP |
| Self-rated health: | | | | | | | | |
| Poor | 1.05 | 0.81 | 1.08 | 0.80 | 0.91 | 0.37 | 1.01 | 0.47 |
| Good | 0.98 | 0.55 | 1.02 | 0.61 | 0.85 | 0.06 | 0.92 | 0.06 |
| abs. t-value | 6.4 | 5.6 | 5.6 | 4.2 | 5.6 | 5.0 | 8.4 | 6.3 |
| Height (relative to cohort and sex specific median): | | | | | | | | |
| Below | 1.01 | 0.64 | 1.06 | 0.70 | 0.87 | 0.16 | 0.94 | 0.21 |
| Above | 0.99 | 0.60 | 1.02 | 0.61 | 0.84 | 0.07 | 0.92 | 0.04 |
| abs. t-value | 1.8 | 1.1 | 4.5 | 2.1 | 3.1 | 1.8 | 3.3 | 3.5 |

Notes: CRP values available in 1998 and 1999 only.

cohort-sex-specific height distribution generally have lower blood fibrinogen and CRP levels than individuals in the lower half.

### 2.4.2 Descriptive Relationship Between Education and Health

Table 2.3 shows the descriptive relationship between education (measured as the age when the respondent left school) and the three health outcomes: self-rated health (again dichotomized to good versus poor), log fibrinogen level, and log CRP level. In each regression, we control for cohort (year and month of birth), season of birth, height and sex. We also control for survey year to account for possible unobserved differences across survey. For each of our measures, the results shown in table 2.3 provide evidence for a significant association between education and health. Leaving school one year later is associated with about a 5 to 6 percentage point decrease in the probability of reporting poor general health in the older cohorts and a 3 to 4 percentage point decrease in the younger cohorts. When the sample is restricted to respondents participating in the nurse visit, these associations become somewhat smaller. Also, controlling for cohort, season of birth, height, and survey year reduces the strength of the association. Still, the slope of the education-self-assessed health gradient is fairly large. It corresponds to more than ten years of age. Our findings for subjective health are corroborated by the more "objective" biomarkers. Each year of education is associated with a reduction in the blood fibrinogen level by 1.5 (women) to 1.9 percent (men) in the older cohorts and by between 1.8 percent (men) and 2.1 percent (women) in the younger cohorts. Controlling for covariates reduces this association but it remains highly significant. The effect size corresponds to about 2 to 3 years of age for men and women, respectively, i.e. the effect size is somewhat smaller than for self-rated health. For log CRP levels, effect sizes are in the range of about 3 years of age.

Table 2.3
Descriptive (OLS) Regressions of Health Measures on Education

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
|---|---|---|---|---|---|---|---|---|
| Dep. Variable: | Poor health | Poor health | Poor health$^{+}$ | Poor health$^{+}$ | log(fibrin.) | log(fibrin.) | log(CRP) | log(CRP) |
| Reform Cohorts: | 1947 | 1973 | 1947 | 1973 | 1947 | 1973 | 1947 | 1973 |
| | | | | *Men - no controls* | | | | |
| Age left school | -0.054*** | -0.044*** | -0.053*** | -0.032*** | -0.019*** | -0.018*** | -0.102*** | -0.074*** |
| | (0.004) | (0.003) | (0.006) | (0.004) | (0.003) | (0.003) | (0.018) | (0.020) |
| | | | | *Men - with control variables* | | | | |
| Age left school | -0.051*** | -0.042*** | -0.048*** | -0.030*** | -0.015*** | -0.016*** | -0.102*** | -0.073*** |
| | (0.004) | (0.003) | (0.006) | (0.004) | (0.003) | (0.003) | (0.019) | (0.020) |
| N | 5577 | 5925 | 2735 | 3074 | 2737 | 3074 | 1611 | 1630 |
| | | | | *Women - no controls* | | | | |
| Age left school | -0.063*** | -0.039*** | -0.058*** | -0.033*** | -0.015*** | -0.021*** | -0.075*** | -0.110*** |
| | (0.004) | (0.003) | (0.005) | (0.004) | (0.003) | (0.003) | (0.017) | (0.018) |
| | | | | *Women - with control variables* | | | | |
| Age left school | -0.059*** | -0.038*** | -0.054*** | -0.033*** | -0.011*** | -0.020*** | -0.070*** | -0.106*** |
| | (0.004) | (0.003) | (0.005) | (0.004) | (0.003) | (0.003) | (0.017) | (0.018) |
| N | 6199 | 7252 | 2909 | 3409 | 2911 | 3410 | 1749 | 1848 |

Notes: Cluster corrected standard errors in parentheses; * p<10%; ** p<5%; *** p<1%; Control variables include birth cohort, month or season of birth (depending on data source), height, and survey year; $^{+}$: Restricted to observations with valid fibrinogen values.

## 2.5 Regression-Discontinuity Design Results

The findings described in the preceding section reveal significant and partly sizeable associations between education and various measures of health. In this section we study whether this association is causal. As described above, we make use of two general increases in the minimum school leaving age in 1947 and 1973 that affected all cohorts born in or after April 1933 and September 1957, respectively. Results of instrumental variables regression for the fuzzy discontinuity design are shown in table 2.4. The first stage parameter shows the effect of the treatment dummy on the average school leaving age within the estimating samples. Here we find considerable differences between men and women and reform cohorts. In line with our graphical analysis in section 2.2, we find that the 1947 reform had a stronger effect on the education of women than on the education of men. In 1973, the effect was slightly stronger for men. First stage F-statistics (for the instrument) are larger or close to 10 for all regressions except one, indicating that our results do not suffer from a weak instrument problem.

Turning to the FRD parameters, we find that education has a mixed effect on health self-ratings. For men in the 1947 reform cohort, the point estimate is *plus* 2 percentage points, indicating that education might actually harm health. However, the standard error is 15 times as large as the one we get for the OLS estimate in table 2.3. Statistically, the 2 percentage points are neither different from zero nor different from the OLS estimate of minus 5 percentage points. For men in the 1973 reform cohort, our IV point estimate is negative and somewhat larger than the OLS coefficient, but again, it is neither different from zero nor different from the OLS coefficient. For women in the 1947 cohort, we obtain an IV estimate of minus 7 percentage points, i.e. a larger effect than OLS, that is statistically different from zero. In contrast, in the 1973 cohort, we find an implausibly large positive effect of education on the probability of reporting poor health. Overall, although most of these results are not inconsistent with a positive causal effect of education on health, it does also not lend much credibility to such an assertion.

Table 2.4

Fuzzy Regression Discontinuity Estimates for the Effect of Education on Health

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
|---|---|---|---|---|---|---|---|---|
| Dep. Variable: | Poor health | Poor health | Poor health$^{+}$ | Poor health$^{+}$ | log(fibrin.) | log(fibrin.) | log(CRP) | log(CRP) |
| Reform Cohorts: | 1947 | 1973 | 1947 | 1973 | 1947 | 1973 | 1947 | 1973 |
| | | | | *Men* | | | | |
| First stage parameter | 0.38*** | 0.33*** | 0.32*** | 0.32*** | 0.33*** | 0.32*** | 0.40*** | 0.39*** |
| | (0.08) | (0.07) | (0.09) | (0.10) | (0.09) | (0.10) | (0.14) | (0.13) |
| First-stage F statistic | 23.49 | 22.47 | 14.23 | 10.55 | 14.67 | 10.55 | 8.03 | 9.43 |
| FRD parameter | 0.02 | -0.08 | 0.05 | -0.08 | -0.03 | 0.01 | -0.12 | -0.18 |
| | (0.06) | (0.06) | (0.09) | (0.10) | (0.06) | (0.05) | (0.23) | (0.25) |
| N | 5577 | 5925 | 2735 | 3074 | 2737 | 3074 | 1611 | 1630 |
| | | | | *Women* | | | | |
| First stage parameter | 0.52*** | 0.31*** | 0.41*** | 0.31*** | 0.41*** | 0.31*** | 0.49*** | 0.20 |
| | (0.08) | (0.07) | (0.11) | (0.09) | (0.11) | (0.09) | (0.15) | (0.14) |
| First-stage F statistic | 37.39 | 17.92 | 14.59 | 10.95 | 14.53 | 11.09 | 10.24 | 1.95 |
| FRD parameter | -0.07* | 0.15** | -0.12 | 0.11 | 0.04 | -0.01 | -0.37 | -0.09 |
| | (0.04) | (0.06) | (0.08) | (0.08) | (0.04) | (0.05) | (0.24) | (0.49) |
| N | 6199 | 7252 | 2909 | 3409 | 2911 | 3410 | 1749 | 1848 |

Notes: Cluster corrected standard errors in parentheses; * p<10%; ** p<5%; *** p<1%; Controlling for year and month of birth, survey year, sex, and height; $^{+}$: Restricted to observations with valid fibrinogen values.

Similar to health self-assessments, we do not find convincing evidence for a significant causal effect of education on biomarker levels. Estimates for log fibrinogen levels have mixed signs and are never significantly different from zero. The coefficients for log CRP levels are negative throughout, indicating a positive effect of education on health. Effect sizes are in the vicinity of the OLS estimates - but not significantly different from zero - for men in both reform cohorts and for women in the younger reform cohort. For women in the 1947 reform cohorts point estimates are much larger than OLS estimates. Again, given the large standard errors of our estimates, a Hausman test would not reject the assumption of exogeneity of education.

## 2.6 Robustness Checks

We now discuss robustness checks of our results presented in the preceding section. Following the recommendations in Lee and Lemieux (2009), we primarily test the robustness of our results with respect to the bandwidth around the discontinuity and the functional form of the relationship between the outcome and the treatment-determining variable. Another check is to restrict our sample to respondents with either 14 or 15 years of education in case of the 1947 reform and 15 or 16 years of education in case of the 1973 reform. Among these respondents, the reforms had the largest impact on years of schooling, so that restricting the sample will increase the strength of our instrument.

Our first robustness check is to estimate the FRD parameters using local linear regression and varying the bandwidth from one year to eight years. The results are shown in table 2.5. With the exception of very small bandwidths which lead to imprecise estimates due to a substantial loss of information, the results appear to be qualitatively robust to changing the bandwidth (beginning at about $h = 2.5$ years). However, effect sizes appear to become smaller in absolute value when the bandwidth is increased but this does not necessarily affect statistical significance because estimates also get more precise.

Table 2.5

Estimates for the Effect of Education on Health: Various Bandwidths

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
|---|---|---|---|---|---|---|---|---|
| Dep. Variable: | Poor health | Poor health | Poor health$^{+}$ | Poor health$^{+}$ | log(fibrin.) | log(fibrin.) | log(CRP) | log(CRP) |
| Reform Cohorts: | 1947 | 1973 | 1947 | 1973 | 1947 | 1973 | 1947 | 1973 |
| Bandwidth | | | | | | | | |
| | | | | *Men* | | | | |
| 1 | -0.10 | 0.09 | 0.69 | 6.67 | 1.43 | 0.71 | -2.79 | 16.05 |
| 1.5 | 0.02 | -0.09 | -0.13 | -0.01 | -0.10 | -0.05 | -0.67* | -0.33 |
| 2 | 0.04 | -0.10 | -0.13 | -0.03 | -0.15* | -0.01 | -0.67** | -0.29 |
| 2.5 | 0.06 | -0.07 | -0.03 | 0.04 | -0.09 | 0.03 | -0.40 | -0.30 |
| 3 | 0.10 | -0.06 | 0.08 | 0.02 | -0.08 | 0.05 | -0.39 | -0.26 |
| 3.5 | 0.07 | -0.05 | 0.11 | -0.00 | -0.06 | 0.02 | -0.29 | -0.25 |
| 4 | 0.02 | -0.08 | 0.05 | -0.08 | -0.03 | 0.01 | -0.12 | -0.18 |
| 5 | 0.03 | -0.07 | 0.10 | -0.06 | -0.02 | -0.01 | 0.01 | -0.07 |
| 6 | 0.01 | -0.09 | 0.08 | -0.09 | -0.01 | -0.02 | -0.03 | -0.17 |
| 7 | -0.03 | -0.08 | 0.05 | -0.05 | -0.02 | -0.02 | 0.02 | -0.07 |
| 8 | -0.04 | -0.06 | 0.04 | -0.01 | -0.03 | -0.03 | -0.05 | -0.21 |
| | | | | *Women* | | | | |
| 1 | 0.79 | 0.06 | 0.10 | 0.34 | 0.03 | -0.19 | 1.78 | -0.82 |
| 1.5 | 0.00 | 0.07 | -0.14 | 0.03 | 0.02 | -0.01 | -0.23 | -0.73 |
| 2 | 0.00 | 0.10 | -0.16 | 0.10 | 0.06 | -0.04 | -0.39 | -1.24 |
| 2.5 | -0.05 | 0.13** | -0.14 | 0.20 | 0.00 | 0.06 | -0.18 | -0.70 |
| 3 | -0.04 | 0.09* | -0.11 | 0.06 | 0.00 | 0.03 | -0.21 | -0.30 |
| 3.5 | -0.05 | 0.16** | -0.11 | 0.11 | 0.04 | 0.01 | -0.26 | -0.27 |
| 4 | -0.07* | 0.15** | -0.12 | 0.11 | 0.04 | -0.01 | -0.37 | -0.09 |
| 5 | -0.08** | 0.06 | -0.15** | 0.03 | 0.02 | 0.03 | -0.17 | 0.13 |
| 6 | -0.07** | 0.08* | -0.11* | 0.02 | 0.00 | -0.01 | -0.21 | 0.10 |
| 7 | -0.06** | 0.09* | -0.10** | 0.04 | -0.02 | 0.01 | -0.20 | 0.07 |
| 8 | -0.07*** | 0.08 | -0.09** | 0.06 | -0.02 | 0.02 | -0.22 | 0.44 |

Cluster corrected standard errors in parentheses; * p<10%; ** p<5%; *** p<1%; Controlling for month of birth cohort, survey year, season of birth, and height; $^{+}$: Restricted to observations with valid fibrinogen values.

Table 2.6

Estimates for the Effect of Education on Health: various Polynomial Trends and Bandwidths

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
|---|---|---|---|---|---|---|---|---|
| Dep. Variable: | Poor health | Poor health | Poor health$^{+}$ | Poor health$^{+}$ | log(fibrin.) | log(fibrin.) | log(CRP) | log(CRP) |
| Reform Cohorts: | 1947 | 1973 | 1947 | 1973 | 1947 | 1973 | 1947 | 1973 |
| Polynomial (k), Bandwidth (h): | | | | | | | | |
| | | | | *Men* | | | | |
| k=1, h=4 | 0.02 | -0.08 | 0.05 | -0.08 | -0.03 | 0.01 | -0.12 | -0.19 |
| k=2, h=4 | 0.02 | -0.08 | 0.05 | -0.08 | -0.03 | 0.01 | -0.12 | -0.18 |
| k=3, h=4 | 0.12 | -0.06 | 0.03 | 0.10 | -0.11 | 0.03 | -0.64 | -0.39 |
| k=4, h=4 | 0.12 | -0.06 | 0.04 | 0.10 | -0.11 | 0.03 | -0.64 | -0.39 |
| k=1, h=8 | -0.04 | -0.07 | 0.04 | -0.01 | -0.03 | -0.03 | -0.05 | -0.23 |
| k=2, h=8 | -0.04 | -0.06 | 0.04 | -0.01 | -0.03 | -0.03 | -0.06 | -0.22 |
| k=3, h=8 | 0.04 | -0.09* | 0.11 | -0.08 | 0.01 | -0.02 | 0.02 | -0.14 |
| k=4, h=8 | 0.04 | -0.09* | 0.11 | -0.08 | 0.01 | -0.02 | 0.02 | -0.14 |
| | | | | *Women* | | | | |
| k=1, h=4 | -0.07* | 0.15** | -0.12 | 0.11 | 0.04 | -0.01 | -0.37 | -0.10 |
| k=2, h=4 | -0.07* | 0.15** | -0.12 | 0.11 | 0.04 | -0.01 | -0.36 | -0.09 |
| k=3, h=4 | -0.02 | 0.12 | -0.13 | 0.12 | 0.02 | 0.03 | -0.15 | -0.66 |
| k=4, h=4 | -0.02 | 0.12 | -0.13 | 0.12 | 0.02 | 0.03 | -0.16 | -0.64 |
| k=1, h=8 | -0.07*** | 0.08 | -0.09** | 0.06 | -0.02 | 0.02 | -0.22 | 0.39 |
| k=2, h=8 | -0.07*** | 0.08 | -0.09** | 0.06 | -0.02 | 0.02 | -0.22 | 0.44 |
| k=3, h=8 | -0.06* | 0.10** | -0.16** | 0.03 | 0.01 | -0.01 | -0.20 | -0.16 |
| k=4, h=8 | -0.06* | 0.10** | -0.16** | 0.03 | 0.01 | -0.01 | -0.20 | -0.16 |

Cluster corrected standard errors in parentheses; * p<10%; ** p<5%; *** p<1%; Controlling for survey year and height, season of birth; $^{+}$: Restricted to observations with valid fibrinogen values.

Table 2.7

Estimates for the Effect of Education on Health:Restricted Samples

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
|---|---|---|---|---|---|---|---|---|
| Reform Cohorts: | 1947 | 1973 | 1947 | 1973 | 1947 | 1973 | 1947 | 1973 |
| | | | | *Men* | | | | |
| OLS parameter | -0.067*** | -0.086*** | -0.050* | -0.080*** | -0.023 | -0.031** | -0.194*** | -0.169** |
| | (0.021) | (0.019) | (0.030) | (0.022) | (0.015) | (0.015) | (0.073) | (0.070) |
| IV First stage | 0.63*** | 0.33*** | 0.67*** | 0.30*** | 0.67*** | 0.30*** | 0.74*** | 0.34*** |
| parameter | (0.03) | (0.03) | (0.03) | (0.04) | (0.03) | (0.04) | (0.04) | (0.06) |
| First-stage F-statistic | 499.92 | 111.48 | 417.41 | 49.26 | 422.46 | 49.26 | 365.18 | 30.42 |
| FRD parameter | -0.02 | -0.10 | -0.03 | -0.07 | -0.03 | 0.06 | -0.12 | 0.32 |
| | (0.05) | (0.09) | (0.06) | (0.14) | (0.04) | (0.08) | (0.19) | (0.41) |
| N | 3690 | 3496 | 1790 | 1800 | 1792 | 1800 | 1077 | 965 |
| | | | | *Women* | | | | |
| OLS parameter | -0.086*** | -0.083*** | -0.079** | -0.110*** | -0.013 | -0.015 | -0.123 | -0.213** |
| | (0.021) | (0.017) | (0.031) | (0.022) | (0.015) | (0.014) | (0.079) | (0.092) |
| IV First stage | 0.63*** | 0.41*** | 0.59*** | 0.43*** | 0.59*** | 0.43*** | 0.62*** | 0.41*** |
| parameter | (0.02) | (0.03) | (0.04) | (0.03) | (0.04) | (0.03) | (0.05) | (0.04) |
| First-stage F-statistic | 741.47 | 190.28 | 269.84 | 161.49 | 270.34 | 161.49 | 181.49 | 91.69 |
| FRD parameter | -0.03 | 0.15*** | -0.04 | 0.08 | 0.03 | -0.06 | -0.30 | -0.27 |
| | (0.06) | (0.05) | (0.09) | (0.08) | (0.03) | (0.05) | (0.19) | (0.33) |
| N | 4009 | 4064 | 1815 | 1892 | 1817 | 1892 | 1059 | 1009 |

Cluster corrected standard errors in parentheses; * p<10%; ** p<5%; *** p<1%; Controlling for year and month of birth, survey year, sex, and height; $^{+}$: Restricted to observations with valid fibrinogen values.

Changes in the size of the estimates suggest that results might be sensitive to how one models non-linearities in cohort effects. We have thus also experimented with alternative specifications using polynomial cohort trends of varying degrees on samples of varying bandwidths (see table 2.6). This exercise essentially confirms our findings based on local linear regressions. Regression results based on the restricted samples are shown in table 2.7. Notably, whereas the association between years in school an health usually gets larger (as indicated by the OLS regression parameters also reported for comparison purposes), it partly loses significance. This might not only be due to smaller sample sizes, but also due to less variation in the education variable.

## 2.7 Discussion

In this paper we use data from several rounds of the Health Survey for England and the English Longitudinal Study on Ageing to estimate the causal effect of education on health. Our identification comes from two increases in mandatory school leaving age in 1947 and 1973. We are not the first to exploit these reforms for causal analyses in a regression discontinuity approach. However, the specific contribution of our paper is the use of biomarkers (blood fibrinogen and C-reactive protein) in addition to health self-reports as health outcome measures. We do not argue that biomarkers represent "better" or "more precise" measures of health. Rather, we use them as indicators of health that are complementary to subjective measures such as self-rated health. We analyze blood fibrinogen and blood C-reactive protein because high levels in each are known risk factors for cardiovascular disease. Thus, our analyses allow identifying whether education has a causal effect not only on manifest conditions but also on the risk of developing a disease.

While education is clearly correlated with lower levels of fibrinogen and C-reactive protein (indicating worse health and higher risk of cardiovascular disease for respondents with a smaller number of years in school), our results contain no evidence for a significant *causal* effect of education on the examined biomarkers. However, we find some evidence for an effect of education

on self-rated health among women. A puzzling finding in this context is that education appears to cause poor health in the younger reform cohorts.

Overall, our results are only partly in line with earlier studies analyzing the effect of the 1947 and 1973 UK education reforms on health outcomes. For instance, one earlier study using the same data set and exploiting the 1947 reform only (Clark and Royer (2008)) finds no significant effect of education on self-rated health. This is most likely due to the fact that – in contrast to Clark and Royer (2008) – we perform separate analyses for men and women. Pooled analyses of our data would as well yield insignificant effects and lead us to conclude that there is only weak evidence for a causal effect of education on subjective health. Earlier work using different data but exploiting the same reform, such as Oreopoulos (2006) or Silles (2009), generally finds significant positive effects of education on self-rated health. With respect to more "objective" health measurements (log fibrinogen and CRP blood levels), our finding confirms the findings in Clark and Royer (2008) (looking at body mass index and blood pressure) but is at odds with other studies using objective outcomes such as mortality (Lleras-Muney (2005)).

Currently, we can only speculate about the reasons for the differences in results across different studies that all use credible identification strategies. Although a causal effect of education on health (both direct and indirect) is theoretically plausible, we believe that there are a couple of reasons why the effect may actually be hard to identify in observational studies. As other authors, we use regression discontinuity in connection with changes in compulsory schooling for identification. It is well known that RD estimators have good internal validity but that external validity is a problem. For instance using our fuzzy RD design, we are (only) able to identify local treatment effects, i.e. for compliers at the cut-off date. The main effect of the reforms studied in the present paper on education was to keep those who wanted to quit as early as possible in school for one more year. One important question is what has been learned in this one year? There is some evidence suggesting that this year had some positive effect on the wages of the affected cohorts (Oreopoulos (2006), Devereux and Hart (2010)), i.e. something has been learned and people have become more productive in the labor market (the

higher income apparently had no sizeable impact on health). One explanation for the absence of an effect on objective measures of health is that what needs to be learned to make individuals more productive producers of their own health is different from skills that are valued in the labor market. It is likely that the emerging literature on health literacy sheds more light on this issue (Nutbeam (2008)).

Another reason why we may not find good evidence for a causal effect of education on health measured by biomarkers is that our samples are limited to individuals born shortly before and after the relevant cut-off dates for being affected by the reforms. The reason not to choose too wide intervals is obvious. The more birth cohorts are included, the harder it becomes to maintain the assumption that no unobserved factors that influence health have changed in parallel to the reform. One example for such unobserved factors affecting the validity of the instrument is medical progress. Put differently, the instrument loses validity when the sample is extended too far because the exclusion restriction does no longer hold. Of course, by including (local) cohort trends and adult height we hope to account for unmeasured factors, but the risk that the cohort trend is incorrectly specified rises with the number of cohorts included in the regression. The downside of "staying close" to the discontinuity is that the number of observations may quickly become too small to get precise estimates. This may also be one explanation for our non-findings with respect to biomarkers. However, we do find significant effects of education on self-rated health in samples of similar size. The contradiction between results for self-rated health and biomarkers could thus also be due to differential reporting styles of respondents of different education levels. This issue has raised some attention in the recent literature (e.g. Bago, O'Donnell, and van Doorslaer (2008), Jürges (2008)), but cannot be solved in the present paper.

Finally, it must also be noted that our parameters only identify the effect of education for compliers to the two specific reforms of raising mandatory school leaving age. Interventions at other stages of the life-cycle might have more systematic causal effects on health. For instance, a recent strand of the human capital literature has stressed the importance of early childhood ed-

ucation for the development of cognitive and non-cognitive skills (Cunha et al. (2006)). If early childhood education changes the whole lifetime path of human capital accumulation, early interventions might substantially improve health, while later life interventions like increasing the number of school years remain largely ineffective.

It is therefore important to investigate the effect of education on health at other margins of education. For instance, the inclusion of specific health subjects into the general curriculum might be different from a general increase in the years in schooling. Alternatively, pre-school programs that provide administered healthy food servings might have permanent effects on eating patterns. Therefore only studies that test more specific hypotheses or that evaluate more targeted interventions will shed further light on the effect of education on health status.

## 2.8 Appendix: Nurse Visits and Selectivity of the Biomarker Samples

In this appendix, we look at possible differences between individuals with valid measurements for blood fibrinogen and blood CRP and the full HSE samples. A non-valid blood fibrinogen or CRP level measurement can have a variety of reasons and can essentially be described as a multi-stage process. To get a valid blood sample, the following conditions have to be fulfilled. First, the respondent has to agree to the nurse visit. Second, the nurse visit actually has to take place. Third, the respondent has to be scheduled for the blood sample. For instance, in 1998 respondents younger than 18 were not asked for a blood sample. Fourth, the respondent is not ineligible for blood sampling due to medical reasons (pregnant women, people with clotting or bleeding disorders, and people with a history of fits or convulsions). Fifth, respondents have to agree to have their blood drawn. Sixth, the nurse has to be successful in drawing blood and seventh, the laboratory has to be successful determining the fibrinogen and CRP levels.

To illustrate the relative importance of various reasons for not obtaining a valid blood sample, we focus on the 1998 round of HSE. Of 15,447 respondents 18 or older, only 1,011 or 6.5 percent refused a nurse visit at the time of the individual interview. The main reasons given for the refusal were that respondents had already given enough time for the survey (25 percent), had enough medical tests recently (19 percent), were not interested (18 percent) or were too busy (17 percent). Of the 14,436 respondents who agreed to the nurse visit, 9 percent refused the nurse visit later. Of the 13,197 respondent visited by a nurse, 6 percent were not scheduled for the blood sample due to medical reasons and 10 percent refused the blood sample. The main reasons given for refusing the blood sample were fear of needles (60 percent), recently had a health check (20 percent), and previous difficulties with venepuncture (16 percent). For 12 percent of the 11,238 respondents consenting to have their blood drawn, no usable samples to determine fibrinogen levels could be obtained by the nurse (numbers vary a little bit for CRP). The number of successfully determined blood fibrinogen levels was 9,913 but 607 cases were

excluded from further analysis because respondents took medication affecting fibrinogen levels (this restriction does not apply to CRP levels). Eventually, 60 percent of the age-eligible sample have usable data on blood fibrinogen levels and 67 percent have usable information on blood CRP levels.

It is beyond the scope of this appendix to analyze the empirical determinants for non-participation in the nurse visit or the blood sampling in much detail. Some multiple regression analyses are shown table 2.8 which contains the results of probit regressions explaining the probability of participating in the nurse visit and agreeing to the blood sample (conditional on participating in the nurse visit) for our 1947 and 1973 reform cohorts. We estimate a separate regression for each reform cohort. The coefficients reported represent marginal effects.

First, we note that across the four survey years, participation rates for nurse visits and conditional participation in the blood sample are close to 90 percent for each reform cohort. Second, demographic and health variables are jointly significant only as explanatory variables for agreeing to the blood sample, not for agreeing to the nurse visit per se. We find that women are generally less likely to participate in nurse visits and blood sampling. Education appears to have mixed effects on the probability of participating in nurse visits and blood sampling. We find a significant positive effect of 1.1 percentage points per year only on the willingness of giving blood in the younger sample. Further, they have opposite signs in both samples. Being in good health has a positive effect on participation in the blood sample. A more detailed analysis of the reasons for refusing, by self-rated health status, reveals that those in poor health were much more likely to have had a recent blood test and that they were currently too ill, whereas those in good health were more likely to express a fear of needles. Finally, we note that willingness to participate in the nurse visit was particularly low in the 1999 survey.

Overall, our regression results suggest a certain degree of selectivity of our biomarkers samples with respect to both health and education so that sample selection bias is a potential problem. Considering the subject of our analysis, it would of course be hard to find valid exclusion restrictions for health and education to estimate a sample selection model. To deal with possible

selection biases we check whether we find any differences in regression results for self-rated health when using the full sample and when using the selective sample of respondents for which we have valid blood measurements. As noted in the text, we find only small changes in our results, so that we believe that sample selectivity should not be a cause of concern.

Table 2.8
PROBIT REGRESSIONS EXPLAINING NON-REFUSAL TO NURSE VISIT AND BLOOD SAMPLE

| | (1) | (2) | (3) | (4) |
|---|---|---|---|---|
| Agree to ... | nurse visit | blood sample | nurse visit | blood sample |
| Reform Cohorts: | 1947 | | 1973 | |
| Age (in years) | 0.002 | -0.005 | 0.004 | 0.005 |
| | (0.004) | (0.004) | (0.003) | (0.003) |
| Female | -0.001 | -0.025* | -0.023* | -0.015 |
| | (0.013) | (0.013) | (0.011) | (0.011) |
| Age left school | -0.001 | -0.005 | 0.002 | 0.011** |
| | (0.005) | (0.005) | (0.004) | (0.004) |
| Good health | 0.011 | 0.087** | 0.008 | 0.042* |
| | (0.014) | (0.015) | (0.016) | (0.017) |
| Year 1998 | -0.040 | -0.006 | -0.039 | -0.048* |
| | (0.025) | (0.024) | (0.020) | (0.019) |
| Year 1999 | -0.264** | 0.004 | -0.183** | -0.037 |
| | (0.066) | (0.046) | (0.041) | (0.034) |
| Year 2000 | -0.032 | -0.065 | | |
| | (0.036) | (0.040) | | |
| N | 2890 | 2506 | 3352 | 2937 |
| Average percentage | 86.7 | 87.7 | 87.6 | 90.4 |
| Chi-squared test for health and demographics | 1.02 | 42.35** | 6.03 | 20.88** |

Marginal effects; standard errors in parentheses; * significant at 5%; ** significant at 1%

# Chapter 3

# Parental Income and the Dynamics of Health Inequality in Early Childhood – Evidence from the United Kingdom

## 3.1 Introduction

There is widespread evidence on the positive relationship between socioeconomic status and health, often referred to as "the Gradient". Starting with the seminal work by Case, Lubotsky, and Paxson (2002), various studies confirmed that this gradient has antecedents in childhood, i.e. children from high-income families have on average better health than those from low-income families.[1] The mechanisms that underlie this phenomenon are not well understood yet. Several studies pointed out that the gradient becomes steeper as children age in a number of countries, including the US, Canada,

[1] In fact, in epidemiology, the first evidence regarding the relation between socioeconomic status and child health was published much earlier (e.g. Egbuonu and Starfield (1982)).

and Australia.[2] While the existence of the gradient of health in childhood is an established finding in the literature using European micro data, the evidence regarding the evolution of the gradient in childhood is more ambiguous. Case, Lee, and Paxson (2008) and Currie, Shields, and Price (2007) provided cross-sectional evidence backing that inequality increases until the age of 8 years in the United Kingdom. For older children, the findings are sensitive with respect to the time period considered. The results in Currie, Shields, and Price (2007) support the hypothesis that inequality diminishes for older age groups, whereas Case, Lee, and Paxson (2008) find that health inequality persists or even grows larger for older age groups. Propper, Rigg, and Burgess (2007) question whether inequality increases throughout early childhood in the UK at all. Recent evidence from Germany suggests a similar pattern: while there is a strong case for the existence of the health gradient, this gradient does not become steeper for older children (Reinhold and Jürges (forthcoming)). These findings suggest that health inequality is largely determined before birth and in the very first years of life and therefore motivate focussing research on the drivers of health inequality in early childhood.
Building on this prior evidence, this paper investigates the evolution of health inequality by following up children from a British cohort study throughout early childhood. It is the motivation of this paper to better understand the formation of the gradient in the first years of life. Our hypotheses are guided by the insight that parental income potentially affects a child's health through two different channels. On the one hand, it can be used to maintain good health. On the other hand, it can be used to restore good health once a child has been afflicted by some condition. This article makes two specific contributions. First, we test for the presence of these two effects with panel data from a European country. Second, we extend this framework by investigating whether the persistence of diseases is related to parental income.

In line with the previous literature, we distinguish two possible explanations for the finding that the health gap between children from high income households and those from low income households widens with age. First, higher parental income could be associated with a lower probability of suffering

[2]For the US Case, Lubotsky, and Paxson (2002) and Condliffe and Link (2008), for Canada: Currie and Stabile (2003), for Australia: Khanam, Nghiem, and Connelly (2009).

from certain diseases. Hence, with age children from low-income families possibly accumulate a higher number of diseases and therefore the health gap between children from high income families and those from low income families widens. Evidence supporting the hypothesis that parental income decreases the risk of incurring diseases has been provided by analyses based on American and Canadian data. Currie and Stabile (2003) argue that it is this mechanism that explains why the gradient becomes steeper for older children in Canada.[3]

Second, income can mitigate the negative consequences of health conditions. Previous studies have shown that conditional on having a particular disease, children from low-income households are more likely to translate this condition into poor subjective health status and that this mechanism partially explains the evolution of health inequality in the US (Case, Lubotsky, and Paxson (2002), Condliffe and Link (2008)). Taking advantage of the panel dimension of our data, we investigate whether the consequences of health conditions depend on parents' income in the United Kingdom.

Besides the assessment of these two explanations, we shed light on the question why children from low income families are more likely to report poor subjective health status conditional on having a disease. We identify one possible channel by again exploiting the panel dimension of our data. Parental income could be related to the duration of a particular disease a child suffers from. We test this hypothesis by looking at the association between parental income and the probability of retaining diseases from one period to the next.

Similar to previous studies, we find strong evidence for an association between parental income and child health. Our results indicate that children from low income households are more susceptive to health conditions of the respiratory system. Moreover, children from low-income families are more likely to translate longstanding health conditions into poor subjective health status. Our analyses show that even 5 years after a certain health condition arrived, children from low income households have a higher probability of poor health. This result indicates that parental income plays an important role in buffering the consequences of health shocks. This evidence also sug-

[3]For the US, results have been provided by Case, Lubotsky, and Paxson (2002), Condliffe and Link (2008), and for Canada by Currie and Stabile (2003).

gests that the way children cope with diseases in part explains the fact that inequality increases as children age in the very first years of life, even though by virtue of the British National Health Service (NHS) all children have equal access to medical care. This finding is new to the literature. Even though the Canadian health insurance system is roughly comparable to the British one, Currie and Stabile (2003) provide evidence that there is no such effect in Canada by applying a methodological setup similar to ours.
Moreover, we find that children from low-income families are more likely to keep certain adverse conditions for a longer period.

One source of concern for the interpretation of previous studies is the possible endogeneity of parental income. Endogeneity can arise for two reasons. Parents might react to their child falling ill by reducing labor supply. Thus, a correlation of parental income with poor child health possibly reflects causality running in both directions. In addition to this problem of reversed causation, there is a second concern for endogeneity, as both parental income and child health might be due to common causes such as parental health or child health in earlier periods. Propper, Rigg, and Burgess (2007) suggest that once controlling for parental health status, the evidence for an effect of parental income on child health almost disappears.
Our approach tackles several of these concerns. The availability of four waves of the Millennium Cohort Study allows to use additional information on parents' background as well as on a child's health status at birth. As we are interested in events that occur after birth, controlling for birth weight allows us to keep initial health stock constant and also proxies for the quality of nutritional intake before birth and parental risk behavior. We average income over four waves of the Millennium Cohort Study, as this reduces the sensitivity to short-run reductions in income due to reduced parental labor supply.
Finally, we assess to what extent mothers reduce their labor supply in response to their child falling ill. The analysis provides only weak support for the hypothesis that mothers reduce their labor supply when their child suffers from ill health.

This paper is organized as follows: the next section lays out the empirical

framework of our analysis. In section 3.3, our data set is introduced. Section 3.4 presents our results. We first inspect the relation between parental income and the prevalence of diseases (section 3.4.1) and then analyze the impact of contemporary and lagged health conditions on various health outcomes. In section 3.4.2, the consequences of health conditions on subjective health status are investigated. Section 3.4.3 shows evidence on the relation between the persistence of diseases and parental income. In section 3.5.1, we inspect the problem of reversed causation by investigating the labor supply reactions of mothers to ill health of their child. The last section summarizes our findings and concludes.

## 3.2 Conceptual and Empirical Framework

This section lays out a conceptual framework for the empirical analysis. It is our objective to explain why health inequality increases as children grow older. Following the literature, we distinguish two effects that can explain this pattern.

### 3.2.1 Prevalence of Health Conditions by Parental Income

Health disparities could become more pronounced for older children just because children from poor families accumulate a higher number of health conditions as they age. There are several arguments that substantiate this hypothesis. It is obvious that it is not income per se that protects a child's health status but investments in health status that become affordable with higher income. Living conditions in specific areas are reflected in housing prices. It is therefore not surprising that parental income is correlated with the quality of housing as characterized by air pollution and the incidence of violence (McLoyd (1998), Chen, Matthews, and Boyce (2002)). Moreover, parental income has been shown to correlate with the quality of the nutrition children get (Patrick and Nicklas (2005)).[4] All these material factors are

[4]For a very extensive review of the epidemiological literature on the relation between socioeconomic status and child health, see Chen, Matthews, and Boyce (2002).

likely to affect the probability of developing health conditions in the medium term. However, it is not only for diseases that result from exposure to toxic substances or other living conditions that one could expect to find higher prevalence rates for children from poor families. For almost any health condition a theoretical argument can be made, why one would expect a gradient in socioeconomic status (SES). For example, some evidence suggests that individuals with low SES live and work in more stressful environments (e.g. Adler and Newman (2002)). If parents pass on this stress to their offspring, children from low SES households could be more susceptive towards mental diseases. We therefore do not restrict the analysis to health conditions for which there already is a proven etiological link with parental income, but rather investigate the full spectrum of health conditions.
To test the hypothesis that the prevalence of diseases is related to parental income, we estimate regression models of the following form:

$$C_{it} = \alpha_0 + \alpha_1 Inc_i + X_{it}\delta + u_i \tag{3.1}$$

$C$ represents an indicator for a particular health condition that takes the value 1 if child $i$ is reported to suffer from this condition at time $t$, $Inc_i$ reflects the household income of child $i$ and $X_{it}$ includes other health relevant covariates. Income does not have a time index as it is averaged over all available observations of household income. We control for other dimensions of socioeconomic status such as parental education and a dummy variable that indicates the belonging to an ethnic minority. $X$ also includes a control for (the logarithm of) birth weight to proxy for health status at birth as well as for the quality of care that parents provide. Birth weight has been shown to be associated with parental risk behavior and poor nutritional intake during pregnancy (e.g. Meara (2001), Blake et al. (2000), Kramer (1987)). Therefore controlling for birth weight allows to proxy for the effect of (unobservable) parental quality which likely confounds the effect of parental income.
We assess whether the prevalence of health conditions is related to parental income by estimating $\alpha_1$ separately for the second, third, and fourth wave of the MCS. If $\alpha_1$ is smaller than zero, higher parental income is associated

with a lower probability of suffering from a certain illness.

### 3.2.2 Consequences of Health Conditions by Parental SES

The second hypothesis we test states that parental income affects a child's health status by mitigating the negative consequences of health conditions. If this effect was available, the gradient would become steeper just because higher income equips families to better cope with illnesses. Socioeconomic status has been shown to be related to the quality of care received (Williams (1990)) which could result in faster recoveries from diseases. It is also possible that the quality of physicians and hospitals is lower in deprived areas as compared to high SES areas. Moreover, children from high-income families have more attentive parents who consult professional care at an earlier stage. This is reflected in the fact that children from low SES families having less regular visits at physicians but higher probabilities to make use of emergency units in hospitals (Naclerio, Gardner, and Pollack (1999), Pamuk et al. (1998)).
For these reasons high income parents are possibly better able to cushion the consequences of adverse health conditions that occurred to their offspring.

To test for this effect, we estimate the following models:

$$PoorHealth_{it} = \beta_0 + \beta_1 C_{it} + \beta_2 Inc_i + \beta_3 C_{it} * Inc_i + \beta_4 X_{it} + \epsilon_i \quad (3.2)$$

$$PoorHealth_{it} = \beta_0 + \beta_1 C_{it-n} + \beta_2 Inc_i + \beta_3 C_{it-n} * Inc_i + \beta_4 X_{it} + \epsilon_i \quad (3.3)$$

where $PoorHealth_{it}$ represents an indicator for poor health status of child $i$ at time $t$, $C$ is an indicator for a particular health condition, $Inc_i$ represents the household income of child $i$ and $X_{it}$ includes the same covariates as above. The dependent variable takes a value of 1 if child $i$ has good, fair or poor subjective health status and 0 otherwise.
The coefficient for the interaction effect between income and health conditions, $\beta_3$, indicates whether among those children afflicted by a specific condition in period $t$, income cushions the consequences of this condition. A negative estimate for $\beta_3$ suggests that children from high income families are

better able to cope with diseases. The difference between equations 3.2 and 3.3 is that we estimate the effect of lagged health conditions in equations 3.3, as opposed to the immediate effect of health conditions in equation 3.2. In equation 3.3, we investigate whether the medium term consequences of health conditions are buffered by income. We use the one wave (about 2.5 years) and two waves (about 5 years) lags to trace the differential effect of illnesses over time.
We look at 9 specific conditions but also estimate equations 3.1-3.3 for an indicator that equals 1 if child $i$ has any longstanding condition, similar to previous related research (e.g. Condliffe and Link (2008)).

One problem can come up when using an indicator for longstanding conditions instead of looking at specific conditions to explain subjective health status in equation 3.2: If children from poorer households are more likely to suffer from severe health conditions than high-income children, the indicator of longstanding conditions will represent on average lighter conditions for these children. In this case, $\beta_3$ not only reflects that children are differently affected by diseases, depending on parental income, but also that children are suffering from different diseases, depending on parental income. This makes the cushioning effect of income and the prevalence effect of income indistinguishable. We are therefore convinced that it is important to estimate equations 3.2 and 3.3 for specific health conditions. However, there is also a point for collapsing indicators of single conditions to one indicator that represents the presence of any longstanding condition. The estimation of the interaction effects relies on those children that were affected by a condition. However, some specific conditions affect only few children. The estimation of the corresponding effects therefore likely lack precision. To make sure that the interaction effects ($\beta_3$) are estimated with sufficient precision, we impose a minimum of 55 cases for each condition and period. If there are less than 55 cases, children are categorized as suffering from "other conditions".

### 3.2.3 Is Persistence of Diseases Related to Parental Income?

We hypothesize that children from low-SES families are more negatively affected by health conditions over time because the duration of diseases is affected by parental income. The third hypothesis we test states that the persistence of diseases is linked to parental income. We term this effect the **recovery effect** of income and formalize it as follows: conditional on having a particular disease, higher income is associated with a lower probability of still having this disease after $n$ periods. We are not aware of any previous study linking the duration of a comprehensive set of health conditions to parental income. We will test for this mechanism by looking at the probability of suffering from a disease $C$ in period $t$, conditional on having reported this condition in the previous survey:

$$C_{it} = \gamma_0 + \gamma_1 C_{it-n} + \gamma_2 Inc_i + \gamma_3 C_{it-n} * Inc_i + \theta X_{it} + v_{it} \qquad (3.4)$$

If the probability of recovering from a disease in $t - n$ positively depends on parental income, then this will be reflected in a negative sign of the coefficient $\gamma_3$.

We do not estimate family fixed effects regressions of equations 3.1-3.4 for two reasons. First, fixed effects regressions would calculate the effect of changes in parental income over time on a child's health status. Since we are interested in the effect of permanent income (rather than transitory shocks to income), we do not consider that changes in income are a better proxy for permanent income than the mean of income averaged over four waves. Second, we estimate several dynamic regression equations that use lagged values as dependent variables. We would need more observations per individual in order to estimate these dynamic regression models with individual fixed effects.

## 3.3 Data and Descriptive Statistics

We use data from the British Millennium Cohort Study (MCS). The MCS is a longitudinal study that complements previous British cohort studies that were based on individuals born in 1958 and 1970, respectively. The MCS started in 2001 with a sample of 18.818 infants at the age of about 9 months from England, Ireland, Scotland and Wales. Interviews have been conducted every two to three years since. The main interview partner in almost all cases is the natural mother. MCS also comprises partner interviews, cognitive tests (in later waves) and teacher interviews to monitor the mental, physical and cognitive development of the cohort members.[5] The MCS questionnaires cover several topics besides health status such as parental socioeconomic and occupation status, details on the pregnancy, information on the time use of the cohort members and their parents as well as questions regarding the development of the child's character and abilities. Our main analysis is based on the second, third and fourth wave of MCS.[6] Due to changes in the question style, we cannot use information on diseases from the first wave of MCS. However, we extract important information such as birth weight and parental income from the first wave.

Table 3.1 presents descriptive statistics of our data. 15% of all children in the second and 19% in the third and fourth wave are reported to suffer from longstanding conditions at the time of the interview. Only 1.6% of all children in the second wave and 3.6% of all children in the third wave have more than one severe condition. We analyze the prevalence and impact of those 9 health conditions that are most common in the sample as well as an indicator that captures all remaining (but less common) severe afflictions and one indicator that equals one if a child has any condition. The basis of our classification of diseases are ICD-10 categories. We impose a minimum of 55 cases for each condition and wave. All conditions below that prevalence threshold have been coded as "other condition". Diseases of the respiratory system as well as skin diseases are the most frequent conditions, affecting

[5]Access to the data via the UK data archive, University of Essex, is gratefully acknowledged.

[6]We abbreviate the single waves of MCS by MCS2, MCS3 and MCS4 in the following.

Table 3.1
Descriptive Statistics

| Variable | Mean | Std. Dev. | N |
|---|---|---|---|
| Child's Age in years (MCS1) | 0.81 | 0.04 | 18,552 |
| Child's Age in years (MCS2) | 3.14 | 0.21 | 15,582 |
| Child's Age in years (MCS3) | 5.22 | 0.24 | 14,677 |
| Child's Age in years (MCS4) | 7.23 | 0.25 | 13,855 |
| Main Respondent not nat. mother (MCS2) | 0.02 | | 15,588 |
| Main Respondent not nat. mother (MCS3) | 0.03 | | 15,244 |
| Main Respondent not nat. mother (MCS4) | 0.03 | | 13,855 |
| **Demographics (MCS1):** | | | |
| Household Size | 4.01 | 1.34 | 18,552 |
| Mother's Age at Birth | 28.3 | 5.96 | 18,513 |
| Mother's Age when left School | 17.46 | 2.45 | 18,324 |
| Household's Net Income | 21,337 | 16,848 | 16,941 |
| Lone mother | 0.19 | | 18,552 |
| Mother not working | 0.53 | | 18,552 |
| Migrant Household | 0.14 | | 18,552 |
| **Health Variables:** | | | |
| Birth weight (kg) | 3.34 | 0.589 | 18,487 |
| Fraction low birth weight ($<$ 2.5 kilogram) | 0.078 | | 18,353 |
| Prevalence of Longstanding Condition (MCS1) | 0.55 | | 18,552 |
| Prevalence of Longstanding Condition (MCS2) | 0.15 | | 14,898 |
| Prevalence of Longstanding Condition (MCS3) | 0.19 | | 14,678 |
| Prevalence of Longstanding Condition (MCS4) | 0.19 | | 13,855 |
| Share more than 1 Condition (MCS2) | 0.016 | | 14,898 |
| Share more than 1 Condition (MCS3) | 0.036 | | 14,678 |
| Poor Health (MCS3) | 0.18 | | 15,168 |
| Poor Health (MCS4) | 0.13 | | 13,779 |

4-8% of the sample. Illnesses of the nervous system as well as eye problems are the most rare conditions. Only about 60 children are reported to suffer from these conditions in each wave, which corresponds to about 0.3% of the sample.[7]

As in Currie and Stabile (2003) and Condliffe and Link (2008), our main outcome variable of interest to assess the consequences of diseases is an indicator for subjective health status, which has been assessed in the third and fourth wave of the MCS. Subjective health status has been evaluated by the

[7] A detailed summary of all prevalence rates can be found in the chapter appendix.

main respondent in the MCS, which is in almost all cases the natural mother of the cohort members. It is measured on a five item scale from excellent to poor. Subjective health status is an important marker of health. It has been shown that subjective assessments of health status significantly predict future health status even when controlling for objective indicators of health status (Idler and Benyamini (1997)). We collapse subjective health status into a binary indicator that equals 1 if a child is in good, fair or poor health. 18% of all children in the third wave and 13% in the fourth wave are reported to suffer from poor health according to our measure.
Income in the MCS is reported as total annual household income in income bands. We take the middle of each income band and transform nominal income to real income by dividing by the UK consumer price index.
To calculate the relevant measure of income, we take the average of total household income over all available waves, take the logarithm hereof and finally subtract the sample mean, as suggested by previous studies on this subject (Case, Lubotsky, and Paxson (2002)). The first step ensures that we are as close as possible to permanent income, which we consider the relevant measure of income. The second step accounts for a decreasing marginal effect of income on health. The last step facilitates the interpretation, as we evaluate the effect of income at the sample mean in all subsequent analyses. We discard observations with missing information on income, which accounts for about 10% of all observations.

## 3.4 Results

### 3.4.1 Parental Income and the Prevalence of Health Conditions

This section presents the results for the test of the hypothesis that parental income is related to the probability that children incur diseases. We estimate equation 3.1 with data of the second, third and fourth wave of the MCS separately. This setup allows to study the association between parental income and disease prevalence for three distinct age groups. If parental income protects child health by lowering the exposure to health specific risks, we expect

to find a relation between parental income and disease prevalence in older age groups as children need to accumulate a certain dose of health risk factors until a disease emerges.

Table 3.2 presents our results. Every coefficient corresponds to one regression. The left hand part of the table shows the results for the second wave of MCS, the right hand part those for the third and fourth wave. The first row displays the estimated coefficients for the effect of parental income on an indicator that equals 1 for children having any longstanding condition. The lower rows present the results for specific conditions. At the mean of income and for the second wave of the MCS, there is no significant evidence for the hypothesis that an increase in income prevents longstanding health conditions in general. The coefficient $\alpha_1$ is negative but not significant at conventional levels. Looking at specific conditions, our results display that the association between income and disease prevalence is negative for seven out of ten indicators. The largest "protective" effect is obtained for problems related to the respiratory system and skin problems. However, none of the estimated coefficients is significant at the 5% level.

The results for the third wave of MCS differ slightly. The risk of experiencing any longstanding condition is negatively (and significantly) associated with income. A negative relationship holds for 8 out of 10 conditions indicators. The largest coefficients are obtained for respiratory diseases as well as for problems related to the digestive system and otherwise classified diseases. Coefficients are significant at the 5% level for diseases of the respiratory system and "other diseases".

The results for the fourth wave of MCS are similar to those from the third wave. They highlight the close association between higher parental income and a lower probability of experiencing a longstanding disease in particular of the respiratory system. None of the other relationships is significant.

Overall, these results provide some supportive evidence for the hypothesis that income prevents adverse health conditions. Apparently, the relation between income and the prevalence of health conditions varies with age and with the single condition we look at.

Table 3.2
The Prevalence of Health Conditions and Parental Income

| **Sample:** | **MCS2** | | **MCS3** | | **MCS4** | |
|---|---|---|---|---|---|---|
| Condition | $\alpha_1$ | | $\alpha_1$ | | $\alpha_1$ | |
| any longstanding | -0.0113 | (-1.63) | -0.0449*** | (-6.05) | -0.0412*** | (-5.25) |
| mental disorders | 0.000950 | (0.93) | -0.00133 | (-0.76) | -0.00339 | (-1.48) |
| diseases of nervous system | -0.000188 | (-0.17) | 0.000446 | (0.34) | 0.00264 | (1.64) |
| eye diseases | -0.00188 | (-1.51) | 0.000956 | (0.51) | -0.000700 | (-0.37) |
| ear diseases | 0.000394 | (0.31) | -0.00380 | (-1.54) | -0.00368 | (-1.46) |
| diseases of respiratory system | -0.00572 | (-1.51) | -0.0289*** | (-5.62) | -0.0280*** | (-5.23) |
| diseases of digestive system | -0.000764 | (-0.37) | -0.00243 | (-1.35) | -0.000218 | (-0.11) |
| diseases of the skin | -0.00500 | (-1.42) | -0.00426 | (-1.18) | -0.00407 | (-1.12) |
| diseases of genitourinary system | -0.000548 | (-0.36) | -0.000466 | (-0.31) | -0.00188 | (-1.30) |
| congenital malformations | 0.00222 | (0.84) | -0.00116 | (-0.65) | 0.00169 | (0.92) |
| other | -0.00266 | (-0.72) | -0.00999** | (-2.78) | -0.00510 | (-1.27) |
| N | 12551 | | 13081 | | 11630 | |

$t$ statistics in parentheses; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. This table presents regression results for equation 3.1. The single estimations are run separately for the single waves of the MCS and each coefficient corresponds to one regression. Control Variables include a child's age, mother's age at birth, log of household size, mother's education, log of birth weight as well as dummy variables indicating the child's sex, lone mother, main respondent not natural mother, migrant household and twin births.

There is only weak evidence supporting the hypothesis that children from poor households accumulate disadvantages, which evolve to manifest health conditions over time. The evidence supporting this hypothesis is strongest for diseases of the respiratory tract.

### 3.4.2 Parental Income and the Consequences of Health Conditions

Tables 3.3 and 3.4 present the results for the estimation of equations 3.2 and 3.3. We start with a discussion of the results on the left hand part of table 3.3. The first column of table 3.3 indicates that all health conditions have considerable positive (i.e. harmful) and significant immediate effects on the probability of having poor health. The largest coefficients are obtained for diseases of the nervous system as well as for diseases of the respiratory and genitourinary systems. The estimates for $\beta_2$ have the expected negative sign and are statistically significant, which indicates that the probability of poor child health status decreases with household income, even conditional on longstanding conditions. The coefficient for the interaction term of health conditions and income, $\beta_3$, is negative and significant for the longstanding conditions indicator that equals one if a child has any health condition. It is negative and significant at the 5% level for skin problems and diseases of the nervous system. The coefficients on all but one other specific health conditions are negative but insignificant. This finding supports the hypothesis that the way children respond to several diseases, depends on parents socioeconomic status.

The right hand part of the table shows the results for the one period lagged health conditions. Most conditions still have a considerable effect on subjective health status 2.5 years after a health problem was stated. This effect is particularly large, when considering diseases of the nervous system and mental health problems. The coefficient for the interaction effect is negative and significant for the longstanding conditions indicator. However, the magnitude of the effect is substantially smaller compared to the immediate effect. $\beta_3$ is negative for all but two conditions but not significant at the 5% level for any specific condition, except for diseases of the nervous system.

Table 3.3
The Severity of Health Conditions and Parental Income in MCS3

| | Contemporary Health Conditions (MCS3) | | | | | | Lagged Health Conditions (MCS2 to MCS3) | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Condition** | $\beta_1$ | | $\beta_2$ | | $\beta_3$ | | $\beta_1$ | | $\beta_2$ | | $\beta_3$ | |
| any condition | 0.316*** | (31.61) | -0.034*** | (-5.24) | -0.069*** | (-4.49) | 0.174*** | (14.62) | -0.054*** | (-6.70) | -0.039* | (-2.21) |
| mental | 0.244*** | (5.23) | -0.062*** | (-8.74) | 0.059 | (0.78) | 0.383*** | (3.99) | -0.064*** | (-7.99) | -0.018 | (-0.11) |
| nervous | 0.422*** | (7.25) | -0.061*** | (-8.66) | -0.209* | (-2.24) | 0.290*** | (3.88) | -0.063*** | (-7.81) | -0.328* | (-2.16) |
| eye | 0.092* | (2.39) | -0.062*** | (-8.66) | -0.048 | (-0.81) | 0.036 | (0.61) | -0.064*** | (-7.92) | 0.022 | (0.21) |
| ear | 0.236*** | (7.48) | -0.060*** | (-8.47) | -0.041 | (-0.90) | 0.157* | (2.39) | -0.063*** | (-7.85) | -0.059 | (-0.71) |
| respiratory | 0.354*** | (22.72) | -0.051*** | (-7.45) | -0.012 | (-0.51) | 0.232*** | (10.12) | -0.059*** | (-7.43) | -0.051 | (-1.41) |
| digestive | 0.316*** | (7.02) | -0.060*** | (-8.51) | -0.105 | (-1.44) | 0.156*** | (3.84) | -0.063*** | (-7.80) | -0.056 | (-0.95) |
| skin | 0.195*** | (9.20) | -0.058*** | (-8.17) | -0.079* | (-2.47) | 0.093*** | (4.11) | -0.061*** | (-7.62) | -0.038 | (-1.14) |
| genitourinary | 0.375*** | (7.01) | -0.062*** | (-8.68) | -0.030 | (-0.34) | 0.148* | (2.56) | -0.063*** | (-7.86) | -0.064 | (-0.76) |
| congenital | 0.260*** | (5.96) | -0.062*** | (-8.66) | -0.024 | (-0.35) | 0.161*** | (5.10) | -0.063*** | (-7.80) | -0.029 | (-0.66) |
| other | 0.331*** | (15.30) | -0.057*** | (-8.14) | -0.055 | (-1.42) | 0.168*** | (7.15) | -0.063*** | (-7.84) | 0.015 | (0.39) |
| N | 13079 | | | | | | 10385 | | | | | |

$t$ statistics in parentheses; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. This table presents results for the regression models represented by equations 3.2 (left hand part of table) and 3.3 (right hand part). Control variables include those indicated in the notes to table 3.2.

Table 3.4
The Severity of Health Conditions and Parental Income in MCS4

| | Lagged Health Conditions (MCS3 to MCS4) | | | | | | Lagged Health Conditions (MCS2 to MCS4) | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Condition** | $\beta_1$ | | $\beta_2$ | | $\beta_3$ | | $\beta_1$ | | $\beta_2$ | | $\beta_3$ | |
| any condition | 0.188*** | (18.30) | -0.039*** | (-5.60) | -0.084*** | (-5.08) | 0.122*** | (10.75) | -0.041*** | (-5.40) | -0.079*** | (-4.64) |
| mental | 0.188*** | (3.84) | -0.063*** | (-8.72) | -0.124 | (-1.50) | 0.184 | (1.88) | -0.056*** | (-7.34) | -0.076 | (-0.57) |
| nervous | 0.231*** | (3.47) | -0.064*** | (-8.90) | -0.022 | (-0.20) | 0.192* | (2.40) | -0.055*** | (-7.31) | -0.158 | (-1.08) |
| eye | 0.043 | (1.19) | -0.063*** | (-8.79) | -0.109 | (-1.59) | 0.125* | (2.00) | -0.055*** | (-7.26) | -0.155 | (-1.41) |
| ear | 0.108*** | (3.57) | -0.063*** | (-8.77) | -0.044 | (-0.89) | 0.074 | (1.25) | -0.056*** | (-7.33) | -0.098 | (-1.06) |
| respiratory | 0.219*** | (13.35) | -0.054*** | (-7.65) | -0.051 | (-1.88) | 0.139*** | (6.57) | -0.051*** | (-6.69) | -0.080* | (-2.40) |
| digestive | 0.240*** | (4.99) | -0.062*** | (-8.62) | -0.233** | (-2.67) | 0.127*** | (3.37) | -0.054*** | (-7.06) | -0.213*** | (-3.93) |
| skin | 0.138*** | (6.19) | -0.060*** | (-8.41) | -0.069* | (-2.06) | 0.063** | (2.91) | -0.056*** | (-7.29) | -0.005 | (-0.17) |
| genitourinary | 0.225*** | (4.03) | -0.063*** | (-8.76) | -0.098 | (-1.03) | 0.069 | (1.30) | -0.056*** | (-7.41) | 0.016 | (0.19) |
| congenital | 0.112** | (2.68) | -0.064*** | (-8.98) | 0.037 | (0.50) | 0.112*** | (3.62) | -0.055*** | (-7.29) | -0.035 | (-0.78) |
| other | 0.209*** | (9.10) | -0.058*** | (-8.17) | -0.119** | (-2.95) | 0.113*** | (5.11) | -0.054*** | (-7.13) | -0.052 | (-1.30) |
| N | 10283 | | | | | | 9331 | | | | | |

$t$ statistics in parentheses; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. This table presents results for the regression models represented by equations 3.3. Control variables include those indicated in the notes to table 3.2.

The left hand part of table 3.4 presents our results for the effect of diseases reported in the third wave of MCS on the probability of poor health in MCS4. Although the relative importance of some diseases slightly changes, the estimates of $\beta_1$ and $\beta_2$ are similar in magnitude to those shown in table 3.3. The coefficient for the interaction effect $\beta_3$ suggests that the severity of diseases is much larger for the transition from the third to the fourth wave of MCS compared to the effect of diseases from the second wave to the third one. $\beta_3$ is negative for all but one of the conditions and significant for digestive and skin diseases as well as for otherwise classified conditions. The effect is in particular large for diseases of the digestive system. An increase in parental earnings by one log point (which corresponds to more than doubling income), among all children with these specific problems, reduces the probability of having poor health by 23%.

The right hand side of the table presents the results for the effect of 5 years lagged conditions. The estimates of $\beta_3$ underline that there is evidence for differential severity of diseases by parental income in the United Kingdom. Still, all but one of the coefficients of the interaction effects are negative. Parental income affects the probability of good health even 5 years after conditions of the respiratory or digestive system were reported. This result provides strong evidence in favor of the hypothesis that parental income plays an important role in coping with diseases. However, the size of the coefficients differs substantially by the single conditions, which emphasizes the advantage of inspecting distinct health conditions, rather than an indicator for any longstanding health condition.[8]

[8] Case, Lee, and Paxson (2008) pointed out that using single conditions as explanatory variables in a regression model represented by equation 3.2 neglects the problem of multi morbidity. If parental income is correlated with the total number of conditions children are facing, the association between single health conditions and subjective health is confounded by the number of conditions a particular child is suffering from. In this case, the regression model represented by equation 3.2 would suffer from an omitted variable bias, where the omitted variables are all other conditions child $i$ is suffering from. Although income and the number of diseases in excess of one are not strongly related in our data, we also estimate models with additional controls for the number of conditions and a model that controls for all conditions at once. Both modifications change our results only slightly. We conclude that the total number of conditions does not confound our results.

### 3.4.3 The Recovery-Effect of Income

The last section provided some evidence in favor of the hypothesis that parental income can cushion the consequences of certain diseases. This section tests whether the recovery-effect can serve as one explanation for this result. The recovery-effect hypothesis stipulates that parental income is positively associated with the chances for a fast recovery.

Table 3.5 presents our estimation results for equation 3.4. The left hand side of the table shows the effect of diseases reported in MCS2 on the probability of reporting these conditions in MCS3. The right hand part displays the respective effects of conditions in MCS2 on the probability of still suffering from these conditions in MCS4. We start with a discussion of the results on the left hand side. The estimates of $\gamma_1$ indicate that all past conditions have a large effect on present health conditions. The most persistent diseases are mental and nervous diseases as well as diseases of the respiratory system. Eye problems as well as diseases of the digestive system are those diseases with the lowest level of persistence. The estimates for $\gamma_2$ confirm the results of section 3.4.1, where we found a close association between parental income and in particular diseases of the respiratory system. The last column presents our estimates for the coefficient on the interaction $\gamma_3$ between income and lagged health conditions. Our results back the hypothesis that persistence of longstanding diseases is related to parental income. This finding applies to the indicator of longstanding conditions as well as to diseases related to the digestive system, but it does not apply to any other specific condition in a statistically significant way. The coefficient is particularly large for diseases of the digestive system: An increase by one log point in parental income increases a child's chances to recover from these health problems by 21%.

Turning to the results on the right part of the table, we observe that all conditions are significantly predicted by reporting of these conditions 5 years ago. The estimates for $\gamma_3$ are mostly negative but significant only for genitourinary diseases. The effect of digestive diseases disappears.

Table 3.5
Is Persistence of Diseases Related to Parental Income?

| Period | MCS2 to MCS3 | | | | | | MCS2 to MCS4 | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Condition** | $\gamma_1$ | | $\gamma_2$ | | $\gamma_3$ | | $\gamma_1$ | | $\gamma_2$ | | $\gamma_3$ | |
| any condition | 0.362*** | (28.13) | -0.032*** | (-3.88) | -0.042* | (-2.16) | 0.310*** | (22.86) | -0.035*** | (-4.02) | -0.028 | (-1.31) |
| mental | 0.419*** | (4.35) | -0.001 | (-0.71) | 0.010 | (0.05) | 0.303** | (3.06) | -0.003 | (-1.22) | -0.005 | (-0.03) |
| nervous | 0.715*** | (9.44) | 0.001 | (0.75) | -0.030 | (-0.19) | 0.479*** | (5.16) | 0.003 | (1.66) | -0.105 | (-0.56) |
| eye | 0.220*** | (3.67) | 0.0005 | (0.25) | -0.155 | (-1.31) | 0.216*** | (3.43) | 0.0003 | (0.13) | -0.167 | (-1.45) |
| ear | 0.325*** | (4.85) | -0.002 | (-0.76) | -0.103 | (-1.03) | 0.279*** | (4.01) | -0.006* | (-2.16) | -0.011 | (-0.10) |
| respiratory | 0.421*** | (17.76) | -0.023*** | (-4.19) | -0.010 | (-0.28) | 0.342*** | (14.20) | -0.023*** | (-4.09) | -0.049 | (-1.28) |
| digestive | 0.229*** | (6.29) | -0.0001 | (-0.05) | -0.210*** | (-4.14) | 0.187*** | (4.98) | 0.0003 | (0.16) | -0.054 | (-1.05) |
| skin | 0.254*** | (10.66) | -0.002 | (-0.47) | -0.034 | (-0.98) | 0.227*** | (9.38) | -0.004 | (-1.04) | -0.027 | (-0.75) |
| genitourinary | 0.234*** | (4.44) | -0.00003 | (-0.02) | -0.134 | (-1.67) | 0.172*** | (3.54) | -0.002 | (-1.40) | -0.166* | (-2.15) |
| congenital | 0.238*** | (8.12) | -0.002 | (-1.38) | -0.029 | (-0.69) | 0.219*** | (7.30) | 0.001 | (0.74) | 0.081 | (1.79) |
| other | 0.256*** | (11.43) | -0.009* | (-2.26) | 0.039 | (0.97) | 0.233*** | (10.04) | -0.004 | (-1.00) | 0.006 | (0.14) |
| N | 10387 | | | | | | 9331 | | | | | |

$t$ statistics in parentheses; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. This table presents estimation results for equation 3.4. The respective health condition is given in the left column. The left hand part of the table shows the results for the one wave lagged conditions (n=2.5 years), the right hand part shows the results for the 2 waves lagged conditions (n=5 years). Control variables include those indicated in the notes to table 3.2.

Altogether, our results provide only weak support for the hypothesis that children from low-income households are more severely affected by diseases because they suffer from these conditions for a longer time. Evidently, this pattern is very different across diseases. Persistence of diseases might be best suited to explain differential responses to diseases of the digestive system by parental income, but cannot explain these effects for other diseases.

## 3.5 Robustness

### 3.5.1 Maternal Labor Supply and Reversed Causality

It is not obvious, to what extent the association between parental income and child health reflects a causal effect running from parental income to child health. We argued that parental income prevents the incidence and cushions the consequences of adverse health conditions. However, if children become sick, it is possible that parents react by reducing labor supply (and thus income) in order to personally care for their child. In this case, our estimates of the coefficients on parental income would be upward biased estimates for the causal effect of income on child health.

However, several properties of our research design help reducing the labor supply effect of health shocks among children. We average income over four waves of interviews. Even if income temporarily reacts to changes in a child's health status, our measure of income is still relatively close to permanent income. Previous studies found evidence for no effects (Case, Lubotsky, and Paxson (2002)) (for the US) or negative (Frijters et al. (2009)) labor supply effects of a child's development in Australia. Our approach to test the responsiveness of parental labor supply to changes in a child's health status is similar to that proposed by Case, Lubotsky, and Paxson (2002). We focus on the period after a child's birth, and in particular on the consequences of two of the first adverse health conditions, a child can experience: underweight at birth and congenital anomalies.

We estimate the following model:

$$LS_{i,t} = \beta_0 + \beta_1 PH_{i,t-n} + \beta_2 LS_{i,t-n-1} + X_{i,t}\gamma + e_{i,t} \tag{3.5}$$

$LS_{i,t}$ represents a binary indicator of labor supply of child $i$'s mother at time $t$. It takes the value 1 if she works at least part-time. $PH_{i,t}$ represents an indicator for our measure of poor child health. It takes the value 1 if child $i$ is born with low birth weight (less than 2.500 kilogram). In the second set of regressions, we use congenital anomalies as a measure of poor child health. We control for maternal labor supply during pregnancy as well as for the covariates included in previous analyses (see notes to table 3.2). Clearly, this model falls short in reflecting the complexity of labor supply decisions in families. We are convinced that it nonetheless provides important implications for the interpretation of the previous results.

The results are shown in table 3.6. The first three columns present the effect of low birth weight on mothers' labor supply at various ages of the child. Columns 4 to 6 show the results for congenital health problems as regressor of interest. Having low birth weight is negatively associated with maternal labor supply. This result is obtained for mother's employment status at all considered ages of the child. The association is statistically significant at the 10% level for employment status at the age of 2-3 years. Having worked during pregnancy is strongly associated with employment status after

Table 3.6
Child Health and Maternal Labor Supply Reactions

| | (1) | (2) | (3) | (4) | (5) | (6) |
|---|---|---|---|---|---|---|
| | *Indicator Mother works* | | | | | |
| Child Age: | 9 months | 2-3 years | 4-6 years | 9 months | 2-3 years | 4-6 years |
| Low birth weight | -0.016 | -0.026 | -0.019 | | | |
| | (-1.47) | (-1.86) | (-1.35) | | | |
| Congenital diseases | | | | -0.005 | 0.015 | 0.033 |
| | | | | (-0.29) | (0.75) | (1.54) |
| Mother worked during pregnancy | 0.601*** | 0.457*** | 0.380*** | 0.601*** | 0.458*** | 0.381*** |
| | (93.03) | (55.99) | (45.16) | (93.07) | (56.03) | (45.18) |
| $N$ | 18471 | 14852 | 14630 | 18471 | 14852 | 14630 |

$t$ statistics in parentheses; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. This table presents regression estimates on the impact of poor child health on maternal labor supply at various ages of the child. The dependent variable equals 1, if a child's mother works at least part-time. Indicators of poor child health are low birth weight and congenital health problems. Control variables include those indicated in the notes to table 3.2.

childbirth. The results displayed in columns 4 to 6 do not indicate that having a child with congenital health problems decreases the probability of working. This finding applies to all considered ages of the child.
The results presented in table 3.6 indicate that the potential bias due to reversed causation is not very dramatic as the effects on labor supply are quite small and insignificant at conventional levels.[9] If a sick child negatively affected parents' income, then the true causal effect of income would likely be smaller than what the estimates, shown in previous sections, suggest. We conclude that our estimates for the effect of parental income are an upper bound for the true effect of parental income on child health.

### 3.5.2 Parental Health as an Omitted Variable

A second reason why parental income could be an endogenous variable in equations 3.1-3.4 is that omitted common causes drive the variation on both sides of the equation. Factors that are likely related to both parents' income and a child's health status are for instance parental health status or unobservable parental ability. Although some of these features are likely to be reflected in control variables such as mother's education, birth weight, and mother's age at birth, there can still be unobservable heterogeneity that leads to biased estimates. Birth weight, for example, has been shown to correlate with mother's risky behavior during pregnancy, such as smoking and drinking, and therefore reflects a specific feature of parental quality. Parents' health status is a candidate variable for a common cause, as parents' health can have an impact on both their earnings as well as on their children's health status. Two recent articles argue that when controlling for parental health status, the association between parental income and child health disappears (Khanam, Nghiem, and Connelly (2009), Propper, Rigg, and Burgess (2007)). This finding suggests that there is no causal effect of income on child health but that the observed correlation is spurious. The Millennium Cohort Study comprises information on mother's subjective health status and height. Information on father's health status, however, is incomplete. In order to in-

[9] We also inspect whether the effect of poor health on maternal labor supply differs between mothers that worked before birth and mothers that did not work before birth. There are no substantial differences.

vestigate wether mother's health status is an omitted variable that drives our results, we redo the estimation of all models represented by equations 3.1-3.4 with mothers' subjective health status and mothers' height as additional control variables. However, in our data, these additional control variables reduce the effect of income on a child's health status only slightly. None of our main results is changed.[10] We conclude that the results presented in the last sections do not just reflect the association between parent's health and their children's health.

## 3.6 Summary and Conclusions

This article investigates why health inequality increases as children age. Our results confirmed that health inequality by socioeconomic status has its origins in early childhood. We find some support for both hypotheses: the prevalence of diseases as well as the way children cope with diseases are related to parental income. Children from low-income households have a higher probability of experiencing a longstanding health problem. However, this association between parental income and an indicator for longstanding conditions is mainly driven by diseases of the respiratory tract. We obtain this result even when controlling for health status at birth (as proxied by birth weight). One possible explanation for this finding is that parental income is related to the level of air pollution children are exposed to, which eventually increases the probability of incurring diseases of the respiratory system. The finding that the prevalence of certain diseases is correlated with parental income is in line with prior evidence from UK (Currie, Shields, and Price (2007)), US (Condliffe and Link (2008)) and Canada (Currie and Stabile (2003)).

Moreover, depending on their parents' income, diseases put children on different tracks in the development of their health status over time. The mapping of health conditions into subjective health status differs by parental income. Among all children with a particular disease, those coming from low-income households are more likely to have poor health status than their peers from

[10]The results are available from the author upon request.

high-income families. This finding applies to an indicator for longstanding conditions as well as to some specific conditions. Even 5 years after certain diseases arrive, children from low income households are worse off than their peers from high income families in terms of subjective health status.

The magnitude of the estimated coefficients of these interaction effects is surprisingly large compared to the effects found for the US by Condliffe and Link (2008), and much larger than the (insignificant) effects in Canada, estimated by Currie and Stabile (2003). The large effects that we find can be due to our sampling of very small children, which contrasts to the sample used in Condliffe and Link (2008) that includes children at all ages. If our findings were only due to the sampling of small children, this would imply that either the differences in the consequences of illnesses by SES are particularly large in small children or that these differences are quite large in the UK in general.

We suggest and test one explanation to learn more about mechanism underlying this result. We attribute part of the association between adverse effects of diseases and parental income to the fact that low-income children have a lower probability to recover quickly from diseases. Among children that are afflicted by diseases of the digestive or genitourinary systems, those from low SES families are more likely to retain this condition over a period of 2.5 years. Put differently, it appears that income is positively associated with a child's probability of recovering from these diseases. This mechanism explains part of the differences in subjective health among those children that had an illness in the previous period.

There are many other possible aspects of the differences in the consequences of health conditions by parent's SES, which should be addressed in future research. For example, ill children from poor families might be more impaired in their physical and cognitive development, leading to inequalities in other domains.

The main implication of our results is that even in the presence of a universal health insurance system, parental income affects how children cope with ill health. Differences in the quantity and quality of provided or demanded health care provide one explanation for this finding. Health care programs that are tailored to the specific needs of disadvantaged groups, can therefore

be a mean to reduce inequalities.

Our robustness checks revealed that reversed causality in the relationship between household income and child health is a slight concern. We are therefore cautious with interpreting the association between income and child health as representing a pure causal protective effect of income on health. If parents cut their labor supply in response to their child falling ill, the correlation of parental income and child health reflects causality running in both directions. Although our results do not point in this direction, we cannot reject that the true effect of income on child health is possibly smaller, such that the estimated coefficients represent an upper bound for the causal protective effect of income on child health.
Lastly, in contrast to recent studies by Khanam, Nghiem, and Connelly (2009) and Propper, Rigg, and Burgess (2007), our results do not support the hypothesis that the association between income and child health is driven by (the common cause) a parent's health status. In fact, controlling for mother's health status reduces our coefficients only slightly and does not change any substantial result.

**Acknowledgements:**
This article uses data from the University of London, Institute of Education, Centre for Longitudinal Studies:
Millennium Cohort Study: First Survey, 2001-2003 [computer file]. 8th Edition. Colchester, Essex: UK Data Archive [distributor], March 2009. SN: 4683
Millennium Cohort Study: Second Survey, 2003-2005 [computer file]. 5th Edition. Colchester, Essex: UK Data Archive [distributor], March 2009. SN: 5350
Millennium Cohort Study: Third Survey, 2006 [computer file]. 3rd Edition. Colchester, Essex: UK Data Archive [distributor], March 2009. SN: 5795
Millennium Cohort Study: Fourth Survey, 2008 [computer file]. Colchester, Essex: UK Data Archive [distributor], April 2010. SN: 6411.

# 3.7 Appendix

Table 3.7
The Frequency of Health Conditions in the MCS

| Wave | MCS2 | MCS3 | MCS4 |
|---|---|---|---|
| mental disorders | 0.00378 | 0.00892 | 0.01379 |
| diseases of nervous system | 0.00385 | 0.00544 | 0.00686 |
| eye diseases | 0.00385 | 0.00938 | 0.00844 |
| ear diseases | 0.00481 | 0.01811 | 0.01530 |
| diseases of respiratory system | 0.04022 | 0.08180 | 0.07600 |
| diseases of digestive system | 0.01148 | 0.00886 | 0.00931 |
| diseases of the skin | 0.03374 | 0.03824 | 0.03240 |
| diseases of genitourinary system | 0.00674 | 0.00623 | 0.00527 |
| congenital malformations | 0.01905 | 0.00905 | 0.00779 |
| other | 0.03907 | 0.03982 | 0.04063 |
| N | 15588 | 15244 | 13855 |

# Chapter 4

# The Effect of Children on Depression in Old Age

## 4.1 Introduction

Depression is one of the most common health conditions among the elderly. Castro-Costa et al. (2007) estimate that prevalence rates in 10 European countries range between 18.1% (in Denmark) and 36.8% (in Spain) among individuals aged 50 and above. In addition to personal psychological costs, mental health problems are also associated with considerable costs for health care and social security systems. According to the OECD, depression is the primary determinant of disability in developed countries (OECD (2008)). Recent evidence also suggests that depression is an important cause of early retirement in Europe (Alavinia and Burdorf (2008)). The overall costs of depression are estimated to correspond to approximately 1% of GDP in European countries (Sobocki et al. (2006)). Understanding promotive and protective factors for the incidence of depression is therefore a major concern for public health research. This article asks whether children protect or jeopardize parents mental health status. In contrast to the previous and mostly descriptive literature, we put the focus of our investigation on the identification of the causal effect of additional children.

There are arguments for and against a positive effect of children on mental health. Sociologists stress the importance of children within the social net-

work of aging parents (Bures, Koropeckyj-Cox, and Loree (2009)). Children can provide social support and care. A higher number of children might therefore prevent loneliness in old age. Children also provide parents with a sense of gratitude and feelings of meaning in life, which might positively affect mental health (Evenson and Simon (2005)). From an economic point of view, it is not obvious whether a higher number of children is associated with a higher amount of care received by their parents. When care for parents is considered a public good that is provided by children, strategic interaction among children can lead to an inefficiently low quantity of care provided.[1] On the other hand, children can also be a source of strain, economic costs and physical pain. In particular when children are young, the role of parents is physically and mentally demanding. The larger share of responsibility in these years is mostly borne by mothers. Hence, mothers can be particularly vulnerable to mental diseases (Umberson and Gove (1989)). Raising children is associated with both direct costs (e.g. for nutrition and education) and opportunity costs. Opportunity costs arise since the birth of a child can put parents off track in their employment biographies, possibly reducing earnings and the chance of obtaining prestigious positions and increasing the risk of suffering financial shortages (Ross, Mirowsky, and Goldsteen (1990)). The birth of children increases the need for economic resources but can at the same time decrease parents' earnings potential. A recent study by Adda, Dustmann, and Stevens (2010) estimates the loss in wages due to a childbirth to equal 17% for women. The authors attribute this wage loss mainly to the interruption of careers and the associated loss of human capital as well as to the sorting of mothers into child-friendly occupations. Childbearing not only reduces wages but likely also increases the risk of experiencing periods of economic hardship, which in turn is negatively associated with mental health (Ross and Huber (1985), Mirowsky and Ross (2001)).

A number of prior studies investigate the relationship between the number of children and mental health at higher ages. Some of these studies have pointed to differences in prevalence rates of depression by the number of children. However, the interpretation of any association between the number of

[1] For articles modeling strategic interaction among children in the provision of care for their parents, see e.g. Bernheim, Shleifer, and Summers (1985) or Konrad et al. (2002).

children and mental health is made difficult by the complex mechanisms underlying the fertility-health nexus. The empirical identification of the causal effect of additional children on health is complicated by the fact that fertility decisions might correlate with mental health for two reasons.[2] On the one hand, finding a partner and realizing the desired level of fertility might be more difficult for individuals with poor mental health. Large evidence supports the hypothesis that individuals with good mental health status have a substantially higher probability of maintaining stable relationships.[3] On the other hand, fertility preferences of individuals with poor mental health can differ from those of mentally healthy persons. If individuals self-select into their optimal level of fertility, observed fertility patterns might be the result from a mental condition rather than the other way around. Any correlation between the number of children and measures of mental health is therefore the sum of a causal effect of the number of children and a selection effect, both of which have unknown signs.

This article provides the first estimates for the causal effect of additional children on their parents' mental health status. Specifically, we ask whether adding one child to the parity of children exerts a causal effect on the probability of suffering from depression. We focus on the role of biological children for their parents' wellbeing. Our identification strategy builds on three instrumental variables (IV) for the number of children: variables indicating a multiple birth at the first and second birth and a dummy variable that indicates whether the first two children have the same sex. The sex composition of the first children has been shown to be related to the probability of having further children (e.g. Andersson et al. (2006), Hank and Kohler (2000)). We exploit that multiple births, as well as the sex composition of children, result from natural experiments and have an effect on the total number of children, thus allowing to draw causal conclusions. We argue that these instruments allow calculating three different local average treatment effects and therefore provide insights about several groups of compliers and at different margins.

[2]We define fertility as the number of biological children individuals have.

[3]The literature review by Coombs (1991) points out that married individuals have a substantially lower prevalence of psychiatric disorders compared to single, divorced and widowed individuals.

These instrumental variables allow to study both the effect of unexpected increases in the total number of children (as induced by multiple births) and the effect of expected and desired increases in the number of children for an exogenous reason (same sex sibship). Both instruments have been used to investigate the effect of children on several economic outcomes (e.g. Black, Devereux, and Salvanes (2005)). However, they have not been applied to study the long-term consequences of fertility on mental health.
First, we investigate the total effect of additional children on the mental health status of their parents. Second, we explore possible pathways by looking at the probability of critical events that occurred after the last childbirth and that are candidates for mediating variables.[4] We use newly available data from the Survey of Health, Aging and Retirement in Europe (SHARE and SHARELIFE) that provides fertility histories of more than 20.000 elder Europeans, along with extensive information on health status and socioeconomic background.

We find evidence for a negative causal effect of additional children on mental health of elder women. Women who have a third child because of a multiple birth are found to have a higher risk of suffering from a depression. Hence, the relevant margin is the transition from the second child to the third, and the effect is only significant if the additional child was induced by a multiple birth. We do not find evidence for such an effect in the male sample. We suggest that situations of financial hardship and poor overall health status mediate between the number of children and mental health in old age.

This article is organized as follows. The next section briefly reviews the related literature on the relationship between parenthood and mental health as well as related articles that use multiple births and the sex composition of children as instruments for the number of children. Section 4.3 describes our identification strategy in greater detail. In section 4.4, our data set is introduced and section 4.5 presents our main results. The last section summarizes and draws conclusions from our findings.

[4]We do not investigate the effects of the timing of child bearing, as this is partly result of an individual choice and therefore an endogenous variable.

## 4.2 Related Literature

This paper draws on two strands of the literature. Evidence on the relationship between fertility and health outcomes has mainly been provided by public health researchers, psychologists and sociologists. There is a substantial number of articles investigating the manifold long-term consequences of childbearing on health.[5] We concentrate on previous research investigating the fertility – mental health nexus in the following. Several descriptive articles investigate whether parenthood is linked to mental health at higher ages. However, the results provided by the previous literature are surprisingly ambiguous and do not draw a consistent picture yet. The ambiguous evidence may in part be due to differences in definitions of study group and treatments as well as due to differences in the selection of control variables. For example, the relationship between parenthood and mental health appears to depend on whether children are still living at home or have already left the household of their parents (Evenson and Simon (2005)). It also depends on whether the association between biological or step-children and mental health is considered (Bures, Koropeckyj-Cox, and Loree (2009)).
Evenson and Simon (2005) provide evidence backing the hypothesis that parents in general have a higher risk of suffering from depression than childless couples. This finding is driven in particular by families with minor children. This is also supported by Gove and Geerken (1977) who document that children living in the household of their parents generally increase the risk of poor mental health. Evidence suggesting that parenthood is negatively associated with psychological distress has also been provided by Burton (1998) for US data. No association between parenthood and parents' mental well being is documented by Umberson and Gove (1989) and Mirowsky and Ross (2002) for the US and Hank (2010) for individuals from Germany. In contrast

[5] We do neither discuss short-term effects of childbearing on mental health, nor long-term effects on physical health status. For a discussion of the first, see e.g. Weissman and Olfson (1995), for a discussion of the latter, we refer to Grundy and Tomassini (2005) and Hurt, Ronsmans, and Thomas (2006). There is also a literature on the consequences of the timing of fertility, see e.g. Spence (2008) or Mirowsky and Ross (2002) and on the particular consequences of early motherhood (Henretta et al. (2008)). Bures, Koropeckyj-Cox, and Loree (2009) provide evidence on the relationship between parenthood and mental health by different types of parenthood (biological vs. social parenthood).

to this, Koropeckyj-Cox (1998) finds weak evidence in favor of the hypothesis that childless elderly people have a higher probability of suffering from loneliness and depression.
There is no consistent evidence for the relationship between the number of children and mental health. Spence (2008) documents that parents of five or more children do not have worse mental health than parents of one to four children in the US. This evidence is not supported by studies from old age Europe by Buber and Engelhardt (2008), who find evidence for a non-linear association between children and mental health for men, using the SHARE data. Fathers of one to three children are found to be significantly healthier compared to fathers of four or more children and childless men in terms of mental health. The authors find no evidence for such an association for women. Hank (2010) reports no differences in mental health by the parity of children for middle-aged individuals from Germany.

The second strand of the literature, this article is related to, explores the causal consequences of childbearing using instrumental variables. Rosenzweig and Wolpin (1980) were the first to suggest using twin births as an instrumental variable for the number of children. More recent articles also rely on the sex composition of the first born children, exploiting parents' taste for a balanced sex composition as an instrument for additional children (Black, Devereux, and Salvanes (2005), Black, Devereux, and Salvanes (2010), Angrist, Lavy, and Schlosser (2010)). This methodology has mainly been used to empirically test Becker's quantity-quality hypothesis, which states that resource constrained parents can either invest in the quality or in the size of their offspring (Becker (1960)). Other applications include the analysis of the effect of children on parents' labor supply and on mothers' wages (Butcher and Case (1994), Angrist and Evans (1998)) as well as on marital stability and the probability of depending on public welfare programs (Angrist (2004)). To the best of our knowledge, these instruments have not been used before to investigate the causal impact of children on health in old age.

We are aware of only one article investigating the causal effect of fertility on maternal health using an IV setup. Cáceres-Delpiano and Simonsen (2010)

find large detrimental effects of additional children on their mothers' health during their fertile years, using multiple births as an IV for the number of children and drawing on US census data. In particular, additional children appear to increase mothers' risk of high blood pressure as well as for various risky behaviors. This paper is different from the work by Cáceres-Delpiano and Simonsen (2010) in three respects: We investigate mental health as opposed to physical health and consider a very detailed measurement of mental health status. Second, we investigate the long-term effects of childbearing as most individuals in our sample have adult children. Third, we not only rely on multiple births as an IV for fertility but compare the results thereof to estimates from an instrument that identifies a different local average treatment effect.

## 4.3 Identification Strategy

The interpretation of any association between the number of children and their parents' mental health status is rendered difficult by the complex causal mechanisms driving both variables. Any mean difference in mental health scores by the number of children reflects both: a treatment effect running from the number of children to a certain mental state and a selection effect that expresses that people with certain psychological characteristics select into specific patterns of fertility. To disentangle treatment and selection effects, we ideally need a mechanism that randomly allocates children to couples. In such an ideal experimental setting, we could interpret any mean difference in mental health by the number of children as a direct consequence of the number of children. In social science, and in particular for variables like the number of children, such administered experiments are not available. We argue that two events that randomly occur and that affect the number of children, mimic administered experiments and can therefore be used to calculate estimates for specific causal effects of additional children on the mental health status of their parents: multiple births and the first two children having the same gender.

### 4.3.1 Multiple Births as an Instrument for the Number of Children

Multiple births are rare events. They occur in about 1-2% of all births and are therefore considered unexpected events. To see how multiple births affect the total number of children, consider the following setup. Assume that individuals maximize lifetime utility over the total number of children they give birth to during their fertile period. Individual optimization leads to a optimal number $n^*$ of children. If $n^*$ equals 1, an individual needs at least one birth to maximize utility. If the first birth is a twin birth, the total number of children is exogenously increased by 1. If twin births occur randomly, this twin birth has randomly allocated a second child to a couple that ideally wanted only 1 child.
Note that we do not expect the effect of a twin at first birth to be close to unity as for some people $n^* > 1$, so that they would have had more than 1 child anyway. These individuals are termed "always-takers" as they take the treatment (an additional child) regardless of their realization of the instrument (single birth vs. multiple birth). Using the occurrence of a multiple birth at the first birth as an instrument for the total number of children among all those individuals that have experienced at least one birth, allows to calculate the causal effect of this additional child on the outcome of interest for all those individuals that experienced $n = 2 > n^* = 1$ because of a twin birth.[6] Accordingly, our first stage regression looks as follows

$$nchild = \alpha_0 + \alpha_1 multi_1 + \alpha_2 X + \epsilon \tag{4.1}$$

where $nchild$ is the total number of children, $multi_1$ is an indicator that assumes 1 if the first birth was a twin birth and that assumes 2 if the first birth was a triplet birth. $X$ reflects other characteristics relevant to the endogenous variable. These control variables include a full set of age dummies as well as indicator variables for the age at which an individual's first child

[6]Implicitly, we assume that individuals are not constrained in the number of children they give birth to. If individuals were constrained in their capacity to attain $n^*$, our instrument would push some individuals closer to their optimal number of children $n^*$. The validity of the instrumental variable approach remains unaffected when loosening this assumption, as long as the IV monotonically increases $n$ for each individual.

was born in 5 years intervals. We also include country fixed effects to account for the heterogeneity between countries. We do not control for education and other socioeconomic indicators as these variables might be a consequence of early childbearing, rather than a confounding factor. To explore differences by gender, we conduct all analyses for men and women separately.
We also use multiple births at the second birth as an instrument for the number of children. This instrument identifies the treatment effect of an additional child for the group of compliers that would have had only two children in case of the absence of a multiple birth, but was forced into $n = 3$ by the instrument.
The second stage explains our outcome variable of interest (mental health) by the predicted values for the number of children from the first stage.

$$Health = \beta_0 + \beta_1 \hat{nchild} + \beta_2 X + u \tag{4.2}$$

The variable $Health$ represents our indicator for mental health, $\hat{nchild}$ reflects the predicted values from the first stage and $X$ contains the same controls as in equation 4.1. All IV regressions are conducted by using 2 stage least squares.
It is important to notice that multiple births imply, in contrast to consecutive singleton births, that the resulting children grow up at the same time. An additional child induced by a twin birth represents therefore the effect of an additional child plus the effect of having two children growing up at the same time. In our context, the timing of births can play a role when the birth of twins result in events that cause a depression which persists into old age. We come back to this point when investigating pathways for the effect of children on mental health.

### Shortcomings of Multiple Births IV

A valid IV approach requires the instrument to be uncorrelated with the second stage error term. The identifying assumption for the multiple birth IV states that multiple births only affect parents' mental health through the increase in the number of children and by no other means. Multiple births, however, could invalidate this requirement if the probability of a multiple

birth is correlated with unobservable variables. The take-up of fertility treatments increases the individual probability of experiencing a multiple birth and it is likely correlated with observable (e.g. age at birth) and unobservable characteristics of parents. If these unobservable characteristics are directly related to mental health, this would result in a violation of the identifying assumption. However, these treatments became available only from the 1990s onwards. Since almost all of our twin births occurred prior to this date we do not consider the availability of fertility treatments a thread for the validity of our estimates. Moreover, dropping all multiple births occurring after 1990 does not change any of our main results.

### 4.3.2 Sex Composition as an Instrument for the Number of Children

Using sex composition of the first two children as an instrument for the number of children draws on the empirical regularity that parents whose first two children have the same sex, have a higher probability of having a third child than those parents with a balanced sex composition in their first two children. This pattern represents parents' taste for variety. As the realization of a child's gender can be considered an outcome of a natural experiment, this instrument affects a random selection of all those parents with at least two children. Analogously to equation 4.1, our first stage using sex composition of the first two children as an instrument looks as follows

$$nchild = \alpha_0 + \alpha_1 samesex + \alpha_2 X + \epsilon \tag{4.3}$$

where the binary variable *samesex* takes 1 if the first two children have the same sex and 0 otherwise. $X$ includes the same set of control variables as in equation 4.1. We also experiment with sex composition at higher parities as potential instruments. However, in our data set these instruments are not strong enough to provide credible identification. Note that this instrument identifies a different local average treatment effect as compared to the multiple birth instrument for a number of reasons. A third child in response to the instrument is likely to reflect an anticipated and desired increase in

the total number of children, where against a multiple birth is more likely to be an unintended increase in fertility. It is obvious to hypothesize that desired additional children affect parents' wellbeing differently than an unexpected and possibly less desired additional child. Moreover, the timing of child births may play an important role. Twin births induce two children being born and growing up at the same time while births induced by the balanced sex preference of parents occur consecutively. Hence, if children affect parents' mental health because of the demands for personal care when children are young, we might expect the effect of twins to be larger than the respective effect of consecutive singleton births. Lastly, the effect of children born because of the sex imbalance of the first children implies (in contrast to twin births) an additional childbirth which plausibly has a separate effect on health as two singleton births are likely to affect mothers differently than one twin birth.
Using same sex sibship and multiple births as instruments allows to estimate three different local average treatment effects for three specific populations of compliers. In particular, we can study the effect of an unexpected second child and an expected and unexpected third child. However, one shortcoming of this research design is that we are neither able to estimate the effect of the transition from childless couples to one child families, nor are we able to estimate the effect of additional children at a higher birth order.

## 4.4 Data

### 4.4.1 The SHARE Data

We use data from the first and second wave of the Survey of Health, Aging and Retirement in Europe (SHARE). SHARE collects extensive information on health status and both socioeconomic characteristics as well as characteristics of the individual environment (family, social networks). The third wave of SHARE (termed SHAFELIFE) includes retrospective questions about the interviewees biographies such as employment histories, conditions in early

life and fertility histories.[7] SHARE samples about 2.000-3.000 individuals of each participating country. The sample of our analysis includes participants from Austria, Germany, Sweden, Netherlands, Spain, Italy, France, Denmark, Greece, Switzerland, Czechia, Poland, and Belgium. An extensive assessment of mental health has been conducted in the first and second wave of SHARE which took place in 2004 and 2006 respectively. We take the mental health information from the latter wave where possible and the mental health measurement from the first wave, when individuals missed the second wave. We match these variables with the individual fertility biographies provided by SHARELIFE. Our full sample contains 23.028 individuals.

### 4.4.2 Sample Restrictions

We restrict the sample of our IV-analysis to individuals with at least one reported child birth (for the multiple birth at first birth instrument), individuals with at least two births (multiple birth at the second birth instrument), and individuals with at least two children (same sex instrument), to ensure that each individual in the analytical sample could possibly be affected by the instrument. We only consider own (i.e. biological) children. Since we are interested in the total effect of children on parents' wellbeing, we do not distinguish between children alive and those already deceased. The SHARELIFE questionnaire does not directly ask for twin births but rather asks for the year of birth of all natural children. Our twin instrumental variable is therefore constructed in the following way: if a respondent reports that two of his children have been born in the same year, then we assume that they are twins and our instrumental variable assumes 1. If three children are born in the same year, our instrument assumes 2. In all other cases, the IV assumes 0. There are no quadruplets reported in this data set.
The descriptive OLS regression analysis includes observations of all individuals with information on mental health and full fertility biographies, i.e. childless individuals are included. We restrict the female sample to individuals aged between 50 and 90. In our sample the fertility biographies of

[7]For more technical information on the SHARELIFE data see Schröder (2010), for more information on SHARE we refer to Börsch-Supan, Hank, and Jürges (2005).

Table 4.1
Descriptive Statistics

| Sample | no children | | one child | | two children | | more than two children | |
|---|---|---|---|---|---|---|---|---|
| | males | females | males | females | males | females | males | females |
| Age | 65.25 (9.33) | 66.2 (9.92) | 65.8 (9.13) | 65.31 (9.42) | 65.24 (8.65) | 64.26 (9.14) | 67.66 (9.24) | 67.17 (9.67) |
| Euro-D Score | 1.66 (1.88) | 2.54 (2.27) | 1.65 (1.86) | 2.45 (2.18) | 1.50 (1.75) | 2.40 (2.21) | 1.84 (1.91) | 2.73 (2.35) |
| Euro-D Score$\geq$ 4 | 0.17 | 0.31 | 0.16 | 0.31 | 0.14 | 0.30 | 0.19 | 0.35 |
| Ever had depression | 0.13 | 0.23 | 0.14 | 0.25 | 0.13 | 0.24 | 0.13 | 0.25 |
| Use antidepressant drugs | 0.03 | 0.08 | 0.04 | 0.08 | 0.03 | 0.07 | 0.03 | 0.08 |
| Age first birth | | | 29.86 (6.18) | 26.6 (5.71) | 27.49 (4.72) | 24.28 (4.12) | 26.16 (4.30) | 22.83 (3.62) |
| Ever multiple birth | | | 0 | 0 | 0.011 | 0.007 | 0.06 | 0.06 |
| Age youngest child | | | 35.45 | 38.12 | 33.24 | 35.38 | 31.65 | 34.66 |
| $N$ | 1378 | 1459 | 1588 | 1986 | 4130 | 5123 | 3197 | 4166 |

Standard deviations in parenthesis

males start and end later than those of women. Men are less constraint in the timing of fertility than women. As a consequence, men have more time to react to the sex composition of the first two children by having a third child. To account for this effect, we restrict the male sample to include individuals aged between 55 and 90, as dropping individuals between 50 and 54 increases the precision of the estimates for the first stage even though the number of observations is reduced by some 500.
Table 4.1 presents descriptive statistics of our data. On average, individuals in the sample are aged 65. The distribution of age is strongly right-skewed with many individuals between age 50 and 70 and few individuals above 80 years of age. 12.5% of our sample report to have no children. Those individuals with children have on average 2.45 children.
Only 8.3% of the individuals in the sample have minor children at the time of the interview. Only 1.2% have children younger than 10 years. On average, individuals in our sample report that the birth of their youngest child took place 33 years ago. This indicates that the largest fraction of the individuals in the sample has concluded its fertile period a long while ago and that our results reflect the long-term consequences of child bearing.

### 4.4.3 Measurement of Mental Health

Our assessment of mental health is based on the measurement of depressive symptoms provided by the Euro-D scale. Euro-D comprises the measurement of 12 binary indicators that assess the mental condition of interviewees. In particular people are asked about depressive feelings in the last week, hopes for the future, suicide thoughts, feelings of guilt, lessening of interest in things, irritability, appetite, fatigue, ability to concentrate, enjoyable things and tearfulness. A full list of the Euro-D items is provided in table 4.2. The Euro-D scale has been developed with the specific objective to ensure a maximum of comparability across cultural contexts. Its reliability as well as its validity have been confirmed (Prince et al. (1999)). The criterion for the assessment of mental health is the sum of individual symptoms. As dependent variable we use an indicator that takes the value 1 if the individual score is larger than three, which is regarded to be the threshold value for

Table 4.2
List of Euro-D Items to Measure Mental Health

| | **Question item** | **Indicator takes 1 if respondent...** |
|---|---|---|
| 1. | In the last month, have you been sad or depressed? | Says yes |
| 2. | What are your hopes for the future? | Fails to mention any hopes |
| 3. | In the last month, have you felt that you would rather be dead? | Mentions suicidal feelings or wishing to be dead |
| 4. | Do you tend to blame yourself or feel guilty about anything? | Mentions obvious excessive guilt or self-blame |
| 5. | Have you had trouble sleeping recently? | Reports trouble with sleep or recent change in pattern |
| 6. | In the last month, what is your interest in things? | Reports less interest than usual mentioned |
| 7. | Have you been irritable recently? | Says yes |
| 8. | What has your appetite been like? | Reports diminution in desire for food |
| 9. | In the last month, have you had too little energy to do the things you wanted to do? | Says yes |
| 10. | How is your concentration? For example, can you concentrate on a television programme, film or radio programme? | reports difficulty in concentrating on entertainment |
| 11. | What have you enjoyed doing recently? | Fails to mention any enjoyable activity |
| 12. | In the last month, have you cried at all? | Says yes |

depression. Moreover, we evaluate whether an individual reports to ever have suffered from depression as well as a question that assesses whether interviewees currently take anti-depressant drugs. The combination of these three indicators of mental health captures both self-reported depression (assessed by the "ever had" question) and diagnosed and treated depression (represented by the use of antidepressant drugs) as well as latent and possibly undiagnosed mental health problems (as represented by the Euro-D indicator). It has to be kept in mind that individuals undergoing medical treatment might report a Euro-D score that is "artificially" low, if prescribed drugs affect symptoms. Surprisingly, this problem has been largely ignored by the previous literature. There are about 700 observations of individuals that report to take anti-depressant drugs while at the same time having a Euro-D score in the healthy range (i.e. below 4). If in these cases the score is low because of the efficacy of the drugs, our indicator for mental health is no longer a good measure of true mental health but rather reflects the willingness to undergo medical treatment in case of poor mental health. As this is not what we are mainly interested in, we recode these individuals as ill (in the Euro-D sense) and as having suffered from depression.

Overall, table 4.1 documents that women report substantially more depressive symptoms than men. About 16% of all males and about 31% of all women in the sample can be classified as suffering from a depression. The number of people that report to have ever suffered from depression is lower than the means according to the Euro-D criterion. About 3% of males and 8% of females are currently treated with anti depressant drugs. There are no large differences with respect to the single indicators for mental health between individuals with 0,1 or 2 children. The sampled individuals with more than 2 children fare worse in terms of all three dimensions of mental health. However, parents of more than 2 children also have a considerably lower age at first birth, which could indicate a selection effect.

Cross-tabulations of our indicators of mental health reveal that the single indicators are correlated but, still, each indicator implies information that the other two lack. 50.2% of individuals that are classified as suffering from depression according to the Euro-D scale report to have ever experienced a period of depression, i.e., about half of the individuals with a latent symp-

tomatology for depression are not aware of it. About 30% of the individuals who report to ever have suffered from depression currently take antidepressant drugs.

### 4.4.4 Measurement of Possible Pathways

In this article we focus on the long-term consequences of childbearing. We hypothesize that childbearing increases the probability of experiencing critical periods in life, which in turn imply long-term effects on mental health. To empirically assess this hypothesis we investigate the effect of children – using the IV methodology described above – on the likelihood of critical periods in life that took place after the youngest child was born. We then explore to what extent these critical events are related to mental health in old age. The results can shed light on how the birth of a child possibly affects mental health in old age.

We investigate three events: the occurrence of periods of stress, periods of poor overall health and periods of financial hardship which have also been proposed as possible pathways for the link between fertility and mental health by the previous literature (Ross, Mirowsky, and Goldsteen (1990)). We recode the single indicator variables such that they equal 1 if individuals report to have experienced this particular event and it took place after the youngest child has been born. This recoding is needed to make sure that the critical event followed a childbirth and can therefore be attributed to the child birth rather than the other way around.

We cannot distinguish whether a period of poor overall health reflects a period of poor physical or mental health. Nonetheless, we consider it worthwhile to look at the probability of periods of poor overall health for two reasons. First, periods of poor physical health might be a causal link between childbearing and poor mental health in old age. Second, if periods of poor overall health reflect periods of poor mental health, then what we measure is the relation between childbearing and the persistence of poor mental health, which is also an interesting causal mechanism.

# 4.5 Results

## 4.5.1 First Stage Results

This section discusses the results of our first stage, i.e. the effect of the instruments on the total number of children. The results are presented in table 4.3. Note again that we differentiate each analysis by sex and restrict the sample by the number of births. Although this reduces the sample size considerably, it is necessary to make sure that all individuals in the sample are comparable, i.e. could possibly be affected by the instrument. We differentiate the analysis by sex to account for potential heterogeneity in the effect of additional children.

The first two columns in table 4.3 present the estimates for the effect of having two children of the same sex on the total number of children in the sample with all individuals that have at least two children. If individuals have two children of the same sex, they are significantly more likely to have an additional child. The probability increases by 7.9% for men and 9.3% for women. Overall the size of the effect of a same sex sibship is slightly larger but comparable to the effect size in the US (6-7% Angrist and Evans (1998)), Israel (7%, Angrist, Lavy, and Schlosser (2010)) and Norway (8.2% Black, Devereux, and Salvanes (2010)). We also experiment with sex composition at higher parities and separate instruments for two boys vs. two girls as first children, none of which improves upon the single same sex instrument in terms of instrument strength. The sex composition of the first two children offers a borderline strong instrument for our male sample (first stage F-statistic of 9.5) and a relatively strong instrument for the female sample (F-statistic of 17).

Columns 3 and 4 show the impact of having a multiple birth at the first birth. On average, having a multiple birth increases the number of children by about 0.81 children. The results for men and women do not differ substantially. A multiple birth at the second birth (columns 5 and 6) results in an increase in total fertility by 0.82 children for men and by about 0.75 for women. These results suggest that multiple births imply an enormous variation in the total number of children. The F-statistics for the instruments on

Table 4.3
First Stage Results

| | (1) | (2) | (3) | (4) | (5) | (6) |
|---|---|---|---|---|---|---|
| Dep. Variable: | | | *Number of Children* | | | |
| Sample | men | women | men | women | men | women |
| Same sex | 0.079*** | 0.093*** | | | | |
| | (3.08) | (4.14) | | | | |
| *mb* at 1.birth | | | 0.815*** | 0.826*** | | |
| | | | (7.07) | (7.55) | | |
| *mb* at 2.birth | | | | | 0.817*** | 0.753*** |
| | | | | | (6.72) | (7.00) |
| *N* | 6835 | 9852 | 8303 | 11950 | 6793 | 9809 |
| F-Stat excl.IV | 9.46 | 17.17 | 50.00 | 56.98 | 45.16 | 49.02 |

*t* statistics in parentheses; * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.
control variables include full set of age dummies, country fixed effects, education dummies, age at first birth in 5 years intervals, indicator for second survey wave. Sample restrictions are as follows: all individuals with at least two children (first two columns), all individuals with at least one reported birth (columns 3 and 4), individuals with at least 2 reported births (columns 5 and 6). *mb* indicates a multiple birth.

the first stage are considerably above the critical threshold of 10, indicating that our instrument does not suffer from a weak-IV problem.
The effect of multiple births on total fertility in Europe is much larger than in Israel (0.43-0.5 Angrist, Lavy, and Schlosser (2010)) which can be explained by a much higher average number of children in Israeli families which makes the total number of children less responsive to multiple births at low parities. Our first stage estimates are comparable to previous estimates for Norway (0.68-0.75 Black, Devereux, and Salvanes (2005)).

### 4.5.2 Descriptive OLS Results

We start discussing our evidence on the relationship between fertility and mental health by documenting the results from an Ordinary Least Squares (OLS) regression. We set up the OLS model by regressing our indicators for mental health on dummy variables that equal one if individual $i$ has 1, 2, 3, 4 or 5 and more children. The omitted category is 'childless individuals'. The control variables included are the same as in the IV regressions. This setup

Table 4.4
OLS-results: Number of Children and Mental Health

| | (1) | (2) | (3) | (4) | (5) | (6) |
|---|---|---|---|---|---|---|
| Dep. Variable: | *Euro-D Score≥4* | | *depression ever* | | *drugs for depression* | |
| Sample | Males | Females | Males | Females | Males | Females |
| Childless | - | - | - | - | - | - |
| child 1 | -0.000845 | -0.0100 | 0.0107 | 0.00547 | 0.0104 | 0.00288 |
| | (-0.07) | (-0.65) | (0.87) | (0.38) | (1.60) | (0.32) |
| child 2 | -0.0198* | -0.0167 | -0.00547 | -0.00487 | 0.0000833 | 0.00210 |
| | (-1.82) | (-1.27) | (-0.53) | (-0.39) | (0.02) | (0.27) |
| child 3 | -0.00939 | -0.0255* | -0.00446 | 0.00258 | -0.00323 | 0.00402 |
| | (-0.77) | (-1.76) | (-0.38) | (0.19) | (-0.53) | (0.47) |
| child 4 | 0.0200 | 0.000758 | 0.00456 | 0.00185 | 0.0108 | -0.00405 |
| | (1.24) | (0.04) | (0.30) | (0.10) | (1.33) | (-0.36) |
| child 5+ | 0.0348* | 0.0469** | -0.0276 | 0.00972 | -0.00385 | 0.00494 |
| | (1.80) | (2.29) | (-1.51) | (0.50) | (-0.40) | (0.40) |
| *N* | 10935 | 13513 | 10935 | 13513 | 10935 | 13513 |
| | *Restricted Sample: At least one Child* | | | | | |
| # of children | 0.00811** | 0.00855** | -0.00518* | 0.000524 | -0.00217 | -0.000508 |
| | (2.53) | (2.55) | (-1.71) | (0.16) | (-1.35) | (-0.25) |
| N | 9458 | 11969 | 9458 | 11969 | 9458 | 11969 |
| | *Restricted Sample: At least two Children* | | | | | |
| # of children | 0.0146*** | 0.0130*** | -0.00345 | 0.00226 | -0.000115 | -0.000230 |
| | (3.79) | (3.25) | (-0.95) | (0.59) | (-0.06) | (-0.10) |
| N | 7770 | 9868 | 7770 | 9868 | 7770 | 9868 |

Clustered standard errors in parentheses; * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.
Control variables include age at first birth (in 5 year intervals), country fixed effects and full set of age dummies.

allows to study mean differences in mental health by the number of children. Table 4.4 shows our results. The results for males are shown in columns 1,3 and 5. Standard errors, clustered at the individual level, are presented in parentheses. Our results do not point to a systematic relationship between parenthood and mental health. Fathers and mothers are not consistently better or worse off than the reference group composed of individuals without children. Moreover, within the group of parents mental health does not systematically differ by the number of children. Only few of the coefficients are significant. Parents of 5 and more children have a 3-4 percentage points in-

creased probability of suffering from a depressive symptomatology according to the Euro-D criterion. This finding, however, does not apply to the other indicators of mental health we investigate. The sign of the coefficient even switches in several cases. These results differ from the results by Buber and Engelhardt (2008), who also use the SHARE data, for two reasons. First, the samples used differ as we used the second wave of SHARE and added all observations of individuals that took part in the first wave of SHARE and in SHARELIFE. Second, in contrast to Buber and Engelhardt (2008), who use an extensive set of control variables, we only control for country fixed effects, age and the age at first birth.
The lower part of the table shows the results for the restricted samples of all individuals with at least one or at least two children. We show these results for the sake of comparability as all IV results presented in the next sections are based on restricted samples. The results are similar to the results shown at the top of the table. More children seem to go in hand with a higher probability for depressive symptoms but no higher probability for the other measures of mental health. The results at the top part suggest that the coefficients are mostly driven by the fifth child.

### 4.5.3 Second Stage Results

This section discusses the results for the 2 Stage Least Squares (2SLS) analysis of the effect of children on mental health. The main results are presented in table 4.5. The first two columns of table 4.5 provide estimates of the effect of children on the respective indicator using the same sex instrument for the number (#) of children . The third and fourth column provide evidence using multiple births at the first birth as IV, the fifth and sixth columns present results for a multiple birth at the third birth as IV for total fertility. Our evidence suggests that increases in total fertility that result from the sex composition of the first two children do not significantly affect parents' mental health status. The estimated coefficients are negative for all indicators of mental health in the males sample. For women, the coefficients are consistently positive and the magnitude of the coefficients suggests a substantial but insignificant effect on self-reported depression and the use of antidepres-

Table 4.5
THE EFFECT OF CHILDREN ON MENTAL HEALTH

| | (1) | (2) | (3) | (4) | (5) | (6) |
|---|---|---|---|---|---|---|
| Sample | males | females | males | females | males | females |
| IV | same sex | | 1.birth is mb | | 2.birth is mb | |
| Dep. Variable: | *Euro-D Score≥4* | | | | | |
| # of Children | -0.108 | 0.0506 | 0.0665 | 0.0195 | -0.0756 | 0.204** |
| | (-0.93) | (0.52) | (1.46) | (0.39) | (-1.46) | (3.17) |
| Dep. Variable: | *Ever had depression* | | | | | |
| # of Children | -0.0368 | 0.149 | 0.0438 | 0.0299 | 0.0634 | 0.173** |
| | (-0.36) | (1.49) | (1.04) | (0.62) | (1.34) | (2.86) |
| Dep. Variable: | *Antidepressant Drugs* | | | | | |
| # of Children | -0.0451 | 0.0843 | 0.000177 | -0.0626* | -0.00725 | 0.100** |
| | (-0.81) | (1.36) | (0.01) | (-2.01) | (-0.29) | (2.66) |
| $N$ | 6835 | 9852 | 8303 | 11950 | 6793 | 9809 |

$t$ statistics in parentheses; $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Control variables include age at first birth in 5 years intervals, country fixed effects and full set of age dummies. Sample restrictions are as follows: all individuals with at least two children (first two columns), all individuals with at least one reported birth (columns 3 and 4), individuals with at least 2 reported births (columns 5 and 6). *mb* indicates a multiple birth.

sant substances. Columns three and four present the results using multiple births at first birth as IV. The results hereof do not show a consistent and statistically significant pattern across our indicators of mental health, either. The estimates for the effect of children on their fathers' mental health are positive but not significant at usual levels of significance for all three indicators of mental health.

In the female sample, neither the Euro-D indicator, nor the probability of reported depression are significantly affected by additional children. However, a second child appears to reduce the likelihood of using antidepressant drugs by 6 percentage points for women (significant at the 10% level).

The last two columns present the results for the twin at second birth instrument. This instrument mostly increased the number of children from two to three children. Our estimates suggest that a third child does not affect fathers' mental health status. The estimated effects are small and none is near the critical significance levels. The coefficients even switch sign when com-

paring the results for the single indicators of mental health. In contrast to this result, a third child appears to strongly affect mothers' mental health. Having a third child increases a woman's probability of suffering from depression by 20 percentage points according to the Euro-D scale and by 17 percentage points according to depression self-reports. A third child induced by a multiple birth also increases the probability of taking anti-depressant drugs by 10 percentage points. Comparing this finding to the results shown in columns 2 and 4, suggests that the third child (rather than the second) appears to be the critical margin for mothers. However, the adverse effect of children resulting from unexpected (and possibly undesired) multiple births is considerably larger (and significant) compared to the effect of a third child resulting from the sex composition of the first two children.

### 4.5.4 How Do Grown-up Children Affect Parents' Mental Health?

Table 4.6 presents the estimated effects of additional children on the probability of experiencing specific crises. Similar to the results on mental health, we do not find consistent evidence for an effect of the third child resulting from the sex composition of the first two children on the probability of critical events for either sex.

Contrasting to this result, children resulting from a multiple birth appear to affect their parents life course. The estimates for the effect of the second child (shown in columns 3 and 4) suggest that an additional child might even reduce the probability of particularly stressful periods in life. There is no evidence for an effect of the second child on periods of poor health status or on periods of financial hardship.

The last two columns point out that the third child induced by a multiple birth at the second birth significantly affects the probability of crisis for both sexes. For men, a third child increases the probability of experiencing periods of stress by 14.5 percentage points and the probability of periods characterized by financial hardship by 13 percentage points. For women, the probability of periods of stress is not increased by a third child. However, the third child significantly increases the likelihood for periods of poor overall

health (by 15 percentage points) and financial hardship (by 13 percentage points).

Table 4.7 provides evidence for the relation between the experience of these critical events and mental health using the full sample. We conduct an OLS regression of our measures of mental health on the indicators for crises and the control variables used in all previous models. Our results support the hypothesis that critical periods in life are linked to mental health in old age. All of the critical events are associated with significant increases in the probability of depression in old age for both sexes. Periods of stress are linked to an increase of 4-8 percentage points in the probability of depression.

Individuals who suffered situations of financial hardship have a higher probability of 3 to 6 percentage points for depression. Women are considerably more sensitive with respect to these events than men.

However, the results in table 4.7 cannot be given a causal interpretation as

Table 4.6
Pathways for the Relationship Between Fertility and Mental Health

| | (1) | (2) | (3) | (4) | (5) | (6) |
|---|---|---|---|---|---|---|
| Sample | males | females | males | females | males | females |
| IV | same sex | | 1.birth is mb | | 2.birth is mb | |
| Dep. Variable: | *period of stress* | | | | | |
| # of Children | -0.092 | -0.007 | -0.094 | -0.139** | 0.145** | 0.042 |
| | (-0.64) | (-0.07) | (-1.61) | (-2.48) | (2.08) | (0.66) |
| Dep. Variable: | *period of poor health* | | | | | |
| # of Children | -0.147 | 0.091 | -0.043 | -0.002 | 0.064 | 0.148** |
| | (-0.97) | (0.85) | (-0.73) | (-0.03) | (0.95) | (2.27) |
| Dep. Variable: | *period of financial hardship* | | | | | |
| # of Children | -0.038 | 0.131 | -0.044 | -0.002 | 0.133** | 0.133** |
| | (-0.35) | (1.48) | (-0.98) | (-0.05) | (2.52) | (2.53) |
| $N$ | 6835 | 9852 | 8303 | 11950 | 6793 | 9809 |

$t$ statistics in parentheses; * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.
Each coefficient represents one regression. Control variables include age at first birth in 5 years intervals, country fixed effects and full set of age dummies. Sample restrictions are as follows: all individuals with at least two children (first two columns), all individuals with at least one reported birth (columns 3 and 4), individuals with at least 2 reported births (columns 5 and 6). *mb* indicates a multiple birth.

Table 4.7
Crises and Depression

| Dependent Variable: | (1) | (2) | (3) | (4) |
|---|---|---|---|---|
| | *euro-D Score≥4* | | *depression ever* | |
| | Males | Females | Males | Females |
| Period of stress | 0.0364*** | 0.0590*** | 0.0689*** | 0.0765*** |
| | (4.49) | (6.72) | (8.98) | (9.15) |
| Period of poor health | 0.112*** | 0.125*** | 0.0690*** | 0.112*** |
| | (14.07) | (14.24) | (9.14) | (13.50) |
| Period of financial hardship | 0.0314*** | 0.0608*** | 0.0316*** | 0.0537*** |
| | (3.04) | (5.66) | (3.22) | (5.25) |
| $N$ | 9452 | 11950 | 9452 | 11950 |

$t$ statistics in parentheses; * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.
Control variables include age at first birth in 5 years intervals, country fixed effects and full set of age dummies.

we cannot rule out other mechanisms driving the correlation between critical events and depression in old age. Taking the evidence from tables 4.6 and 4.7 together, our results support the hypothesis that children affect their parents' mental health status in old age by increasing the risk of experiencing critical periods earlier in life.

## 4.6 Conclusions

This article provides first evidence for the causal effect of an additional child on parents' probability of suffering from depression. In contrast to the previous and mostly descriptive literature, we used instrumental variables for the number of children to estimate the magnitude of the causal effect.
Our results indicate that there is a large heterogeneity in the effect of children on mental health. We find supportive evidence for relatively large and significant positive effects for women using the twin birth instrument, i.e., having three rather than two children increases the probability of poor mental health by 10-20 percentage points. In respect to the baseline, a third child (induced by a multiple birth) increases the odds for depression by about 70 percent. The magnitude of these effects seems surprisingly large. However,

given that we obtain similar results for three different indicators of mental health, we are confident that the effect is accurately measured and internally valid. Comparing the magnitude of these coefficients to those estimated by the previous literature is also reassuring. Angrist (2004) and Cáceres-Delpiano and Simonsen (2010) provide evidence that children resulting from multiple births have substantial negative effects on marital stability, increase the risk of poverty and lead to high blood pressure and obesity of mothers. If these effects accumulate over time, it is not surprising that additional children have adverse long-term effect on mental health.

There is no evidence for a similar effect for men. There is also no significant evidence for such an effect when using the same sex instrument, although the point estimates suggest a positive (i.e. harmful) effect for women.

Moreover, we find no evidence for the transition from 1 to 2 children to negatively affect parents' mental health status.

The finding that a third child resulting from a twin birth is detrimental while a third child resulting from the sex composition does not appear to affect mental health, can be attributed to the fact that our instruments identify different local average treatment effects. The multiple birth instrument forces individuals into a possibly unintended level of fertility which affects parents in a different way than a desired and anticipated increase in fertility. Moreover, the timing of births induced by the instruments differs: twin births result in the stress of raising two infants of equal age. In contrast to this, children resulting from the same sex instrument are born in a consecutive order where parents decide upon the exact timing of births. This argument might explain the heterogeneity in the causal effects by the single instruments. Moreover, since we do not find an effect of children induced by the same sex instrument on depression in old age, we conclude that it is not the stress of an additional childbirth that affects parents' mental health but rather the number of children and the circumstances of their respective births.

We propose an explanation for our findings. Children resulting from a multiple birth increase parents' probability of suffering financial shortages and increase women's probability of experiencing periods of particular bad overall health. These two critical events are associated with poor mental health in

old age. women react substantially more sensitive than men to these critical events. This finding also implies that periods of dramatic financial shortages are likely to bear long-term mental costs, in particular for women.

Before drawing conclusions from our findings, we discuss two limitations of our econometric approach. First, the use of instrumental variables usually restricts the interpretation of the identified effects to the narrow population of compliers and does not allow generalizations beyond this particular group of individuals. In our case, the compliant population is composed of individuals with a certain number of births who gave birth to an additional child because of a multiple birth or because the first two children have the same gender. As only few individuals experience these events, the generalizability of our results has been questioned. We think that our results speak for a broader population as the event of a (possibly unplanned) child can happen also in other contexts (e.g. teenage pregnancies). However, at present we cannot test whether this generalization is justified. Unfortunately, information on unplanned pregnancies is usually not available in large surveys.

The second concern calls into question the fact that children investigated in our analyses were born 30-40 years ago and social policy has made progress since then. Children's allowances have been increased in most countries and child care programs now better allow reconciling active labor force participation with family live. We cannot tell whether the long-term effects of children born nowadays will be comparable to those estimated from the SHARE-data. However, the results shown in those recent studies cited above suggest that multiple births are still an event that threatens parent's wellbeing.

Our findings carry a number of policy implications. First, as there is no evidence for a positive effect of children on mental health in old age, to encourage getting children as a mean to prevent depression in old age seems not an appropriate action. On the other hand, our results suggest that declining fertility levels are not one cause for the increase in the prevalence of depression in most European countries.

Second, as multiple births seem to be particularly harmful, measures to prevent multiple births should be taken into account. The risk of a multiple birth considerably increases when relying on in-vitro fertilization, as the number of implanted embryos usually exceeds 1. The adverse effects of multiple births

as evidenced in our study must be traded-off against the higher chances of a successful conception.
Third, our results suggest that multiple births increase the risk of financial shortages. Higher children's allowances for multiple births are one way of reducing the risk of poverty as a consequence of fertility shocks even though this might invoke fairness concerns.

**Acknowledgements:**
This paper uses data from SHARELIFE release 1, as of November 24th 2010 and SHARE release 2.3.1, as of July 29th 2010. The SHARE data collection has been primarily funded by the European Commission through the 5th framework programme (project QLK6-CT-2001- 00360 in the thematic programme Quality of Life), through the 6th framework programme (projects SHARE-I3, RII-CT- 2006-062193, COMPARE, CIT5-CT-2005-028857, and SHARELIFE, CIT4-CT-2006-028812) and through the 7th framework programme (SHARE-PREP, 211909 and SHARE-LEAP, 227822). Additional funding from the U.S. National Institute on Aging (U01 AG09740-13S2, P01 AG005842, P01 AG08291, P30 AG12815, Y1-AG-4553-01 and OGHA 04-064, IAG BSR06-11, R21 AG025169) as well as from various national sources is gratefully acknowledged (see www.share-project.org/t3/share/index.php for a full list of funding institutions).

# Chapter 5

# The Effect of Changes in Household Composition on ADHD

## 5.1 Introduction

Attention Deficit Hyperactivity Disorder (ADHD) is a neuro-behavioral disorder characterized by the joint presence of attention deficits and physical hyperactivity. ADHD has spread dramatically in the past two decades. The prevalence rate is now estimated to range between 6 and 12% among minors, without large differences across industrialized countries (Biederman and Faraone (2005), Faraone et al. (2003)).[1] Several recent articles show that ADHD implies considerable negative long-term consequences for affected individuals: it adversely affects human capital acquisition, thus reducing later earnings (Fletcher and Wolfe (2008), Currie and Stabile (2006), Ding et al. (2009)), it favors engaging in criminal activities (Fletcher and Wolfe (2009)) and it is related to consumption of illegal substances (Bieder-

[1]The term prevalence is defined as the ratio of diseased individuals to the total size of the relevant population. As there is usually imperfect knowledge about the number of diseased individuals, the number of diagnoses is oftentimes taken as a proxy. This leads to the problem that one cannot distinguish an increase in the number of diagnoses from an increase in the number of diseased individuals. As public awareness for ADHD rose, it is not surprising that the number of diagnoses increased. Whether the true prevalence of ADHD has increased over the last decades is therefore an unsettled issue.

man et al. (1998)). ADHD not only causes disadvantages for children and families affected by ADHD but also entails tremendous costs for health care and schooling systems. Pelham, Foster, and Robb (2007) estimate the cost per child suffering from ADHD to be about 14.500 USD per year including health care costs and costs for special education.[2] Understanding the causes of ADHD is crucial for a better treatment of affected children and for possible policies to limit the further spread of the disorder. The previous literature has mainly pointed to physical causes of ADHD, namely genetic disposition and the toxicological environment before birth and in early ages. The more recent literature emphasizes the interplay between genetic disposition and environmental factors in the incidence and shape of mental disorders (e.g. Nigg (2006), Counts et al. (2005)).[3] However, little is known yet about the environmental conditions that contribute to ADHD.

In this article, we test whether symptoms of ADHD evolve as a reaction to changes in environmental circumstances. Our hypotheses are guided by the insight that in a situation where children compete for parents' resources certain behavioral reactions can prove superior vis-à-vis others. Parents' time spent with children is possibly one such scarce resource in the household. If these resources get scarcer, a change in behavioral strategies could prove worthwhile. We hypothesize that ADHD-type behavior could just be one consequent behavioral response.

We consider two changes in the household composition of a child that plausibly exert stress on the individual child. First, we test whether a father's absence in the household has an effect on children. Since our estimation is based on within-child variation, the effect of father's absence on a child's behavior represents the effect of father's move-out of the household. We consider this event representing the climax of a family conflict which possibly affects children in the formation of their skill set. Moreover, the move-out of a father likely is associated with less quality time that fathers spend with

[2]Since the calculation in Pelham, Foster, and Robb (2007) does not take all future costs into account (e.g. the decreased earnings potential is not included), we consider this to be the lower bound of the true economic costs of each case of ADHD that accrue to society.

[3]A large discussion deals with the question whether ADHD should be defined as a mental health condition, a behavioral disorder or a character trait. We follow the mainstream literature here considering ADHD a health condition and using the terms "condition" and "disorder" interchangeably.

each child remaining in the household.

Second, we test whether the birth of a sibling affects children. One possible mechanism by which the birth of siblings can affect older children is by reducing the amount of familial resources a child gets.[4] An increase in family size, for example, implies that parents can spend less time per child relative to the time before the increase, assuming that parents' time budget does not change. Only recently, several studies have shown that children with a lower birth order (i.e. older children) actually have better outcomes along several dimensions than children with a higher birth order.[5] Moreover, Price (2008) has documented that first born children get more parental quality time than their later born siblings. We hypothesize that an increase in family size and the associated change in the intra-familial allocation of resources can nonetheless be a stressful experience for two reasons. First, although Price (2008) shows that lower birth order is associated with a higher amount of quality time children spent with their parents, the birth of a child can result in a deterioration of a child's subjective position within the family and a perceived loss of status. Second, even if lower birth order children received more resources by their parents than higher birth order children, the birth of children could be stressful to older children if the higher amount of resources they get is an equilibrium outcome, i.e., the result of a specific behavior.

We consider it relevant to contrast the effect of a supposedly rather mild event such as the birth of a sibling to the effect of a family dissolution represented by fathers' absence which poses a major mental burden for children (Amato and Keith (1991)).

ADHD-type behavior is a continuum. On the one extreme side, very focused, attentive and calm children are represented. On the opposite tail of the distribution, children with a distinct health condition are represented. However, not only clinical conditions can cause impairment. Currie and Stabile (2009) provide suggestive evidence showing that even a small number of symptoms

[4]Formally spoken and assuming that each child is treated equally by parents, the share of parents' resources each child receives is 1/n. The birth of a sibling decreases this share to 1/(n+1) for each child.

[5]Iacovou (2001) and Gary-Bobo, Prieto, and Picard (2006) establish that children with a lower birth order perform better in terms of several test scores and educational attainment.

can cause impairments in terms of educational attainment and test scores. To account for this ambiguity, we focus our analysis on both outcomes on a binary indicator for a severe ADHD symptomatology as well as on a score that sums up individual symptoms.

We contrast our estimates of the effect of familial events on ADHD to estimates on two other indicators of a child's mental health status, namely on a depression index and an index for anti-social behavior. Since it is widely acknowledged that environmental factors contribute to the formation of depressive symptoms and anti-social behavior, we use the estimated effects on a depression index and an index for anti-social behavior as a benchmark.

This article has several advantages compared to the previous literature on the social origins of ADHD. First, we use a large panel data set that follows up on children over a period of 8 years on average. In contrast to this, previous studies use mostly small samples. Second, our assessment of ADHD and related disorders is based on screener questionnaires, i.e. questionnaires that were asked to all mothers of the sampled children. We therefore avoid biases that could result from selective diagnosis or treatments decisions.[6]

Third, our panel data set allows to control for unobserved heterogeneity. This is a crucial advantage of our study design because a large part of the literature argues that (unobservable) genetic disposition is the most important determinant of ADHD. Moreover, controlling for unobserved heterogeneity allows to account for the effect of toxic substances each child was exposed to until the age of four – the second major determinant of ADHD according to the literature. Thus, any effect that we find is either due to a purely environmental effect or to an interaction between genetic disposition and social

[6]There is now evidence suggesting that the probability of being diagnosed with ADHD is not based on individual symptoms alone. Two recent articles show that the probability of being diagnosed with ADHD depends on the relative age of children within their school class (Elder (2010), Evans, Morrill, and Parente (2010)). It is argued that this finding probably results from an (inadequate) comparison of children that are within one school grade but at different developmental stages, which leads to inappropriate diagnoses. Moreover, in a recent article, Bokhari and Schneider (forthcoming) show that the degree of school accountability affects the number of diagnoses and prescriptions of drugs used to treat ADHD in the US. The latter result indicates that teachers and headmasters of schools respond to increased performance pressures by referring more children to an ADHD evaluation. This evidence highlights the importance to use screener questionnaires rather than diagnoses as indicators for ADHD.

environment. To the best of our knowledge, no previous study has provided convincing evidence for such an effect.
Our results support the hypothesis that ADHD is strongly related to changes in household composition. The number of ADHD symptoms increases when fathers leave the household as well as when household size increases by the birth of a brother or sister. Thus, ADHD appears to be determined by changes in the social environment just like other disorders such as depressive mood and conduct disorders. The magnitude of the effects on ADHD is comparable to the magnitude of the effects on depressive symptoms and anti-social behavior.
The next section reviews related studies on the determinants of ADHD. In section 5.4, our econometric approach is described in detail. The main results are presented in section 5.5, along with a set of robustness exercises that confirm our main findings. The last section concludes and discusses directions for future research.

## 5.2 Related Literature on the Causes of ADHD

### 5.2.1 Genes

Genetic disposition is the primary argument to explain why ADHD symptoms often cluster within families.[7] Previous research suggests that as much as 80% of the variance in ADHD symptoms could be determined by genetic disposition (Biederman and Faraone (2005) and the references therein).
There are three strands of the literature on the genetic origins of ADHD. The genetic disposition argument is backed by studies that compare concordance rates of ADHD-symptoms in monozygotic and dizygotic twins. Monozygotic twins share 100% of genes whereas dizygotic twins share on average only 50% of genes. Monozygotic twins should therefore either both have the genes for ADHD or not have the genes. Hence, if genes played a role in ADHD prevalence, monozygotic twins should have a higher concordance rate than dizygotic twins. This hypothesis is backed by evidence from several studies

[7]We can only briefly touch upon the different strands of this literature. For a very extensive review on the causes of ADHD see for example Nigg (2006).

(e.g. Edelbrock et al. (1995), Willcutt, Pennington, and DeFries (2000)).[8]
A second strand of the genome-hypothesis research draws on samples of adopted and unadopted children and their respective families. In these studies, ADHD patterns in families with (biological) children are compared to patterns in families with adopted children. Adopted children have shown to be more often unlike their (adopting) families with respect to ADHD symptomatology in comparison to children living in their biological family (e.g. Sprich et al. (2000)). This finding is taken as evidence for a genetic determinism as the proportion of shared genes is higher within relatives by blood than among adopting families and their adopted children. However, since both adopted children and adopting families are highly selected samples, the validity of causal conclusions drawn from this type of studies has been disputed (e.g. Timimi and Leo (2009) p. 65 onwards).
The third line of research identifies specific genes on the individual DNA that are related to symptoms of ADHD. The idea that a single "gene for ADHD" exists that deterministically codes whether a child will suffer from ADHD or not has been abandoned. It is now well established that a number of genes is responsible for the phenotype of ADHD and that the disposition expressed by these genes interacts with environmental conditions in a complex and yet poorly understood way.

### 5.2.2 Toxicological Environment

Exposure to several substances during a mother's pregnancy has been shown to be associated with symptoms of ADHD. Well documented correlates of ADHD are increased lead levels in the child's blood and the mother's tobacco and alcohol consumption during pregnancy.[9] It is suspected that these substances can affect the cerebral development of the fetus and the newborn child which later in life increases the probability of behavioral disorders (see Nigg (2006) and the references therein).

[8]For a criticism of this methodology see for example the extensive discussion in Joseph (2006) p. 39 onwards.

[9]see Tuthill (1996) for lead exposure, see Linnet et al. (2003) and the references therein for tobacco and alcohol consumption.

### 5.2.3 Social Environment

The empirical base for a conclusive statement about the social causes of ADHD is still rather small. Most previous studies use small and selected samples and apply methods that point out correlations rather than causal relationships.

While there is some research on the effect of familial conflicts and parents' divorces on a child's symptoms of ADHD, the literature on the effect of fathers' absence and sibship size on ADHD is quite small. Family conflicts likely precede the move-out of fathers in families. Moreover, since familial conflict could be an event exerting mental stress on children just like father's absence or the birth of siblings, we consider the literature on family conflict to be informative and comparable to our approach. There is now quite extensive evidence on the positive correlation between family conflict and children having ADHD (Biederman et al. (2002), Counts et al. (2005), Biederman et al. (1995)). For example, Biederman et al. (2002) document that familial conflicts are significantly more often reported in families with ADHD children than in families without. However, this evidence is often regarded to reflect a mechanism running from ADHD in children to familial conflict or to represent that households in which children with ADHD live, often also include parents with ADHD (because of the heritability of genes), which itself increases the likelihood of repeated familial conflict. For example, the British National Collaborating Centre for Mental Health states in its guidelines for the diagnosis and treatment of ADHD that "discordant family relationships, however, may be as much a consequence of living with a child with ADHD as a risk of the disorder itself" (National Collaborating Centre for Mental Health (2009) p.31).

A related strand of the literature has investigated the effect of marriage dissolution on the mental health status of affected children. While it is well acknowledged that children whose parents divorced have a higher probability of suffering from emotional disorders, only few studies included measures of ADHD.[10] Breivik and Olweus (2006) show that parents' divorce is significantly correlated with children's self report of ADHD-symptoms in a large

[10]For a general overview over the literature on the association between divorce and the mental health status of children, see Amato (2001) and Amato and Keith (1991).

sample of Norwegian children. Cherlin, Chase-Lansdale, and McRae (1998) is one of the few articles on this topic to use a regression model that accounts for unobserved heterogeneity, using data from the British National Child Development Study. Their results suggest that parents' divorce affects the probability that a child experiences emotional disorders. However, since Cherlin, Chase-Lansdale, and McRae (1998)'s main variable of interest is an index of mental health that includes besides measures of ADHD symptoms also other dimensions of mental health, it is not possible to infer specifically on the impact of parental conflicts on ADHD.

Growing up without a father in the household has been documented to be associated with an increased risk of experiencing a number of adverse outcomes such as early childbearing and low educational achievement (e.g. McLanahan and Sandefur (1994)). However, results specifically on ADHD are rare. Harel and Brown (2003) find for a sample of children from Rhode Island that children living with a single parent do not have a higher probability of being referred to an ADHD evaluation by their teacher than children growing up with both of their natural parents. However, the presence of stepparents increases the probability of such a referral as well as of a medical treatment for ADHD.

The evidence on the role of siblings is very small and did not yield consistent evidence (e.g. Biederman, Faraone, and Monuteaux (2002)). Mostly, sibship size is considered one dimension of a psycho-social "adversity" index, whose other dimensions are familial conflict, socioeconomic status (SES) and parents' psychopathology. The overall sibship size has not been shown to predict ADHD (Biederman et al. (1995)). This finding could partially reflect that an association between sibship size and ADHD mixes up the effect of sibship size and the birth order effect. Some evidence even suggests that single children have a higher probability to be treated for ADHD (Harel and Brown (2003)). We are not aware of any studies investigating the consequences of the birth of a sibling in a panel framework.

Although the previous literature has pointed to a correlation between adverse familial events and children's ADHD symptomatology, the mainstream literature does not consider these events a causal determinant of ADHD

symptoms.[11] An international consensus statement of leading researchers in the field of ADHD states that ADHD is not driven by family conflict and parenting quality (Barkley et al. (2002)). While family environment is not considered to cause ADHD, it is accepted though that an intact family environment may help to manage children with ADHD or may cushion the consequences of it (Johnston and Mash (2001), American Psychiatric Association (2000)).

Most recently, economists have started to investigate the determinants of ADHD diagnoses. Two articles have shown that the relative age of children within their school class is an important determinant of diagnosis and treatment of ADHD (Elder (2010), Evans, Morrill, and Parente (2010)). Children that are relatively young in their school class have an increased risk of being diagnosed with and treated for ADHD. This finding suggests that either the social context (here: relative age effects) puts pressure on ADHD-like behavior of infants or that diagnosis and treatment decisions are influenced by inadequate comparisons with older children.

Moreover, a related literature in economics explores the determinants of non-cognitive skills. The notion of non-cognitive skills (e.g. Heckman and Rubinstein (2001)) encompasses characteristics such as persistence, tenacity and self-discipline which are similar to the specific skills absent in children who suffer from ADHD. Both ADHD as well as non-cognitive skills have substantial predictive power for outcomes in later life such as earnings and educational achievements (e.g. Heckman, Stixrud, and Urzua (2006), Fortin (2008)). Although ADHD has mainly been defined as a disease or a severe behavioral disorder, we consider traits of the disorder (namely the attention deficit symptoms) as an extreme form of a lack of specific non-cognitive skills. Since we do not look at specific symptoms of ADHD, we leave a more elaborate attempt to link these two literatures open for future work.

[11] Nigg (2006) for example states that except for extreme psychological trauma which account for a tiny fraction of all ADHD cases, the effects of parenting on ADHD are "generally nil" (p. 256). Barkley (2000) names the hypothesis that family environment causes ADHD a "myth" that has been proven wrong by large evidence.

## 5.3 Data

### 5.3.1 Data Set

We use data from the National Longitudinal Survey of the Youth (NLSY) Children and Young Adults Survey. The original NLSY is a panel survey of men and women born between 1957 and 1964. The survey started in 1979 and conducted biannual surveys since then. In 1986, a new panel was started surveying the children of the female NLSY participants. Since then, every second year this separate survey was conducted.
We restrict our analysis to children aged 4 to 15. 4 is the minimum age for children in the survey. 16 is considered an age when children begin leaving their parents' household and starting their own one. Therefore, our indicators become more difficult to interpret. We make use of all waves conducted between 1986 to 2008, which allows to analyze up to 6 observations of each child. To take part in the NLSY Children and Young Adults Survey, children are required to live in the household of their natural mothers. Children older than 15 years may stay in the sample while leaving the household of the mother. Since we are interested only in children aged 15 and below, we know that all sampled children live in the household of their mothers (for details of the sampling procedure, see Center for Human Resource Research (2004) p. 6).

### 5.3.2 Measurement of Mental Disorders

Our assessment of mental health is based on screener questionnaires. In each interview mothers are asked a set of questions that screen symptoms of mental disorders without explicitly asking for the disorder itself. This method of assessing mental health avoids several biases. First, parents might overstate their child's ADHD history to excuse poor achievements in school. Second, diagnoses have been shown to depend on a child's relative age in its school class, which indicates that the context matters for the probability of diagnosing ADHD (Elder (2010), Evans, Morrill, and Parente (2010)). We therefore consider an analysis based on scores that are derived from questionnaires to be a superior measure of ADHD for our purpose as compared to data on

diagnoses.

The questionnaires in the NLSY comprise 5 questions to evaluate ADHD-type behavior, 5 questions on depression and anxiety as well as 6 questions on anti-social behavior.[12] The items in the questionnaire have been proposed by the American Psychiatric Association (2000) in the Diagnostic and Statistical Manual of Mental disorders (IV) and are a subset of items that are frequently used as a diagnostic questionnaire. According to the American Psychiatric Association, ADHD should be diagnosed if a certain number of symptoms persists over a period of at least 6 months. A diagnosis further requires that these symptoms must impair a child in its normal activities in at least two settings (e.g. at home and in school). We cannot test the degree of impairment attached to ADHD in this article but we refer to the recent literature that has convincingly shown that symptoms of ADHD are related to poor achievements in school, which suggests a severe degree of impairment (e.g. Currie and Stabile (2009)).

The full diagnostic questionnaire of the Diagnostic and Statistical Manual of Mental Disorders (IV) includes 9 items to assess attention deficits and 9 items on hyperactivity. The diagnosis of ADHD (combined type) is suggested if on both scales at least 6 out of 9 criteria are met and persisted over a period of at least 6 months. A predominantly inattentive or a predominantly hyperactive-impulsive ADHD can be diagnosed if only on 1 scale at least 6 criteria are met (American Psychiatric Association (2000)).

These items (or closely related ones) have also been used by previous economists to assess the consequences of mental disorders on human capital acquisition(e.g. Currie and Stabile (2009), Vujic, Webbink, and Koning (2008), Fletcher and Wolfe (2008)).

We exploit the single indicators of mental disorders in two ways. First, we look at the raw additive score of symptoms. Second, we construct a binary indicator that takes the value 1 if a child is at the top end of the distribution of the respective score. We set the target cut-off at the 90th percentile. The 10% threshold is a reasonable cut-off, given that the prevalence rate of ADHD is estimated to lie between 6 and 12% (Biederman and Faraone (2005)). This cut-off threshold has been widely used in previous studies (e.g.

[12]The full list of items can be found in the appendix to this chapter.

Currie and Stabile (2006), Fletcher and Wolfe (2008)).
Our criterion is even more selective than the criterion suggested by the American Psychiatric Association (2000), since in our case, the top 10% of the distribution satisfy 5 out of 5 criteria whereas the APA suggests that meeting 6 out of 9 criteria suffices for a diagnosis. If we took the same ratio as suggested by the APA to construct the cut-off in our variables, more than 20% of boys would have been classified as having an ADHD symptomatology. We test the sensitivity of our results with respect to the choice of the cut-off value in the robustness section.
The upper 10% are mostly represented by the maximum score in each dimension, i.e. for ADHD, the top 10% children have 5 out of 5 possible symptoms. Since the single indicators only assume integer values, the exact percentage of the cut-off differs from 10%. See the descriptive statistics for details. An alternative approach would have been to use age standardized scores for mental disorders and to set the cut-off at exactly 90%. We decided not to use age-standardized scores to facilitate the interpretation of our results.

The assessment of depression and anxiety disorders and anti-social behavior is also based on screener questionnaires. Since the respective diagnostic questionnaires of the American Psychiatric Association (2000) are designed for older children, adolescents, and adults, it is not adequate for the very small children sampled in the NLSY Children and Young Adults sample.[13] The questionnaire used in the NLSY is therefore an adapted version of the APA diagnostic questionnaire. The assessment of depression and anxiety is based on 5 items that include amongst others the question whether a child feels worthless or inferior, or whether a child feels that no one loves him. The assessment of anti-social behavior is based on 6 items, which among others ask whether a child is regularly cruel to others or whether a child deliberately destroys things.

[13]For example, the questionnaire of the American Psychiatric Association (2000) suggested to assess anti-social behavior asks whether individuals committed violent attacks that include the use of weapons.

### 5.3.3 Descriptive Statistics

Table 5.1 presents the descriptive statistics of the NLSY data. The sampled children are on average 9 years old. The minimum age of the sampled children is four years, the maximum is 15. Since interviews collecting the relevant variables are conducted biannually, the maximum number of interviews a child could participate in is 6. About 12% take part in 5 or 6 interviews, the average is 3.7 interviews per child. Children from Hispanic and black families account for about half of the sample.

In about one fifth of all observations, at least one newborn sibling is reported to live in the household. In this case, the number of newborn siblings is mostly one. In only 2% of all observations, more than 2 newborn siblings live in the household. Four outliers with very high values for the number of newborn siblings have been dropped from the data set. Overall, about 35% of all children experience variation in the number of newborn siblings during the observation period.

The fraction of children whose father is not living in the same household is quite substantial. About 40% of all children sampled in 1996 do not live in the same household as their fathers. Overall, about 20% of the sampled children experience that their father leaves or joins the household during the observation period. The persistence of father's absence is about 94% from one wave to the next one, i.e., the share of fathers moving (back) to their children is small.

We present the means of the single behavioral indicators for the 1996 NLSY cross-section. Not surprisingly, boys have on average a higher hyperactivity score than girls. About 8% of all boys in the sample and about 4% of girls are reported to suffer from 5 out of 5 symptoms of ADHD. It is a general finding that boys suffer about twice as often from ADHD as girls. The fraction of boys and girls suffering from 5 out of 5 symptoms of ADHD commensurates with prevalence rates for ADHD estimated in previous studies (e.g. Faraone et al. (2003)). The gender differences in anti-social behavior and depressive symptoms are less pronounced.

The persistence in ADHD symptomatology is quite low indicating that for many children ADHD symptoms are a temporary phenomenon rather than

Table 5.1
Descriptive Statistics

| | *Boys* | | *Girls* | |
|---|---|---|---|---|
| **Variable** | **Mean** | **Std.dev** | **Mean** | **Std.dev** |
| *Full Sample:* | | | | |
| Age | 9.13 | 3.02 | 9.11 | 3.02 |
| Fraction Hispanic | 0.21 | | 0.20 | |
| Fraction Black | 0.30 | | 0.31 | |
| Number of Observations | 18.140 | | 17.671 | |
| *1996 Cross-Section:* | | | | |
| Hyperactivity-Score | 1.99 | 1.59 | 1.44 | 1.46 |
| Depression-Score | 1.16 | 1.21 | 1.25 | 1.23 |
| Anti-social-Score | 1.64 | 1.62 | 1.19 | 1.39 |
| No symptoms of ADHD | 0.23 | | 0.36 | |
| More than 3 symptoms of ADHD | 0.2 | | 0.14 | |
| 5 out of 5 symptoms of ADHD | 0.08 | | 0.04 | |
| At least 4 out of 5 symptoms for Depression | 0.06 | | 0.06 | |
| At least 5 out of 6 symptoms for Anti-social behavior | 0.15 | | 0.08 | |
| Father not living in household (HH) | 0.40 | | 0.43 | |
| Fraction having at least 1 sibling aged 0-2 in HH | 0.18 | | 0.18 | |
| Number of Observations | 2.084 | | 2.061 | |

a persistent one. Two years after a child has been reported to be in the top category for ADHD, the child has a probability of only 36% to again be in the top category. We will address the persistence of the effect of our treatment variables in section 5.5.4. Symptoms of hyperactivity are strongly correlated with symptoms of anti-social behavior (r=0.53) and depression (r=0.44).

## 5.4 Empirical Strategy

Our empirical strategy is based on the following regression model

$$Disorder_{it} = \beta_0 + \beta_1 Sib_{it} + \beta_2 Dad_{it} + \gamma X_{it} + \nu_i + \epsilon_{it} \tag{5.1}$$

The dependent variable *Disorder* represents the mental health indicator of interest. The regressor *Sib* equals the number of siblings aged 0 to 2 years of child $i$ that currently live in the household in which child $i$ lives. Note that these siblings are not necessarily biological siblings of child $i$, but can as well be half-siblings or children of child $i$'s mother's partner. We consider the distinction between biological and half-siblings of minor importance for our argument.

The variable *Dad* equals 1 if the father of child $i$ is missing in the household and 0 otherwise. The vector $X$ contains the full set of age dummies and the full set of survey year fixed effects. Controlling for age and time flexibly is important as the prevalence of ADHD varies over time and by age.[14] $X$ also includes the total number of children living in the household.

Most important in this empirical setup is the child specific fixed effect $\nu_i$. The fixed effect absorbs time constant unobserved heterogeneity. Most importantly, this represents a child's and parents' genetic disposition for ADHD, parental quality and SES to the extent that these variables do not change over time. Moreover, fixed effects also reflect the complete history of exposure to environmental toxins of child $i$ until $i$ entered the panel. The fixed effect strategy allows us to state that any evidence suggesting that $\beta_1$ or $\beta_2$ is significant, accounts for either the effect of changes in the social environment or the effect of an interaction between genetic disposition and changes in the social environment ("gene-environment-interaction"). Our results cannot be driven by a genetic disposition alone, as the latter is constant over time.

We contrast our results for the ADHD score and indicator with results using (analogously constructed) indicators for depressive symptoms and anti-social behavior. We consider this a helpful extension since it facilitates the interpretation of the magnitude of the estimated effects. All regressions are estimated by linear models.

[14] We also experiment with linear, quadratic and higher order polynomials for age and survey year. Our main results are not sensitive with respect to the specification of age and survey year effects.

## 5.5 Results

In this section we present the main results of our analysis. The next section presents graphical evidence on the relation between fathers' absence and mental disorders. Section 5.5.2 presents our main regression results which are extended in sections 5.5.3 to 5.5.5.

### 5.5.1 Graphical Evidence

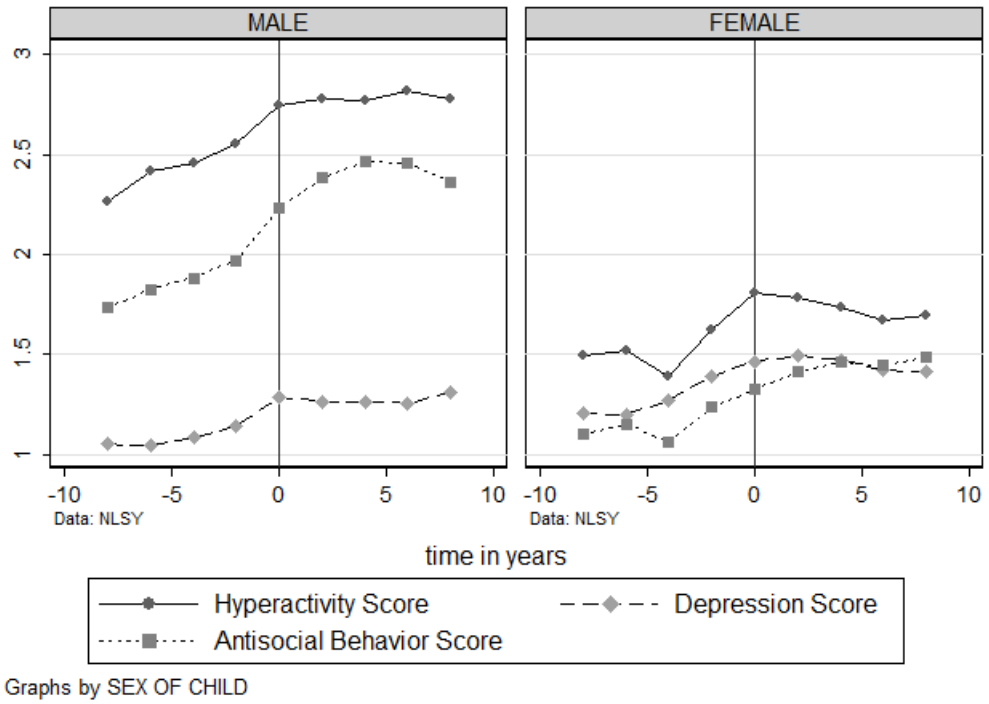
We start discussing our evidence by depicting the evolution of mental disorders over time. Figure 5.1 graphs the means of the hyperactivity, depression and anti-social behavior scores of all children whose father moves out any time during the observation period. The time axis is normalized such that $t = 0$ marks the point in time at which the father was first reported to be absent in the household of child $i$. All scores depicted are age-standardized. The graphs are characterized by a pronounced upward trending for all time series and both sexes before $t = 0$. This is not surprising for the period before a father moves out since family conflict is likely to precede the move-out of the father and since this conflict is likely to be reflected in problematic behavior. The levels of the hyperactivity and anti-social behavior scores are considerably higher for boys than for girls.

The graphs point to three findings. First, they document that as fathers move out, children's mental health scores deteriorate. At $t = 0$, all three scores move upwards for boys. The jump at $t = 0$ is less pronounced for girls. Remarkably, all three scores do not fall back to their initial levels but rest on a higher level for both sexes. This suggests that growing up without a father in the household persistently affects problematic behavior. Second, the graphs show that the peak in the single scores is not reached before $t = 0$. This is an important finding since the reversal of causality could be a concern for our hypothesis. If a child's mental disorder precedes her father's move-out, this could indicate that father's absence is the result of a child's mental disorders rather than the other way around.[15] Third, the graph indicates that the increase in the ADHD-score at $t = 0$ is comparable to the increase

[15] This point will be addressed more thoroughly in section 5.5.6.

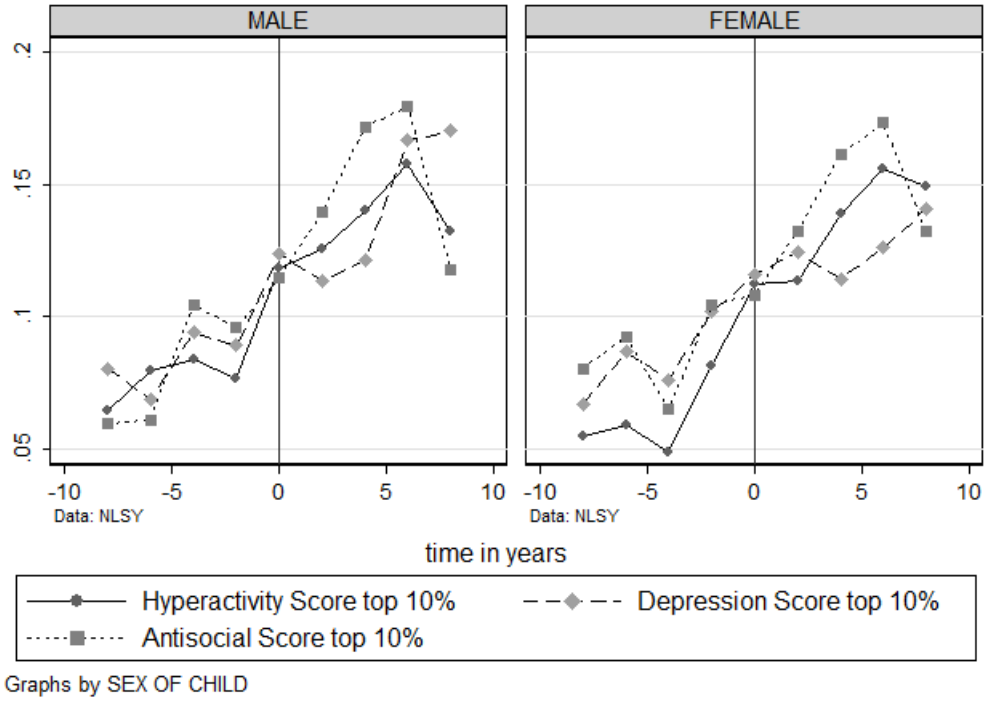
in the scores for anti-social behavior and depressive symptoms, suggesting that children are affected in either dimension by a similar magnitude.
Figure 5.2 shows the evolution of the three binary mental health indicators of all children whose father leaves the household while the child is part of the NLSY. The colored lines mark the share of children that are reported to have a very high score (approximately commensurate with the 90th percentile) for the underlying disorder. As in figure 5.1, the time series are characterized by a positive trend until $t = 0$. At the time of father's move-out, each indicator jumps by about 2-4% to a higher level, on which it persists for $t > 0$ or even further increases. Overall, the graphs presented suggest a negative effect of the absence of fathers on their children's mental health status.

Figure 5.1
Father's Absence and Behavioral Disorders: Scores




This graph depicts the evolution of the three behavioral disorder scores in children whose father leaves the household. The point in time at which this occurs is normalized to $t = 0$. All scores have been age-standardized.

Figure 5.2
Father's Absence and Mental Disorders: Top Decile




This graph depicts the evolution of behavioral disorders represented by the share of children that are in the top decile of the respective score. The underlying scores have been age-standardized.

### 5.5.2 Regression Results

Table 5.2 presents our regression results for equation 5.1. The first two columns show the results for the ADHD score, first estimated by OLS and then by the fixed effects (FE) model. The upper part of the table presents the results for boys, the lower part those for girls. In all OLS regressions, standard errors are clustered at the individual level. The OLS results show that both the absence of fathers as well as the birth of siblings are significantly associated with higher ADHD-scores for both sexes. As a father leaves the household, the number of symptoms increases by 0.39 for boys and by 0.29 for girls which roughly corresponds to one fourth of the standard deviation of the ADHD-score. The OLS-coefficients, however, may represent the effect

Table 5.2

The Effect of Familial Stress on Mental Disorders: Hyperactivity and Depression

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
|---|---|---|---|---|---|---|---|---|
| Dependent Variable: | ADHD-Score | | ADHD-top-10% | | Depression-Score | | Depression-top-10% | |
| Method: | OLS | FE | OLS | FE | OLS | FE | OLS | FE |
| Sample: | *Only boys* | | | | | | | |
| Father absent in HH | 0.388*** | 0.0367 | 0.0560*** | 0.0217* | 0.340*** | 0.0430 | 0.0355*** | 0.0128 |
| | (10.29) | (0.92) | (9.48) | (2.52) | (12.81) | (1.31) | (7.61) | (1.71) |
| Number of Sib 0-2 | 0.0974** | 0.131*** | 0.00429 | 0.00483 | -0.00355 | 0.0596** | -0.00499 | -0.000386 |
| | (3.16) | (5.31) | (0.76) | (0.91) | (-0.15) | (2.94) | (-1.04) | (-0.08) |
| $N$ | 17604 | 17604 | 17604 | 17604 | 17604 | 17604 | 17604 | 17604 |
| $R^2$ | 0.04 | 0.66 | 0.02 | 0.48 | 0.07 | 0.60 | 0.02 | 0.43 |
| Sample: | *Only girls* | | | | | | | |
| Father absent in HH | 0.289*** | 0.0552 | 0.0303*** | 0.0153* | 0.267*** | 0.107** | 0.0255*** | 0.0230** |
| | (8.29) | (1.43) | (6.68) | (2.25) | (9.79) | (3.16) | (5.34) | (2.91) |
| Number of Sib 0-2 | 0.149*** | 0.120*** | 0.00967* | 0.0122** | -0.00499 | 0.0515* | -0.00511 | 0.00557 |
| | (5.06) | (5.09) | (2.01) | (2.94) | (-0.21) | (2.49) | (-1.10) | (1.15) |
| $N$ | 17178 | 17178 | 17178 | 17178 | 17178 | 17178 | 17178 | 17178 |
| $R^2$ | 0.05 | 0.63 | 0.02 | 0.46 | 0.06 | 0.59 | 0.02 | 0.42 |

$t$ statistics in parentheses; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

This table shows estimates for a regression of equation 5.1. Control variables include the full set of age and survey year dummies, the number of children in the household as well as individual fixed effects where indicated (FE). OLS standard errors are clustered by individuals.

of omitted and confounding variables rather than the effect of the respective variables. The second column presents the results of the fixed-effects regressions. The estimation of the coefficients is based on within-individual variation, i.e. the effects represent the consequences of changes in the independent variable within one child rather than mere level differences between children with and without a father in the household. The coefficient on the absence of fathers is considerably smaller as compared to the OLS estimates and not statistically significant for both sexes. In contrast to this, the coefficients on additional siblings remain in the same order of magnitude and highly significant for both sexes, as compared to the OLS coefficients. The birth of a sibling increases the average ADHD-score of children by about 0.12 symptoms. The differences between OLS and FE-estimates suggest that the omitted variable bias (in the OLS regressions) is positive and large for the father's absence coefficient whereas it is small for the coefficient on additional siblings.

Columns 3 and 4 present the results using the binary indicator that takes 1 if child $i$ has 5 out of 5 possible symptoms as the dependent variable. Comparing the results for this indicator with those for the ADHD symptom score, allows to distinguish whether the changes in household composition we investigate affect ADHD symptomatology within a "normal" range or whether they push the behavior beyond a threshold which is considered a disorder. The OLS regression coefficients for the absence of fathers are again highly significant for both sexes. The coefficient for additional siblings is significant only for the girls sample. The fixed-effects regressions provide supportive evidence for the hypothesis that both the move-out of fathers as well as the birth of siblings considerably affect the probability of having a severe symptomatology of ADHD. The move-out of fathers increases the probability of ADHD for boys by 2.2 percentage points and by 1.5 percentage points for girls. The effect of additional siblings is significant for girls and similar in magnitude to the effect of father's move-out. The discrepancy between the results for the ADHD-score and the binary ADHD indicator emphasizes that the move-out of father's does not affect ADHD symptomatology everywhere in the distribution of the symptom score to the same extent. Our results suggest that those children at the lower end of the score of symptoms might

be less affected than children that already have 3 or 4 symptoms before the move-out of their fathers. By contrast, the effect of additional siblings appears to increase the ADHD-score at the lower end of its distribution.

The right hand part of the table documents the results for analogous regressions on a depression score and on a binary indicator for attaining the top-category in the depression score. The OLS results point to a close association between the absence of fathers and depressive symptoms. However, when controlling for unobserved heterogeneity, this association becomes looser for both sexes. The relationship is significant for girls, with the absence of fathers associated with an increase in the probability that girls are reported to have a severe degree of depressive symptoms by 2.3 percentage points. Additional siblings increase the number of depressive symptoms in children by about 0.05 symptoms.

Table 5.3 presents the results for anti-social behavior. The OLS estimates indicate a close association between anti-social behavior and both the absence of fathers and the birth of siblings. The coefficients of the fixed effects models are considerably smaller than the OLS-coefficients indicating that omitted variables (such as socioeconomic status) largely drive the association between a father's absence and his child's degree of anti-social behavior. Both sexes are affected by changes in household composition in terms of anti-social behavior. Our estimates indicate that when fathers leave the household, symptoms of anti-social behavior increase significantly by 0.08 for boys but remain constant for girls, holding unobserved heterogeneity constant. The birth of siblings increases anti-social behavior significantly for both sexes. The effect size is in a similar order of magnitude as the effect of fathers' absence. Compared to the results for ADHD, the effects are substantially smaller.

Comparing the effects of fathers' absence and newborn children across our dependent variables of interest, it is remarkable that the effects on ADHD are large and robust as compared to the effects on depressive symptoms and anti-social behavior. This is strong supportive evidence for the hypothesis that ADHD is a behaviorial response to environmental conditions, just like other mental disorders.

### 5.5.3 What Explains Symptoms of ADHD?

The $R^2$ of the single analyses provides further insight about the relative explanatory power of the regressors. On average, the fixed effects explain about 55-60% of the variation in the mental health scores and about 42-55% of the variance in the binary indicators, without large differences across the single mental disorders. Given that the fixed effects capture the joint impact of genes, time-constant environmental factors as well as the stock of toxic substances children were exposed to before entering the panel, it is not surprising that fixed effects account for a large fraction of the explained variance in the regressands. Our results, however, suggest that the isolated effect of genes accounts for less than 80% of the variance in ADHD symptoms. The partial $R^2$ also suggests that the absence of fathers and the birth of

Table 5.3
The Effect of Familial Stress on Mental Disorders: Anti-social Behavior

| | (1) | (2) | (3) | (4) |
|---|---|---|---|---|
| Dependent Variable: | Anti-social Score | | Anti-social-top-10% | |
| Method: | OLS | FE | OLS | FE |
| | *Only boys* | | | |
| Father absent in HH | 0.519*** | 0.0885* | 0.0512*** | 0.00989 |
| | (13.99) | (2.26) | (9.70) | (1.33) |
| Number of Sib 0-2 | 0.0390 | 0.0668** | -0.00213 | 0.00523 |
| | (1.25) | (2.76) | (-0.41) | (1.14) |
| $N$ | 17604 | 17604 | 17604 | 17604 |
| $R^2$ | 0.07 | 0.67 | 0.04 | 0.54 |
| | *Only girls* | | | |
| Father absent in HH | 0.365*** | 0.0339 | 0.0253*** | 0.0117* |
| | (11.94) | (0.95) | (7.49) | (2.16) |
| Number of Sib 0-2 | 0.0352 | 0.0841*** | 0.00677 | 0.0138*** |
| | (1.24) | (3.86) | (1.54) | (4.19) |
| $N$ | 17178 | 17178 | 17178 | 17178 |
| $R^2$ | 0.06 | 0.61 | 0.02 | 0.46 |

$t$ statistics in parentheses; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$
Control variables include the full set of age and survey year dummies, the number of children in the household as well as individual fixed effects where indicated (FE). OLS standard errors are clustered by individuals.

siblings explain only a small fraction of the variance in ADHD symptoms. The partial $R^2$ of these regressors is below 1%, as is the isolated contribution of each of the following regressors: race, sex, age and survey year fixed effects (results not shown).

### 5.5.4 Does the Effect on Mental Disorders Persist?

In this section, the temporal structure of the effect of our events of interest is addressed. A diagnosis of ADHD requires a number of symptoms of ADHD to persist over a minimum period of 6 months (American Psychiatric Association (2000)). The questions in the NLSY refer to behavior within at least the last three months. Since the NLSY interviews are conducted only every second year, we cannot recover information on behavioral problems within the last 6 months. We investigate the persistence of mental disorders over 2 years and again contrast the results for ADHD to those for depression and anti-social behavior. Since this is a "tighter" definition of persistence compared to the criterion suggested by the American Psychiatric Association, we are confident that this test provides credible information on the dynamic effect of familial events. The regression setup is analogous to equation 5.1, except that we use lagged values of fathers' absence and the birth of siblings instead of contemporaneous values. The results are presented in table 5.4. Compared to the estimates of the contemporaneous effects presented in tables 5.2 and 5.3, the effect for the two year lagged events do not change in a consistent manner. Our results suggest that a father's move-out as well as the birth of a sibling does not have persistent effects on mental health in all dimensions. Comparing the results for the ADHD indicator with the results for the other indicators suggests that the effect of father's absence on boys' ADHD symptomatology disappears while the effect of born siblings gets larger. This finding indicates that the effect of born siblings is persistent for boys ADHD scores and quite large compared to the effect on other mental disorders. The results for girls are presented in the bottom part of the table. As compared to the contemporaneous effects, the effect of additional siblings becomes slightly smaller whereas the effect of father's absence persists. This result stands in contrast to the effects on depressive symptoms and anti-social

Table 5.4
Dynamic Effects of Fathers' Absence and Birth of Siblings

| | (1) | (2) | (3) |
|---|---|---|---|
| Dependent Variable: | | | |
| Child $i$ belongs to top decile in | ADHD | Depression | Anti-social |
| | | *Only boys* | |
| L2.Father absent in HH | -0.0127 | -0.00277 | -0.00612 |
| | (-1.04) | (-0.29) | (-0.57) |
| L2.Number of Sib 0-2 | 0.0176** | 0.00636 | 0.00671 |
| | (2.70) | (1.27) | (1.17) |
| $N$ | 11368 | 11368 | 11368 |
| | | *Only girls* | |
| L2.Father absent in HH | 0.0176* | 0.00210 | 0.00450 |
| | (1.98) | (0.21) | (0.58) |
| L2.Number of Sib 0-2 | 0.000301 | -0.0108* | 0.0107* |
| | (0.06) | (-1.98) | (2.53) |
| $N$ | 11220 | 11220 | 11220 |

$t$ statistics in parentheses; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$
*L2.* represents the two years lagged values of the respective regressors. Control variables include the full set of age and survey year dummies, the number of children in the household as well as individual fixed effects.

behavior which become insignificant over the period of 2 years. Summarizing the evidence presented in this section, we conclude that the effects of father's absence in the household and the birth of siblings on symptoms of ADHD weakly persist over time.

We also experiment with larger dynamic regression models. The drawback of these larger models is that selective attrition becomes a problem. The presence of mental disorders increases the probability of dropping out of the sample.[16] If we use higher order time lags, selective attrition is likely to bias our results even more. Since those individuals with the strongest symptomatology for mental disorders are most likely to drop out, our results on persistence likely represent a lower bound for the true persistent effect of father's absence and the birth of a sibling on ADHD.

[16]The results for this analysis is not presented in this paper but is available upon request.

### 5.5.5 Effect Heterogeneity

In this section, we investigate whether the results discussed above are mainly driven by some specific group of individuals or whether they are backed by the whole sample population. We split the sample along the dimensions number of newborn siblings, age of child, maternal education and the race of the child and conduct the analysis presented above for subgroups. We then test pairwise whether the coefficients in the subsamples are equal. For the sake of brevity, we only show the results for the binary indicator that takes 1 if child $i$ has the maximum ADHD score and for the fixed effects regression models represented by equation 5.1. Table 5.5 shows our results. The first three columns report the estimated results for the boys' sample, columns 4-6 those for the girls' sample.

*By Number of Newborn Siblings*

There is some concern that our results could be driven by few families with several newborn children, for example by families with multiple births. Multiple births could be a rare but particularly stressful experience to older children.[17] We show that this is not the case by conducting our analysis for a restricted sample of children with a maximum of 1 sibling aged 0 to 2 years and by comparing the results for this subsample to the results estimated on the basis of the full sample. The results are presented at the top of table 5.5. The estimated coefficients do not differ substantially between children with less than 2 newborns siblings and the entire sample. This finding emphasizes that our results are not driven by few cases with several newborn siblings but that already one newborn sibling affects older children in the development of their mental health status.

*By Age of Child*

The second part of the table shows our results for the original samples split at the median of age. It could be that children respond more (or less) sensitive to changes in household composition in early childhood compared to later childhood. To describe possible differences we conduct the analysis on the sample stratified by age. The point estimates for fathers' absence are

[17]The full sample includes observations of children which are reported to have up to 4 siblings aged 0 to 2 years.

Table 5.5
Effect Heterogeneity: Analysis for Various Subgroups

| | (1) | (2) | (3) | (4) | (5) | (6) |
|---|---|---|---|---|---|---|
| Dep. Variable: | Binary Indicator for ADHD symptomatology | | | | | |
| | Only Boys | | | Only Girls | | |
| *# of siblings aged 0-2:* | *<2* | *full sample* | | *<2* | *full sample* | |
| Father absent | 0.0214* | 0.0217* | | 0.0179** | 0.0153* | |
| | (2.47) | (2.52) | | (2.62) | (2.25) | |
| # of Siblings | 0.0009 | 0.0048 | | 0.0096 | 0.0122** | |
| | (0.14) | (0.91) | | (1.92) | (2.94) | |
| *N* | 17188 | 17604 | | 16755 | 17178 | |
| *Age of child:* | *≤8* | *>8* | | *≤8* | *>8* | |
| Father absent | 0.0176 | 0.0075 | | 0.0222 | 0.0034 | |
| | (1.03) | (0.54) | | (1.60) | (0.30) | |
| # of Siblings | 0.0028 | 0.0021 | | 0.0107 | 0.0056 | |
| | (0.31) | (0.21) | | (1.39) | (0.73) | |
| *N* | 7819 | 9785 | | 7691 | 9487 | |
| *Mothers' years of schooling:* | *<13* | *≥13* | | *<13* | *≥13* | |
| Father absent | 0.0307* | 0.0127 | | 0.0115 | 0.0112 | |
| | (2.52) | (1.11) | | (1.19) | (1.21) | |
| # of Siblings | 0.00399 | 0.00747 | | 0.0136* | 0.0131* | |
| | (0.55) | (0.99) | | (2.39) | (2.24) | |
| *N* | 10851 | 6723 | | 10730 | 6423 | |
| *Race:* | *Hispanic* | *black* | *other* | *Hispanic* | *black* | *other* |
| Father absent | 0.0170 | 0.00873 | 0.0366** | 0.00308 | 0.0139 | 0.0247** |
| | (0.95) | (0.49) | (3.14) | (0.21) | (0.94) | (2.95) |
| # of Siblings | 0.0110 | -0.00118 | 0.00596 | 0.0116 | 0.0233** | 0.00520 |
| | (1.03) | (-0.11) | (0.80) | (1.31) | (2.63) | (1.00) |
| *N* | 3664 | 5175 | 8765 | 3447 | 5223 | 8508 |

*t* statistics in parentheses; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. This table presents the results for equation 5.1 for restricted samples. The sample restrictions are highlighted in *italics*. Control variables include the full set of age and survey year dummies, the number of children in the household as well as individual fixed effects.

considerably larger for younger children. However, the differences between the effects for the two subsamples are not significant. The effect of additional siblings does not differ significantly by age either.

*By Mothers' Education*

In the lower part of the table, we compare the results for the subsample of children coming from highly educated mothers to those of children of less educated mothers. We split the sample at the median of the distribution of mothers' years of schooling, which is at grade 13. Education is one dimension of socioeconomic status and a proxy for permanent income. Socioeconomic status could alter how families cope with specific stressful situations. For example, a large literature suggests that parental income can buffer the consequences of health shocks in children (e.g. Case, Lubotsky, and Paxson (2002)). Our results show that (low) maternal education amplifies the effect of fathers' absence on ADHD in boys. The differences in the effect of fathers' absence is marginally significant. We do not find supportive evidence for a similar effect in the girls' sample. The effect of additional siblings does not differ by maternal education.

*By Race*

The bottom part of the table presents results for the subsamples defined by race. We distinguish between black, Hispanic and all other children. Race is still a marker of socioeconomic status. Our results only weakly point at differences in the effects by race. The effects of fathers' absence are smallest for black and Hispanic children as compared to all other children (significant for boy sample). This finding somewhat contrasts to the results for the highly educated mothers as mothers of Hispanic and black children are on average less educated than all other mothers. The effect of additional siblings does not differ significantly by race.

Overall, the results presented in this section do not robustly suggest that one particular group of children is at a higher risk of developing symptoms of ADHD but our results rather suggest that all children are affected similarly by their fathers' absence as well as by the birth of siblings.

### 5.5.6 Robustness

**Using Different Cut-offs for the ADHD-score**

The American Psychiatric Association suggests to diagnose ADHD if a child meets 2/3 of the criteria assessed by the (DSM IV) questionnaire. Our outcome variable that supposedly indicates a severe ADHD symptomatology assumes 1 if a child is reported to suffer from 5 out of 5 possible symptoms. We used this different cut-off value to match children in our sample classified as "having ADHD" with ADHD prevalence rates which have been estimated to be in the range of 6-12%. In this section, we redo the empirical analysis presented in table 5.2 using different cut-off values in order to inspect the sensitivity of our results. The first new indicator assumes 1 if child $i$ has at least 3 out of 5 symptoms. The second new indicator takes 1 if $i$ has at least 4 out of 5 symptoms. About 20% of boys and 14% of girls in the sample have more than 3 symptoms of ADHD. Table 5.6 shows the regression results using these different cut-off values to generate the binary indicators of ADHD.

The evidence presented in the table suggests that our results are slightly sensitive with respect to the choice of the cut-off value. While fathers' absence is

Table 5.6
Robustness: Different Cut-off Values for the ADHD-score

| | (1) | (2) | (3) | (4) |
|---|---|---|---|---|
| Cut-off Criterion | Score $\geq 3$ | | Score $\geq 4$ | |
| Sample: | Boys | Girls | Boys | Girls |
| Father absent in HH | 0.0192 | 0.0151 | 0.0104 | 0.0233* |
| | (1.41) | (1.20) | (0.87) | (2.33) |
| Number of Sib 0-2 | 0.0320*** | 0.0323*** | 0.0171* | 0.0254*** |
| | (3.80) | (4.20) | (2.34) | (4.16) |
| $N$ | 17604 | 17178 | 17604 | 17178 |

$t$ statistics in parentheses; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$
Control variables include the full set of age and survey year dummies, the number of children in the household as well as individual fixed effects. The dependent variable is a binary indicator that takes 1 if the ADHD score of child $i$ is greater than or equal to 3 (columns 1 and 2) or 4 (columns 3 and 4).

significant at the 10% level for the 5 out of 5 symptoms criterion (table 5.2), it is not significant at lower cut-offs, suggesting that the move-out of fathers pushes some boys from 4 to 5 symptoms but only few from 2 to 3 symptoms. The effect of siblings does not operate at the top end of the ADHD symptom distribution (as suggested by the results from table 5.2), but rather at lower cut-offs. For girls, the birth of siblings favor additional ADHD symptoms everywhere in the upper part of the distribution of the ADHD score. The effect of father's absence operates mainly at the high end of the score.
Overall, the results in table 5.6 confirm our previous evidence. Regardless of the cut-off we take, we find evidence in favor of the hypothesis that changes in household composition affect ADHD symptomatology.

### Do Symptoms of ADHD Precede Fathers' Absence?

This section tests whether a high degree of ADHD symptomatology precedes fathers' absence in the household. One concern for the interpretation of the results presented in the last sections could be that fathers' absence is a reaction to the problem behavior of a child rather than the other way around. Moreover, we argued that familial conflict likely takes place before the move-out of fathers. If it is this conflict that causes children to develop ADHD, we would also expect that ADHD scores increase even before fathers were first reported to be absent in a household. We cannot disentangle these two mechanisms. However, we can investigate to what extent behavioral disorders are observable shortly before fathers leave the household in order to shed light on the possibility of reversed causality. Our approach to test this is similar to the estimation of equation 5.1. We set up a regression model as follows:

$$DAD_{it} = \alpha_0 + \alpha_1 HYPR_{it-1} + \alpha_2 X_{it} + v_i + u_{it} \tag{5.2}$$

$DAD$ indicates the presence of child $i$'s father at time $t$. $\text{HYPR}_{it-1}$ indicates the number of symptoms of child $i$ two years before. The vector $X$ contains the same control variables as before and $v_i$ represents the child-specific fixed effect. If a child's symptoms of ADHD precede the absence of her father, then the coefficient $\alpha_1$ should be positive and significant. The results are presented

Table 5.7
REVERSED CAUSALITY: DOES ADHD PRECEDE FATHERS' MOVE-OUT?

| | (1) | (2) | (3) | (4) |
|---|---|---|---|---|
| Dep. Variable | Father present in Household | | | |
| Sample | Boys | Girls | Boys | Girls |
| L2.Symptoms of ADHD | -0.000321 | 0.00139 | | |
| | (-0.13) | (0.52) | | |
| L2.Symptoms of ADHD | | | -0.00115 | 0.0213 |
| (*top 10% indicator*) | | | (-0.10) | (1.39) |
| *N* | 11368 | 11220 | 11368 | 11220 |

*t* statistics in parentheses; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$
Control variables include the full set of age and survey year dummies, the number of children in the household as well as individual fixed effects.

in table 5.7. We evaluate the regressions for both the symptom score as well as for the binary indicator using 5 out of 5 symptoms to define the threshold for a severe condition as measures of ADHD. Note that the sample size has diminished considerably due to the higher number of consecutive observations needed to estimate the model with fixed effects. This decrease in sample size can result in a selected sample as the probability of leaving the survey correlates with mental disorders.
Our results indicate that ADHD scores do not increase before fathers were first reported to be missing in the household. The estimated coefficients $\alpha_1$ on fathers' absence are close to zero or even negative, indicating that fathers' absence is likely not a consequence of a behavioral disorder of children. These results provide us with confidence that the results presented in the previous sections not just reflect reversed causality but are truly informative about the effect of fathers' absence.

## 5.6 Conclusions

This article provides large sample evidence on the social causes of ADHD. We show that two events occurring frequently within families, increase the probability of developing mental disorders for children.
While it has been widely accepted that mental disorders such as depressive

moods or anti-social behavior are caused by family conflict or parental neglect, this has been doubted for ADHD. Our results show that both the absence of fathers in the household as well as the birth of siblings are related to ADHD, just as they are related to depressive symptoms and anti-social behavior. The effects are quite large and to some extent persist over a period of 2 years.
Moreover, fathers' absence and the birth of siblings shift symptoms of ADHD beyond a threshold which is often considered a critical level. Our results suggest that ADHD-type behavior can be a behavioral response to changes in the familial environment that likely exert stress on children. These findings oppose to the view that ADHD just results from a biological determinism.
In contrast to most previous research, our results cannot be driven by unobservable time invariant characteristics such as genetic disposition or the long-term consequences of exposure to environmental toxins alone. Our findings are in line with two hypotheses. First, changes in household composition cause ADHD. Second, the coincidence of changes in household composition and a genetic disposition for ADHD results in a symptomatology of ADHD. Only access to genetic data will allow to distinguish these two explanations. Our results highlight that a stable familial environment is one key ingredient in a healthy development of children. The increase in divorce rates across all industrialized countries could therefore be one cause for the steep increase in ADHD prevalence. Policy makers cannot interfere with family planing and divorce decisions. However, a greater emphasis can be put on the wellbeing of children by raising awareness for their specific needs and spreading relevant information to young families. However, whether this can prevent the incidence of ADHD remains uncertain and needs further being researched.

We suggest several directions for future research. Given the tremendous social costs of ADHD, it is surprising that little is known about the social origins of ADHD. There are other events which can be suspected to put similar pressure on children, such as a relocation, changes in parental labor force participation or domestic conflicts which are not associated with the move-out of a parent. Researching whether these events favor ADHD will also shed further light on policies to prevent ADHD.
It is straightforward to do the next step to understand why children are

negatively affected by the birth of siblings and their fathers' absence. One way to think about ADHD-type behavior is to consider it a strategy that children apply in order to get resources that are scarce in a household, such as parental quality time. Behavioral disorders could be one way of signalling needs to parents. Modeling interaction between children and looking empirically at their joint behavior could inform about the mechanisms that underly our findings.

Future research could address the interaction between genome and individual environmental conditions in the evolution of ADHD symptoms. There are two approaches to pursue this line of research. Linking a child's mental condition to her parents' and siblings' mental health histories could inform about pathways of the intra-familial transmission of mental disorders. It seems in particular interesting to link paternal mental health histories to a child's mental health and to study whether children respond differently to exogenous shocks when their parents have a history of mental disorders.

The second approach investigates whether specific circumstances "activate" or "deactivate" a genetic disposition. In particular, linking extensive survey panel data with data on the individual genome provides promising directions for future research. Until now, still little is known about the impact of genetic disposition on ADHD patterns. For instance, it is not known whether a disposition increases the probability for ADHD only in conjunction with specific environmental circumstances. If an ADHD symptomatology is developed only when genetic disposition and specific circumstances coincide, then there could be scope for interventions to prevent a child from developing the disorder.

## 5.7 Appendix: Measurement of Behavioral Disorders in the NLSY

The assessment of behavioral problems in the NLSY Children and Young Adults Study is based on 28 questions that are asked to mothers of children up to age 15. These questions are part of the module "mother supplement". The behavioral problems questionnaire incorporates questions on hyperactivity, depression, anti-social behavior as well as questions assessing the following constructs "headstrong", "dependent", "peer conflict" and "withdrawal". In this article we make use of the information on hyperactivity, depression and anti-social behavior. Our single indices are given by the sum of questions that were answered by parents with "often true" or "sometimes true".
The questionnaire is introduced by the following text:
*The following statements are about behavior problems many children have. For each item, think about [Child First Name]'s behavior over the last three months. Then indicate whether the statement is often true, sometimes true, or not true.*
The hyperactivity score contains the following 5 statements:

1. He/she has difficulty concentrating, cannot pay attention for long
2. He/she is easily confused, seems to be in a fog
3. He/she is impulsive, or acts without thinking
4. He/she has a lot of difficulty getting his/her mind off certain thoughts (has obsessions)
5. He/she is restless or overly active, cannot sit still.

The anti-social score has 6 statements:

1. He/she cheats or tells lies
2. He/she bullies or is cruel or mean to others
3. He/she does not seem to feel sorry after he/she misbehaves

4. He/she breaks things on purpose or deliberately destroys his/her own or another's things
5. He/she is disobedient at school
6. He/she has trouble getting along with teachers

The depression and anxiety score has 5 statements:

1. He/she has sudden changes in mood or feeling
2. He/she feels or complains that no one loves him/her
3. He/she is too fearful or anxious
4. He/she feels worthless or inferior
5. He/she is unhappy, sad or depressed

(Underlying variables: DEP1986-2008, ANTI1986-2008, HYPR1986-2008)

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# Eidesstattliche Erklärung

Hiermit erkläre ich, die vorliegende Dissertation selbständig angefertigt und mich keiner anderen als der in ihr angegebenen Hilfsmittel bedient zu haben. Insbesondere sind sämtliche Zitate aus anderen Quellen als solche gekennzeichnet und mit Quellenangaben versehen.

Mannheim, 25. Juni 2011

*Kai Eberhard Kruk*

# Curriculum Vitae

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|---|---|
| 2002 | Graduation from High School (Abitur), Ludwig-Marum-Gymnasium, Pfinztal, Germany |
| 2002-2003 | Volunteer Service Abroad in Italy (in lieu of military service) |
| 2003-2005 | Undergraduate Studies in Economics and Sociology, University of Trier, Germany |
| 2005-2008 | Diploma Studies in Economics, University of Mannheim, Germany |
| 2006-2007 | Participation in Exchange Program with Chulalongkorn University Bangkok, Thailand |
| 2008-2011 | Doctoral Studies in Economics, University of Mannheim |
| 2008-2011 | Research and Teaching Assistant, Mannheim Research Institute for the Economics of Aging (MEA), University of Mannheim |